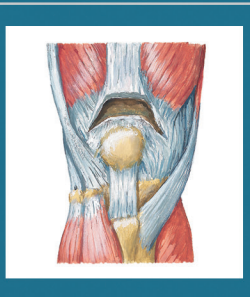
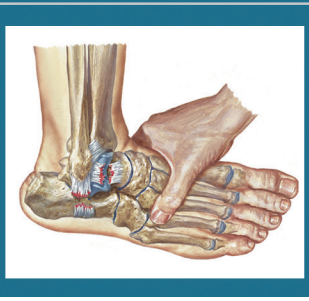
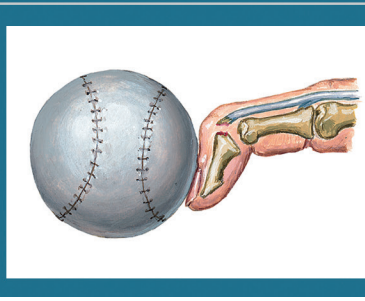
# NETTER'S SPORTS MEDICINE

2nd EDITION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# DEDICATION

*Netter's Sports Medicine* is dedicated to the *Team Physician's Handbook* and to the many thousands of sports medicine physicians and health professionals who have loyally followed and evolved with its content over three spectacular editions. *Netter's Sports Medicine* evolved from the original efforts embodying the *Team Physician's Handbook* and all that it represented for sports professionals through the years.

The Netter editions bring with them a broader and deeper approach to the comprehensive field of sports medicine. Many new topics, chapters, and authors are combined with cutting-edge Netter and other graphics, displayed in a colorful, user-friendly, easy-access format. *Netter's Sports Medicine* invites a much broader audience of sports medicine professionals and promises to be a premier ready reference and a detailed resource for all sports medicine professionals.

This book is also dedicated with respect and honor to the original creators of the *Team Physician's Handbook*, who we failed to acknowledge in the previous edition: W. Michael Walsh, MD, Morris B. Mellion, MD, and Guy L Shelton, PT, whose vision and wisdom made this work possible. May their love of sports medicine, conduct in the field, and integrity as human beings set an example for us all to follow.

And, to the most wonderful, loving family I can imagine: Jessica, my beautiful wife, and Sage, my energetic and inquisitive daughter. They are both my best teachers, and they bring me infinite happiness. Also, to my mother, Susan Madden, and my passed father, Spencer Madden, whose love, patience, belief, and guidance presented me with limitless opportunity.

**—Christopher C. Madden, MD, FACSM**

This book is dedicated to my parents, John and Elissa, who taught me to work hard, enjoy life, and respect other people; to my brother, Peter, and sister, Lisa, as well as their families; and to my husband and best friend, Joe Hindelang, as well as my stepson Joel. I treasure the time and memories we're able to spend together. I have learned so much in my career through my mentors, including my fellowship directors Dave Hough and Doug McKeag, as well as so many colleagues (many of whom are contributors to this book). I feel fortunate that I've had an opportunity to participate in this book with such great contributors and editors.

**—Margot Putukian, MD, FACSM**

This is dedicated to my parents, Margaret and Jimmie, who instilled in me the thirst for knowledge, and to my teachers who had the patience to give me that knowledge, especially to Drs. Jim Puffer, Bob Dimeff, and John Bergfeld. And finally, and most important, to my beautiful and wonderful wife, Sharon Busey, who has given me the love, support, and time to complete this project—I couldn't have done this without you.

**—Craig C. Young, MD**

My dedication of this book goes to my best friend and loving wife, Miriam, who is ever supportive, patient, and enduring in her love, especially in an effort such as this book, with the hours it takes and late nights to make it happen. To my dear children, Madeleine, Cleveland, Shannon, and Torrance, who are the light and diversion to the busy life as an academician, surgeon, and team physician. To my parents, Cleve and Jackie, who gave me great inspiration in pursuing excellence. To my many teachers, mentors, coaches, and orthopaedic partners who along the way have taught me so much and continue to live within me. Finally, to my ultimate mentor and the one who gave so much, my savior, Jesus Christ.

**—Eric C. McCarty, MD**

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# ABOUT THE EDITORS

**Christopher C. Madden, MD, FACSM,** is in private practice on the Front Range of the Rocky Mountains in Colorado. His sports medicine practice is broad, and he has special interests in head injuries, biomechanics, environmental and altitude medicine, backcountry sports, endurance and ultra-endurance medicine, snowboarding injuries, and cycling and mountain biking injuries. Chris edits and writes on a variety of topics in sports publications, ranging from previsit patient education to professional textbooks. He also teaches sports medicine to primary care residents from Rose and the University of Colorado Family Medicine Residency Programs. He is active in the American Medical Society for Sports Medicine, where he is a Past President and member of the Board of Directors, is past Annual Meeting Program Chair, and has served on the Quality Initiatives and Healthcare Transformation Subcommittee, Program Planning Committee, and Public Relations Committees. In 2011, Chris received the AMSSM's highest award, the Founders Award, which is given to "the individual, group or organization who exemplifies the best we can be or do in Sports Medicine." Chris is also a Fellow with the American College of Sports Medicine, where he has served on the Education Committee and has served on and chaired panels at various annual meetings. An avid backcountry enthusiast, Chris loves to mountain bike, trail run, snowboard, ski, hike, mountaineer, rock climb, practice yoga, travel, and spend time with his family.

**Margot Putukian, MD, FACSM,** received her BS degree in Biology from Yale University, where she participated in soccer and lacrosse. She received her MD from Boston University and then did both her internship and residency at the Primary Care Internal Medicine Program at Strong Memorial Hospital in Rochester, New York. She completed her fellowship in sports medicine at Michigan State University. Margot worked as the Director of Primary Care Sports Medicine at Penn State University before starting her current position in January 2004 as the Director of Athletic Medicine and Head Team Physician for Princeton University. Margot also serves as a team physician for U.S. Soccer and the Men's U.S. National Lacrosse Team. Margot is a charter member of the American Medical Society for Sports Medicine (AMSSM), where she served as President from 2004 to 2005, and is currently the President of the AMSSM Foundation. Margot is a medical consultant for Major League Soccer. She currently serves as the Chair for the Clinical Sports Medicine Leadership committee for the American College of Sports Medicine (ACSM) and served previously on the Board of Trustees for ACSM. She served previously on the NCAA Competitive Safeguards and Medical Aspects of Sport Committee and is currently serving as the chair of the Sports Science and Safety Committee for U.S. Lacrosse. She currently serves on the NFL Head, Neck & Spine Committee. Margot has participated in several Team Physician Consensus Statements, NATA Statements, and the Third and Fourth International Consensus Conference on Concussion in Sport in Zurich, Switzerland, as well as the Fifth International Consensus Conference on Concussion in Sport in Berlin, Germany. She has served on the editorial board for *Medicine and Science in Sports and Exercise*, the *Journal of Athletic Training*, *The Physician and SportsMedicine*, and *Athletic Training and Sports Health Care*. She is the proud recipient of the 2007 Dr. David Moyer Team Physician's award presented by the Eastern Athletic Trainer's Association, the 2015 AMSSM Founders Award, and the 2016 ACSM Citation Award.

**Craig C. Young, MD,** is a professor and the Medical Director of Sports Medicine at the Medical College of Wisconsin. He received a BS degree (cum laude) in Biological Sciences from the University of California, Irvine. He is a graduate of the University of California, San Diego School of Medicine. He completed a residency in family medicine at UCLA and a sports medicine fellowship at the Cleveland Clinic Foundation. Dr. Young has served as a team physician for the Milwaukee Brewers since 1994 and for the Milwaukee Bucks since 2016. He has served as a company physician for the Milwaukee Ballet since 1992. He has also served as a physician at the U.S. Olympic Training Center (Chula Vista) and is a physician for the U.S. National Ski and Snowboard Teams. In 2007, he was appointed by the U.S. Olympic Committee as a team physician for the 23rd World Winter University Games in Torino, Italy. Dr. Young is board certified in both family practice and sports medicine. He was the President of the American Society for Sports Medicine (AMSSM) from 2007 to 2008. In 2012, he was the recipient of AMSSM's highest award, the Founders Award, which is given to "the individual, group or organization who exemplifies the best we can be or do in Sports Medicine." His clinical interests include dance medicine, wilderness medicine, female athletes, adolescent athletes, and endurance athletes. His research interests include dance medicine and injury prevention.

**Eric C. McCarty, MD,** is a board–certified and fellowship-trained orthopaedic surgeon with a longtime interest in sports medicine and athletics. He attended college at the University of Colorado, where he excelled and received numerous honors for his exploits in the classroom, as well as on the football field, where he was an All Big-Eight linebacker and also was an Academic All-American. After medical school at the University of Colorado, he completed his training in orthopaedic surgery at Vanderbilt University in Nashville, Tennessee. From there he completed an intensive year of fellowship training in sports medicine and shoulder surgery at the internationally renowned Hospital for Special Surgery in New York City. He subsequently returned to Vanderbilt as a faculty member in the department of orthopaedics. In 2003, Dr. McCarty was recruited from Vanderbilt University to take over the sports medicine and shoulder program and to serve as the head team physician for the University of Colorado and University of Denver athletic programs. His specialized practice involves the care of these collegiate athletes, as well as recreational and highly competitive athletes from the community. In addition to his busy clinical practice, Dr. McCarty is very active in research, teaching, and writing articles in the field of sports medicine and knee and shoulder surgery. He has received grants for his research and frequently gives talks at both the national and international level. Since his playing days, Dr. McCarty continues to maintain a very active lifestyle with his family. He enjoys the activities he grew up with in Colorado, including hiking, cycling, climbing, and skiing. This carries over into his unbridled dedication to returning his patients to their desired activity/sport.

# ABOUT THE ARTISTS

## FRANK H. NETTER, MD

Frank H. Netter was born in 1906 in New York City. He studied art at the Art Student's League and the National Academy of Design before entering medical school at New York University, where he received his MD degree in 1931. During his student years, Dr. Netter's notebook sketches attracted the attention of the medical faculty and other physicians, allowing him to augment his income by illustrating articles and textbooks. He continued illustrating as a sideline after establishing a surgical practice in 1933, but he ultimately opted to give up his practice in favor of a full-time commitment to art. After service in the United States Army during World War II, Dr. Netter began his long collaboration with the CIBA Pharmaceutical Company (now Novartis Pharmaceuticals). This 45-year partnership resulted in the production of the extraordinary collection of medical art so familiar to physicians and other medical professionals worldwide.

In 2005, Elsevier, Inc. purchased the Netter Collection and all publications from Icon Learning Systems. There are now over 50 publications featuring the art of Dr. Netter available through Elsevier, Inc. (in the US: www.us.elsevierhealth.com/Netter and outside the US: www.elsevierhealth.com).

Dr. Netter's works are among the finest examples of the use of illustration in the teaching of medical concepts. The 13-book *Netter Collection of Medical Illustrations*, which includes the greater part of the more than 20,000 paintings created by Dr. Netter, became and remains one of the most famous medical works ever published. The Netter *Atlas of Human Anatomy*, first published in 1989, presents the anatomical paintings from the Netter Collection. Now translated into 16 languages, it is the anatomy atlas of choice among medical and health professions students the world over.

The Netter illustrations are appreciated not only for their aesthetic qualities, but, more important, for their intellectual content. As Dr. Netter wrote in 1949, ". . . clarification of a subject is the aim and goal of illustration. No matter how beautifully painted, how delicately and subtly rendered a subject may be, it is of little value as a *medical illustration* if it does not serve to make clear some medical point." Dr. Netter's planning, conception, point of view, and approach are what inform his paintings and what makes them so intellectually valuable.

Frank H. Netter, MD, physician and artist, died in 1991.

Learn more about the physician-artist whose work has inspired the Netter Reference collection:
**http://www.netterimages.com/artist/netter.htm**.

## CARLOS MACHADO, MD

Carlos Machado was chosen by Novartis to be Dr. Netter's successor. He continues to be the main artist who contributes to the Netter collection of medical illustrations.

Self-taught in medical illustration, cardiologist Carlos Machado has contributed meticulous updates to some of Dr. Netter's original plates and has created many paintings of his own in the style of Netter as an extension of the Netter collection. Dr. Machado's photorealistic expertise and his keen insight into the physician/patient relationship inform his vivid and unforgettable visual style. His dedication to researching each topic and subject he paints places him among the premier medical illustrators at work today.

Learn more about his background and see more of his art at:
**http://www.netterimages.com/artist/machado.htm**

# PREFACE

We are grateful for the opportunity to carry on the widespread popularity of the first edition of *Netter's Sports Medicine*, the history of *Team Physician's Handbook*, and the revered anatomical graphic works of Frank Netter, MD. The second edition continues to embrace a well-organized, colorful, bulleted outline format combined with helpful Netter graphics, tables, figures, pictures, diagnostic imaging, and other medical artwork. The text hosts a national and international author base that represents the best in sports medicine today.

Serving as a team physician is a unique privilege and an awesome challenge. *Netter's Sports Medicine* is written for the multitude of physicians and other health care professionals who are fortunate enough to provide care to a variety of athletes and active individuals in almost any athletic setting imaginable, from pediatric to senior athletics, Little League to professional sports, weekend warrior to Olympic champion, and backcountry mountainside to Super Bowl field.

The book is designed to serve as a comprehensive sports medicine resource and a ready reference in the busy outpatient office, in the training room, on the sideline, and in the long, quiet hours of preparation for sports medicine board certification. Insightful, expert, anecdotal experience fills the void where the most current evidence in sports medicine falls short, and careful considerations of controversies are mindfully presented. The sports medicine literature has grown exponentially since the first edition of *Netter's Sports Medicine* and its predecessor three editions of the *Team Physician's Handbook*, and many new chapters and chapter sections were added and revised to reflect the evolving depth and breadth of our exciting field. The text is divided into user-friendly sections for quick reference, and each chapter includes a Recommended Readings section limited to the best sources. We have added videos to support learning, and additional information supplementing certain chapters is available online. We welcome many new, respected authors who joined us to produce this book, and we are fortunate to continue our lasting relationship with numerous previous authors, who are leaders in their respective areas of emphasis. We thank all the authors who contributed chapters to previous texts, and whose chapter templates continue to provide a strong foundation to build upon.

Whether you are a primary care physician attempting to manage a common or unique musculoskeletal injury in an efficient ambulatory setting, an orthopaedic surgeon trying to gain insight about a medical or psychological problem foreign to the cast or operating room, an athletic trainer trying to figure out a diagnosis in the training room, or a physical therapist pursuing further in-depth sports medicine knowledge, we sincerely hope you find this reference all it is meant to be and more, and we thank you for opening the cover and sharing with us what we feel is one of the highest quality sports medicine works produced to date. Please enjoy.

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- **Chapter 61, Taping and Bracing,** *Rebecca Ann Myers*
  - Video 61-1 Ankle Taping
  - Video 61-2 Thumb Spica Taping
  - Video 61-3 Patellofemoral Taping
  - Video 61-4 Arch Taping
  - Video 61-5 Turf Toe Taping
- **Chapter 79, Snowboarding,** *Thomas R. Sachtleben and Rebecca Ann Myers*
  - Video 79-1 Anatomical Relationships of LPT Fracture
  - Video 79-2 LPT Stress Test Demonstration
- **Chapter 82, Swimming and Diving,** *Jennifer D. Stromberg and Nathaniel S. Jones*
  - Video 82-1 Front Crawl (Freestyle)
  - Video 82-2 Back Stroke
  - Video 82-3 Breaststroke
  - Video 82-4 Butterfly Stroke
  - Video 82-5 Eggbeater Kick
  - Video 82-6 Flip Turn

### PRINTABLE FORMS

- eForm 16-1 Example of a PPE Health History Form for a Wilderness Sports/Adventure PPE
- eForm 27-1, 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad
- eForm 97-1 Medical Record Form
- eForm 97-2 Medical Discharge/ER Transfer Form

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- MANAGING YOUR ANKLE SPRAIN
- MANAGING YOUR BLISTERS
- MANAGING YOUR FINGER SPRAIN
- MANAGING YOUR GROIN STRAIN
- MANAGING YOUR GOLFER'S ELBOW
- MANAGING YOUR HAMSTRING STRAIN
- MANAGING YOUR PLANTAR FASCIITIS
- MANAGING YOUR CONCUSSION
- MANAGING YOUR KNEE SWELLING
- MANAGING YOUR LOW BACK STRAIN
- MANAGING YOUR NECK STRAIN
- CARING FOR YOUR CHILD WITH OSGOOD-SCHLATTER CONDITION
- MANAGING YOUR PATELLOFEMORAL PAIN SYNDROME
- MANAGING YOUR PATELLA TENDINITIS
- MANAGING YOUR ROTATOR CUFF TENDINOSIS
- MANAGING YOUR SHIN SPLINTS
- MANAGING YOUR TENNIS ELBOW

# THE TEAM PHYSICIAN

# 1

Margot Putukian • Christopher C. Madden • Craig C. Young • Eric C. McCarty

## BEING A TEAM PHYSICIAN: A SPECIAL PRIVILEGE, AN AWESOME CHALLENGE

### Special Role

- Team physicians have a unique responsibility of leadership while taking care of athletes.
- Athletes, their parents, and their team administrators expect team physicians to make decisions in terms of clearance to participate in the sport as well as to assess and manage healthcare issues in order to ensure safe participation of athletes.
- In addition, such decisions may be required in a setting of intense time pressure, such as when required on the sidelines.
- Team physicians may play a leadership role within an organization and may provide care for individuals and teams at mass participation sporting events. In addition, team physicians have responsibilities and duties that are both medical and administrative, and these frequently include ethical and medicolegal issues.
- This chapter discusses the requirements of a team physician in terms of medical qualifications, education, and roles and responsibilities.

## THE SPORTS MEDICINE TEAM

- Care of an athlete is a team effort, wherein members of a sports medicine team support each other for the benefit of the athlete and the athletic team.
- An athletic trainer occupies a unique position at the center of the athletic healthcare triangle.
- A team physician cares for the team and also serves as a key player on the sports medicine team comprising the athlete, the team physician, the coach, the athletic trainer, and other supporting health professionals. Similar to the athletic team, sports medicine services are best provided following a team approach (Fig. 1.1).

### Availability

- Availability is a cornerstone for success of a team physician.
  - On the sidelines, at events, and during travel
  - In the training room
  - In the office: Team physicians may have to include special accommodations in their office schedules for athletes who require urgent medical attention.
  - After office hours and weekends: Most athletic activity happens outside a normal work day; thus, team physicians must accommodate this in their coverage considerations.

## DEFINITION OF THE TEAM PHYSICIAN

- Six major professional associations focusing on clinical sports medicine issues collaborated to develop guidance documents for team physicians.
- These "Team Physician Consensus Statements (TPCS)" cover various topics for team physicians, including the definition of team physicians: **TPCS** (see Appendix A, online) and **Sideline Preparedness for the Team Physician: Consensus Statement** (see Appendix B, online).
- All other TPCS are referenced in "Recommended Readings," which are useful resources that cover specific topics and populations.
- The TPCS that defines the qualifications, roles, and responsibilities of team physicians along with guidelines for individuals and organizations seeking to select a team physician were updated in 2013.
- The team physician must be a medical doctor (MD) or doctor of osteopathy (DO) with an unrestricted license in good standing and knowledge of on-field emergency care and basic cardiopulmonary resuscitation techniques as well as musculoskeletal injuries and medical and psychological issues that affect athletes.

## RESPONSIBILITIES OF THE TEAM PHYSICIAN

### Medical Care

- The most important role of the team physician is to address the physical and psychological needs of an athlete.
- In addition to the essential requirements described in the TPCS, it is also desirable that the team physician has additional training and education in sports medicine, with medical specialty and fellowship training and additional American Council of Graduate Medical Education (ACGME)/American Osteopathic Association (AOA) certification in sports medicine, and additional experience, including:
  - Ongoing continued medical education in sports medicine
  - Experience in sports medicine
  - Membership and participation in a sports medicine professional association or society
  - Training in advanced cardiac and trauma life support
  - Ongoing involvement in education and research in sports medicine
- Understanding the complexities of medicolegal, disability, and compensation issues that can occur in sports medicine is helpful.
- To perform effectively, the team physician must maintain a broad and up-to-date knowledge base that addresses athletics as well as medicine.
  - All team physicians should feel comfortable in providing emergency care at sporting events.
  - Training in cardiopulmonary resuscitation (CPR) and automated external defibrillator (AED) use is essential, and additional knowledge of advanced cardiac life support (ACLS) and advanced trauma life support (ATLS) is useful.
  - In addition, the team physician should have knowledge in the following areas:
    - **Medicine:** full-spectrum primary care, including musculoskeletal system, growth and development, cardiovascular and pulmonary medicine, infectious disease, gastroenterology, nephrology, neurology, and other medical areas pertaining to exercise and sports
    - **Psychology and behavior:** mental health issues such as depression, anxiety, eating disorders, alcohol and other drug use/abuse, and psychological response to injury
    - **Pharmacology:** therapeutics, supplements, performance enhancers, recreational drugs, interactions among these agents, and effects on performance
    - **Nutrition and exercise science:** nutrition, exercise physiology, biomechanics, sport-specific issues (e.g., altitude and other environmental issues)
    - **Sport- and population-specific issues:** sport- and population-specific issues, including gender, age, disability, or other unique population factors

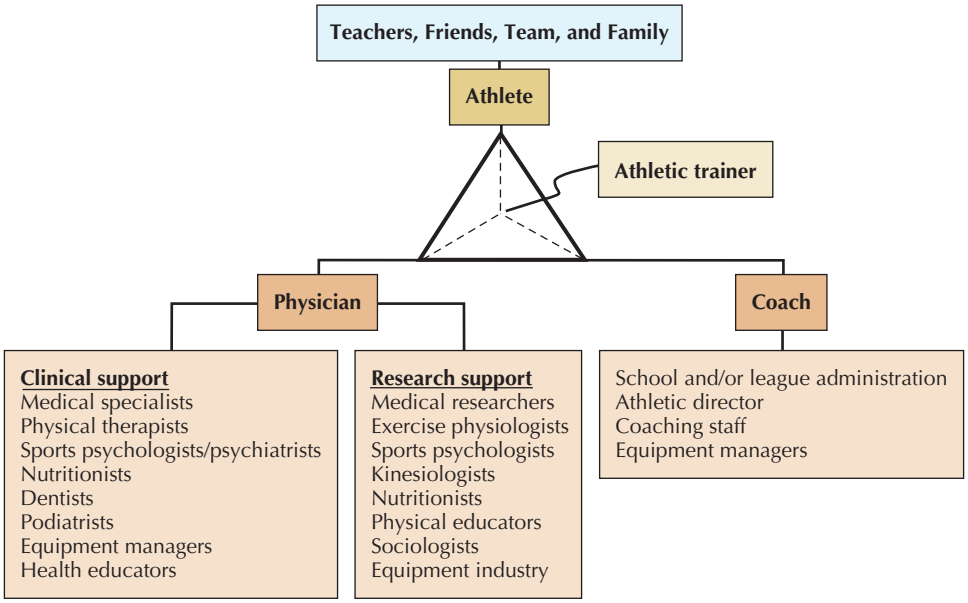


**Figure 1.1.** The sports medicine team. (Modified from Mellion MB. *Office sports medicine.* Philadelphia: Hanley & Belfus; 1996.)

### Additional Medical Responsibilities

- Coordinating assessment and management of injuries and illness on game day, including making decisions about clearance and same-day return to play
- Understanding the importance of pre-participation physical evaluation (PPE), emergency planning, and issues of sudden death and heat illness as well as recovery and rest

## Administrative Responsibilities

The team physician has a range of important administrative responsibilities:

- Establish a chain of command for injury and illness management, including:
  - Involvement in planning and implementation of emergency action plans
  - Involvement in and awareness of protocols and equipment required for sideline preparedness
  - Coordinating assessment and management of game-day injuries and illnesses, including return-to-play decision-making
- Clearance for non-game day participation and return-to-play decision-making
- Understand the importance of injury and illness prevention
- Understand the importance of collaboration with other healthcare providers, including athletic trainers, physical therapists, nutritionists, strength and conditioning specialists, psychologists, and other specialists, in the care of an athlete
- Understand the role of nutrition, supplements, and performance-enhancing agents
- Important decisions regarding PPE and what it may include (e.g., screening for mental health issues, concussion baseline testing, and cardiac screening)
  - Such decisions are often complex and may involve several other stakeholders.
- Additional considerations include preventive measures:
  - Immunizations
  - Educational efforts (e.g., concussion, heat-related illnesses, nutrition, and cardiac illnesses)
  - Injury and illness prevention (e.g., anterior cruciate ligament [ACL] injury prevention or prophylaxis for communicable skin diseases)
- An important administrative responsibility of the team physician is to work with the organizing body (e.g., school, university, or club) to develop an agreement that clearly defines the roles and responsibilities of the team physician as well as the reporting structure for other healthcare providers (e.g., the team physician makes clearance decisions and the athletic trainer reports to the team physician and not the coach)

## Ethical and Medicolegal Responsibilities

The team physician has a range of responsibilities that reflect the many relationships involved in the care of an athlete:

- Responsibilities toward the athlete, the team, and the institution and its representatives must be considered.
- All physicians have ethical responsibilities, but those of team physicians may be somewhat unique given the complex and often public nature of sports participation.
- As stated in the TPCS, "the overriding principle for all physicians, including team physicians, in managing ethical issues, is to provide care focused on what is best for the patient and only for the patient." Several examples of ethical challenges are provided below.

### Informed Consent

- Information provided by team physicians to an athlete and/or his or her parent/guardian must be complete and inclusive of all options so that the athlete can make an informed decision.
- Information should include a discussion of short- and long-term risks and benefits and balance the athlete's autonomy, desires, and optimal medical treatment.
  - For example, an athlete with a meniscus tear amenable to repair should be provided with all information so that he/she can make an informed decision (e.g., short-term benefits of meniscectomy and return-to-play versus potential long-term benefits of repair).

  - Team physicians should provide information with a goal of protecting athletes from injury, re-injury, permanent disability, and themselves.
- Athletes must be counseled and thoroughly informed when there is a valid medical contraindication to participation or resumption of participation.

### Confidentiality

- Team physicians must respect the rights of patients and safeguard their confidentiality within the constraints of the law, respecting both the Health Insurance Portability and Accountability Act of 1996 (HIPAA) as well as the Family Educational Rights and Privacy Act (FERPA).
- Relationship with athletes may have to be clarified in advance. Challenging examples may include:
  - Medical conditions that limit or affect participation
  - Psychological issues that may limit or affect participation
  - Medical issues that may affect other participants
  - Drug testing results

### Conflict of Interest

- There are some situations where team physicians may experience a conflict of interest or a perceived conflict of interest.
- These include situations where a team physician is hired by a professional organization to care for team members, wherein there is a financial relationship with particular organizations, which may lead to a conflict with the care provided to the athlete.

### Influence of Others

- Influence of others, such as teammates, parents, coaches, and administrators, may conflict with the medical care provided to the athlete.
- Team physicians should remain aware of potential implicit and explicit influences, including those provided by the community and media.

### Marketing, Publicity, and Advertising

- A potential ethical issue for team physicians may occur when a company or individual offers compensation for services and medical care provided by them.
- Team physicians may be sponsored by a company or industry with the biased expectation that they will use one product over another.
- Ethical issues can arise when team physicians are expected to endorse or use a new technology (e.g., equipment, treatment modality, or medications) without substantial evidence of efficacy or safety.

### Drug Use

- Team physicians may be asked to prescribe or administer pain medications in order to allow an athlete to participate.
- Athletes using illegal, illicit, or performance-enhancing drugs may pressure team physicians to supply, administer, or help cover-up the use of such drugs.

### Medicolegal Issues

- Certain medicolegal issues may be unique for team physicians. Specific concerns may include:
  - Team physicians should clearly define professional autonomy over medical decisions.
  - Issues regarding HIPAA and FERPA compliance
  - Guidelines, standards, policies, and regulations set by school and governing bodies
  - Rules, regulations, and/or laws of local, state, or federal government
  - Issues regarding the management of on-field (e.g., cardiac, concussion, cervical spine, or heat) injuries or illnesses
  - Issues regarding clearance to play and/or restriction from play, waivers, and return-to-play decision-making
  - Issues regarding medical documentation

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Available at www.ExpertConsult.com.

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# 2 THE CERTIFIED ATHLETIC TRAINER AND THE ATHLETIC TRAINING ROOM

Jodi Schneider

## DEFINITION OF AN ATHLETIC TRAINER

- Athletic trainers (ATs) are healthcare professionals who collaborate with physicians to direct injury prevention, evaluation, management, and treatment of patients under their care (see Chapter 1: The Team Physician).
- ATs work under the direction of physicians, as prescribed by state licensure statutes.
- They are uniquely qualified, allied healthcare providers who are optimally suited as front-line gate keepers and first responders for all athletic-related healthcare issues.
- ATs provide services including injury and illness prevention, emergency care, on-field and clinical diagnosis, patient education, and rehabilitation and therapeutic intervention of acute and chronic injuries and illnesses.
- Apart from responsibilities of injury prevention, evaluation, management, and treatment of patients under their care, communication is one of the most important responsibilities of ATs.
- They also have a responsibility as a liaison between the physician, patient, coaching staff, and support staff to coordinate effective patient-centered care.
- Settings
  - Traditional
    - High school, college, and professional sports
  - Nontraditional
    - Hospital/orthopedic practice
    - Military/special forces
    - Occupational
    - Performing arts
    - For additional information on nontraditional settings for ATs, please visit http://www.nata.org/athletic-training/job-settings (accessed March 2016).

## EDUCATION

- Graduation from a Commission on Accreditation of Athletic Training Education (CAATE) with an accredited, 4-year undergraduate or a 2-year, entry-level masters program to be eligible to sit for the certification exam
- Curriculum CAATE 2015
  - Risk Management and Injury Prevention
  - Pathology of Injuries and Illnesses
  - Orthopedic Clinical Examination and Assessment
  - Medical Conditions and Disabilities
  - Acute Care of Injuries and Illnesses
  - Therapeutic Modalities
  - Conditioning and Rehabilitative Exercises
  - Psychosocial Intervention and Referral
  - Nutritional Aspects of Injuries and Illnesses
  - Healthcare Administration
  - Professional Development and Responsibility
  - Healthcare Professional Development and Responsibility
- 70% of candidates possess a master's or doctorate degree
- After professional education
  - Residency/fellowship: An emerging aspect of an AT's education is optional post-professional residencies or fellowships. These programs are designed to provide an advanced level of clinical and didactic education in specialized areas.

### Licensure

- As of January 2016, all states, with the exception of California, require licensure or registration to practice as an AT.

## ROLES AND RESPONSIBILITIES OF AN ATHLETIC TRAINER

Domains of Practice, National Athletic Trainers' Association (NATA), 2015

### Injury/Illness Prevention and Wellness Protection

- Education of patients, coaches, and administrators
- Implement and assist in the administration of preparticipation physical examination with physicians
  - Preseason musculoskeletal screening
- Playing surface, environmental, and weather safety monitoring
- Screening and referral for mental health and psychological concerns
  - Effective recognition and referral to appropriate care providers
- Supplement monitoring and education and oversight of weight management protocols and safety in weight-class sports
  - Coordination with administration, coaches (including strength and conditioning), sports dietitians, and physicians in developing and implementing monitoring and education

### Clinical Evaluation and Diagnosis

- Acute and chronic injury evaluation on and off the field and effective referral to physician when necessary
- Concussion evaluation, protocol management, and referral to physician

### Immediate and Emergency Care

- Development and implementation of emergency action plans based on the most up-to-date consensus/position statements and best practices. For a detailed list of up-to-date NATA position statements, please visit http://www.nata.org/position-statements.
- Coordination and pre-event communication with local emergency services departments
- Prompt and proficient emergency care based on current standards of care

### Treatment and Rehabilitation

- Management and treatment of injuries and illnesses by using evidence-based practices
- Returning patients to full preinjury function as soon as safely possible
- Determining the ability to safely return with functional testing
- Communication and coordination of rehabilitation and transition to full function with strength and conditioning and coaching staff
- Communication and coordination with external rehabilitation providers when appropriate

### Organization and Administration

- Development and implementation of policies and procedures of the athletic training facility, emergency action plans, and medical coverage of events
- Maintain professional relationships to coordinate patient care with:
  - Coaching staff, including strength and conditioning staff
  - Administrators
  - Dietitians
  - Psychologists/counselors
  - External providers
- Timely and accurate medical record keeping
- Maintenance of supplies and budget of the athletic training facility

## HOW AN ATHLETIC TRAINER AND A PHYSICIAN FUNCTION AS A TEAM

- ATs work under the direction of the team physician based on state practice acts
  - AT license requires a written Plan of Care, which varies from state to state depending upon the legislation, to guide the day-to-day practice of an AT and can include:
    - Timeframe for referral to physician
    - Emergency care procedures
    - Nonprescription medication administration
    - Treatment protocols and modality usage
  - Communicate effectively to provide collaborative patient-centered care
    - AT coordinates effective referrals to physician.
    - Physician and AT work together to make return-to-play decisions.
    - AT communicates frequently on day-to-day progress and status to physician to facilitate modifications to care plan as necessary.
- The team physician and AT work together and develop policies and procedures specific to the institution/facility.
  - Developing, communicating, and enforcing physician-led chain of commands and establishing subsequent AT supervisory positions within the sports medicine department.
  - Development of facility-specific emergency action plans and clearly communicating them to all members of the athletic department
  - Development and implementation of appropriate medical coverage policies
  - Standard operating procedures for sports medicine facilities and the staff
- Members of interdisciplinary treatment and performance teams
  - No greater demonstration of commitment to patient-centered care than development and participation in interdisciplinary treatment and performance teams. Aligning medical, coaching/performance, and academics to provide complete personal care and support to athletes. ATs and physicians are crucial and invaluable members of these teams not only because they provide medicalcare/healthcare but also because of the level of relationship they develop with the athletes. This also provides ATs and physicians varied perspectives to help direct patient care and support.
  - Examples include:
    - Mental health
    - Eating disorders
    - Sport performance
    - Academic performance
    - Life skills/transition to post-athletic life

## ATHLETIC MEDICINE/SPORTS MEDICINE DEPARTMENT MODEL

- Responsibility to provide an effective sports medicine structure/model free of conflict of interest. Determination of the best model varies from setting to setting.
- Several different models have been utilized across the United States, the most common one being Sports Medicine housed within the Athletic Department. Recently, there have been more changes to Sports Medicine housed within University Health Services in an effort to decrease conflict of interest and provide better athlete-centered care. For a comprehensive description of other models along with their advantages and disadvantages, please see "Inter-Association Consensus Statement on Best Practices for Sports Medicine Management for Secondary Schools and Colleges."

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 3 THE PREPARTICIPATION PHYSICAL EVALUATION

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## PREPARTICIPATION PHYSICAL EVALUATION (PPPE)

- History and physical examination, with additional testing as indicated, is performed before participation in sport that meets several objectives and is one of the most important functions provided by the sports medicine physician.
  - Addition of a 12-lead echocardiography (ECG) examination as part of the standardized screening process is controversial.
- Often, this is the first interaction between the physician and the athlete; for many young adults, it may be the first exposure to the healthcare system.
- It does not replace regular physical examinations, although many athletes think that it covers all healthcare needs.
- It encompasses clearance for participation in the sport and provides education and information to athletes regarding issues such as nutrition, supplementation, training and conditioning, injury prevention, and rehabilitation.
- Special considerations of PPPE include age specificity, sex specificity (special concerns for female vs. male athletes), sport specificity (specific demands of each sport should be considered), athletes with special needs, and athletes with physical or intellectual disabilities.

## OBJECTIVES OF THE PPPE

- Emphasize cardiovascular, neurologic, and musculoskeletal issues
- Identify any life-threatening or disabling conditions (e.g., underlying cardiovascular or neurologic abnormalities)
- Identify any conditions that may put an athlete at risk of injury or illness (e.g., underlying ligamentous instability, musculoskeletal abnormalities, organomegaly, or acute medical illness)
- Assess an injury that has not been appropriately rehabilitated
- Assess medical conditions and strength and flexibility deficits that put an athlete at risk of injury
- Assess general health status (e.g., immunizations), fitness, and maturity
- Meet insurance or legal requirements
- Screen for menstrual dysfunction, stress fractures, or disordered eating (female athlete triad, disordered eating in male athletes)
- Introduce athletes to the healthcare system and concepts of preventive medicine
- Offer an opportunity to address issues such as recreational and performance-enhancing substance use and abuse, sexuality issues, depression and emotional issues, and health promotional activities (alcohol and drug abuse, seat belts, helmets, and self-breast or self-testicular examination)

## TIMING

- The PPPE should be performed at least 6 weeks before the beginning of the sport season to allow adequate time for further evaluation of identified problems and treatment or rehabilitation of any conditions or injuries.
- If athletes are unavailable 4–6 weeks before the beginning of an early fall season, examinations performed at the end of the previous school year may be considered. Athletes should report any interval injuries, illnesses, and new medications between their examinations and the beginning of the fall season.
- A detailed medical history may be completed by athletes and/or parents in advance, which may improve the accuracy of the information (e.g., immunization records) and examination efficiency. Internet resources can facilitate the history and interval injury reporting process. An electronic format (ideally a national database) has several benefits, including communication and administration of the PPPE.

## FREQUENCY

- Variable recommendations depend on individual athletes (i.e., age, gender, sport [single or multiple]; their health [underlying medical conditions or injury history], and cost); availability of records from past PPPEs (continuity of care); and requirements of state, city, or athletic governing body.
- **General guidelines** (no consensus about optimal frequency)
  - Comprehensive baseline PPPE before initiating a new sport or attaining a new level (e.g., entry into high school, college, or professional level), every 2 years in younger athletes (e.g., middle and high school students), and every 2–3 years in older athletes
  - Subsequent annual PPPEs may be limited to injuries or illnesses disclosed by an interim health questionnaire; yearly evaluation of the cardiopulmonary system may be appropriate.
  - If an athlete is participating in multiple sports during the year, consider more frequent evaluations.
  - Several **states** require an annual full screening examination (no standard requirements).
  - The **National Collegiate Athletics Association** (NCAA) requires an initial comprehensive PPPE on entrance, followed by interim history in intervening years; limited additional examinations focusing only on new problems.
  - The **American Heart Association** (AHA) recommends initial comprehensive PPPE on entrance for high school and college athletes. The AHA recommends another comprehensive PPPE after 2 years for high school athletes and follow-up interim history and blood pressure measurements annually, along with focused additional examinations for new problems for college student athletes.

## METHODOLOGY

### Office-Based

**Potential advantages:** patient-centered, physician–patient familiarity, privacy, and continuity of care

**Potential disadvantages:** greater cost, limited appointment time, limited physician interest/experience, and lack of communication of pertinent information to school athletic staff

### Coordinated Medical Team-Based (Table 3.1)

**Potential advantages:** specialized personnel, time and cost efficiency, and good communication with school athletic staff

**Potential disadvantages:** rushed examinations, lack of privacy, and inadequate follow-up of identified problems

**Two types of group PPPEs:** multistation (multiple physicians, each at a specialized station) and "locker room" (single or multiple physicians performing complete examinations individually, each in their own area [e.g., locker room]).

**TABLE 3.1 REQUIRED AND OPTIONAL STATIONS AND PERSONNEL FOR COORDINATED PREPARTICIPATION PHYSICAL EVALUATION**

| Required Stations | Personnel |
|---|---|
| Sign-in, height and weight (BMI*), blood pressure, and vision | Ancillary personnel (coach, nurse, and community volunteer) |
| History review, physical examination†, and clearance | Physician |
| **Optional Stations** | **Personnel** |
| Nutrition | Dietitian |
| Dental | Dentist |
| Injury evaluation‡ | Physician |
| Flexibility | Trainer or therapist |
| Body composition | Physiologist |
| Strength | Trainer, coach, therapist, and physiologist |
| Speed, agility, power, balance, and endurance | Trainer, coach, and physiologist |

*Body mass index (BMI) can be calculated from height and weight (for specific age- and gender-adjusted categories, see www.cdc.gov/growthcharts).
†Physical examination can be subdivided if more than one physician is present.
‡A musculoskeletal injury evaluation station may be used to provide a more complete evaluation when a musculoskeletal injury is detected during the required musculoskeletal screening examination.

## Recommendations

- At the final station of a station-based examination, an experienced team physician should be available to review all data and to determine clearance or provide appropriate recommendations.
- Communication between other primary or consulting physicians, athletic trainers, coaches, and parents may be enhanced by carefully documenting the problems and specific recommendations in the clearance section of the PPPE form.
- Cases of special concern may warrant a telephone conversation between the team physician and other involved healthcare providers.
- The 2010 *Preparticipation Physical Evaluation* monograph (see "Recommended Readings") considers "gymnasium examination" to be inadequate to achieve the goals and objectives of the PPPE process.

## Personnel

### Physicians

- PPPEs should be performed by an MD/DO physician, nurse practitioner, or physician assistant, with final clearance by an MD or DO physician.
- Regulations by certain states at the high school level allow other practitioners (e.g., chiropractors or naturopathic clinicians) to perform PPPEs.
- Primary care physicians perform a majority of PPPEs because of their ability to evaluate all organ (i.e., cardiopulmonary, musculoskeletal, neurologic, ophthalmologic, gastrointestinal, genitourinary, and dermatologic) systems.
- Specialists such as orthopedic surgeons, cardiologists, and ophthalmologists or optometrists are key consultants and may be present on site during the screening-station format examination.

### Ancillary

- Medical staff, including athletic trainers, physical therapists, nurses, exercise scientists, dietitians, and sports psychologists, may be involved, particularly during the screening-station format PPPE.
- Nonmedical staff, including coaches, school administrators, and community volunteers, are particularly helpful during the screening-station format PPPE.

## MEDICAL HISTORY

- There is emphasis on screening for cardiovascular and musculoskeletal problems, prior head injuries and other neurologic problems, and significant recent illnesses. In addition, prior heat illness, pulmonary problems, medication problems, inadequate immunizations, allergic reactions, skin problems, and menstruation abnormalities and disordered eating disorders in female athletes should be addressed (PPPE: History Form available at https://www.aap.org/en-us/professional-resources/practice-support/Documents/Preparticipation-Physical-Exam-Form.pdf, accessed March 2016). Medical history is an essential component of the PPPE that detects abnormalities in a majority of athletes.
- Joint completion of history forms by athletes and parents/guardians is recommended when possible, particularly if the athlete is unclear about family or personal history. In addition, parent/guardian should be present or available during PPPE for additional questions that may arise.
- **Cardiovascular history** (Table 3.2):
  - Screen for causes of sudden cardiac death (SCD; Fig. 3.1). The most common cause in people aged <35 years is hypertrophic cardiomyopathy (HCM; Fig. 3.2); in people aged ≥35 years, the most common cause is coronary artery disease (CAD). The PPPE is scrutinized by certain physicians for its ability to detect underlying causes of SCD, particularly in younger patients. However, the AHA states that a certain form of preparticipation screening for high school and college athletes is justifiable and compelling based on ethical, legal, and medical grounds.
- **Personal history is important.**
  - History of exertional chest pain, tightness, or chest pressure, any unexplained syncope or near-syncope, and excessive and unexplained dyspnea/fatigue or palpitations associated with exercise are all significant.
  - Prior recognition of a heart murmur
  - Determine past history of invasive or noninvasive cardiac tests ordered by a physician.
  - Prior history of hypertension or prehypertension noted during examinations
- **Family history is important.**
  - Twenty-five percent of first-degree relatives of patients with HCM exhibit morphologic evidence of HCM in ECG.
  - Other known genetic cardiac conditions associated with SCD (e.g., long QT syndrome, other ion channelopathies, Marfan syndrome, clinically or significant arrhythmias)
  - Premature death (sudden and unexpected or otherwise) before 50 years of age attributable to heart disease in ≥1 relative
  - Disability from heart disease in close relatives aged <50 years
  - Hypertension

## Neurologic Concerns

- It is important to ask questions about previous head or neck injury, concussion, neurologic symptoms, exercise-related syncope, stingers/burners, and seizure disorder.

**TABLE 3.2 THE 14-ELEMENT AMERICAN HEART ASSOCIATION RECOMMENDATIONS FOR PREPARTICIPATION CARDIOVASCULAR SCREENING OF COMPETITIVE ATHLETES**

| Personal Medical History | Family History | Physical Examination |
|---|---|---|
| Chest pain/discomfort/tightness/pressure related to exertion | Premature death (sudden and unexpected or otherwise) in ≥1 relative aged <50 y attributable to heart disease | Heart murmur† |
| Unexplained syncope/near-syncope* | Disability from heart disease in a close relative aged <50 y | Femoral pulses to exclude aortic coarctation |
| Excessive and unexplained dyspnea/fatigue or palpitations, associated with exercise | Hypertrophic or dilated cardiomyopathy, long-QT syndrome, or other ion channelopathies or Marfan syndrome or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members | Physical stigmata of Marfan syndrome |
| Prior recognition of a heart murmur | | Brachial artery blood pressure (sitting position)‡ |
| Elevated systemic blood pressure | | |
| Prior restriction from participation in sports | | |
| Prior heart testing ordered by a physician | | |

*Judged not to be neurocardiogenic (vasovagal) in origin; is of particular concern when occurs during or after physical exertion.
†Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.
‡Preferably taken in both arms.
From Assessment of the 12-lead ECG as a screening test for detection of cardiovascular disease in healthy general populations of young people (12-25 years of age): a scientific statement from the American Heart Association and the American College of Cardiology.

- The NCAA has recommended symptom score, cognitive examination, and balance assessment as "best practices" for every athlete as part of his or her baseline physical examination.
- Concussion history including the number, symptoms, and time out of activity as well as a history for "modifiers" for concussion (e.g., migraine history, learning disability history, or history of depression/anxiety) should be considered as part of the baseline PPE.
- Any positive response mandates more thorough history, physical examination, and evaluation.

### Musculoskeletal Concerns

- Complete history is essential.
- History of previous ligamentous injuries, documentation of surgery, rehabilitation, and time out of play
- History of prior advanced imaging (e.g., radiographs, MRI, CT, or bone scan) for a musculoskeletal problem
- Any positive response mandates careful attention during physical examination, including assessment of ligamentous instability, strength and flexibility deficits/mismatches, and completeness of rehabilitation, as well as consideration for obtaining medical records related to the evaluation.
- If an athlete has had prior surgery, obtain medical records related to the evaluation and a documentation that the operating surgeon has cleared the athlete to return to competition and/or determine the athlete's rehabilitation status.

### Previous Medical Illnesses (Examples)

- Heat exhaustion/illness
- Infectious mononucleosis
- Hepatitis
- HIV disease
- Diabetes
- Sickle cell disease/hemoglobinopathy
- Asthma
- Allergic reactions

### Female Athlete Triad

**Screening questions:**
- Age of menarche and history of amenorrhea or oligomenorrhea
- History of stress fractures, bone injury, or risk factors for osteoporosis
- History and risk factors for disordered eating patterns: questions that ascertain ideal versus current body weight, body image concerns, and pathogenic eating behaviors

### Additional Concerns

Additional concerns not always included on the PPPE form may be addressed on an individual basis. If you do not ask, you might never find out.
- Nutritional issues: fluids, game-day nutrition, and general nutrition
- Supplements and performance-enhancing agents
- Sexuality concerns: pregnancy, sexually transmitted diseases, and sexual orientation (best addressed in a private setting)
- Recreational drugs and alcohol use
- Preventive medicine (e.g., seat belts, helmets, self-breast or self-testicular examination, cholesterol screening, and gynecologic examinations/Pap smear)
- Psychosocial issues: stress management, anxiety, depression, suicide (consider including screening questionnaires such as the Patient Health Questionnaire-9 [PHQ-9] or Generalized Anxiety Disorder-7 [GAD-7] for depression and anxiety, respectively)

## PHYSICAL EXAMINATION (Box 3.1)

- The physical examination should be comprehensive. It should focus on areas of greatest importance in sports participation and address any problems uncovered while recording an athlete's history.
- Adequate exposure during the examination is important.
- The **physical examination form** (PPPE: Physical Examination Form available at https://www.aap.org/en-us/professional-resources/practice-support/Documents/Preparticipation-Physical-Exam-Form.pdf, Accessed March 2016) is generally comprehensive and covers the scope of such examination, but it should not limit the clinician if additional examination is deemed pertinent.

### Height and Weight

- In athletes with excessive weight change, explore the possibility of eating disorders or steroid abuse.
- Body mass index (BMI) should be calculated (gender and age specific; see www.cdc.gov/growthcharts). Understand the indications and limitations of using BMI.
  - Underweight (<5$^{th}$ percentile)
  - Overweight (85$^{th}$–94$^{th}$ percentile)
  - Obese (≥95$^{th}$ percentile)

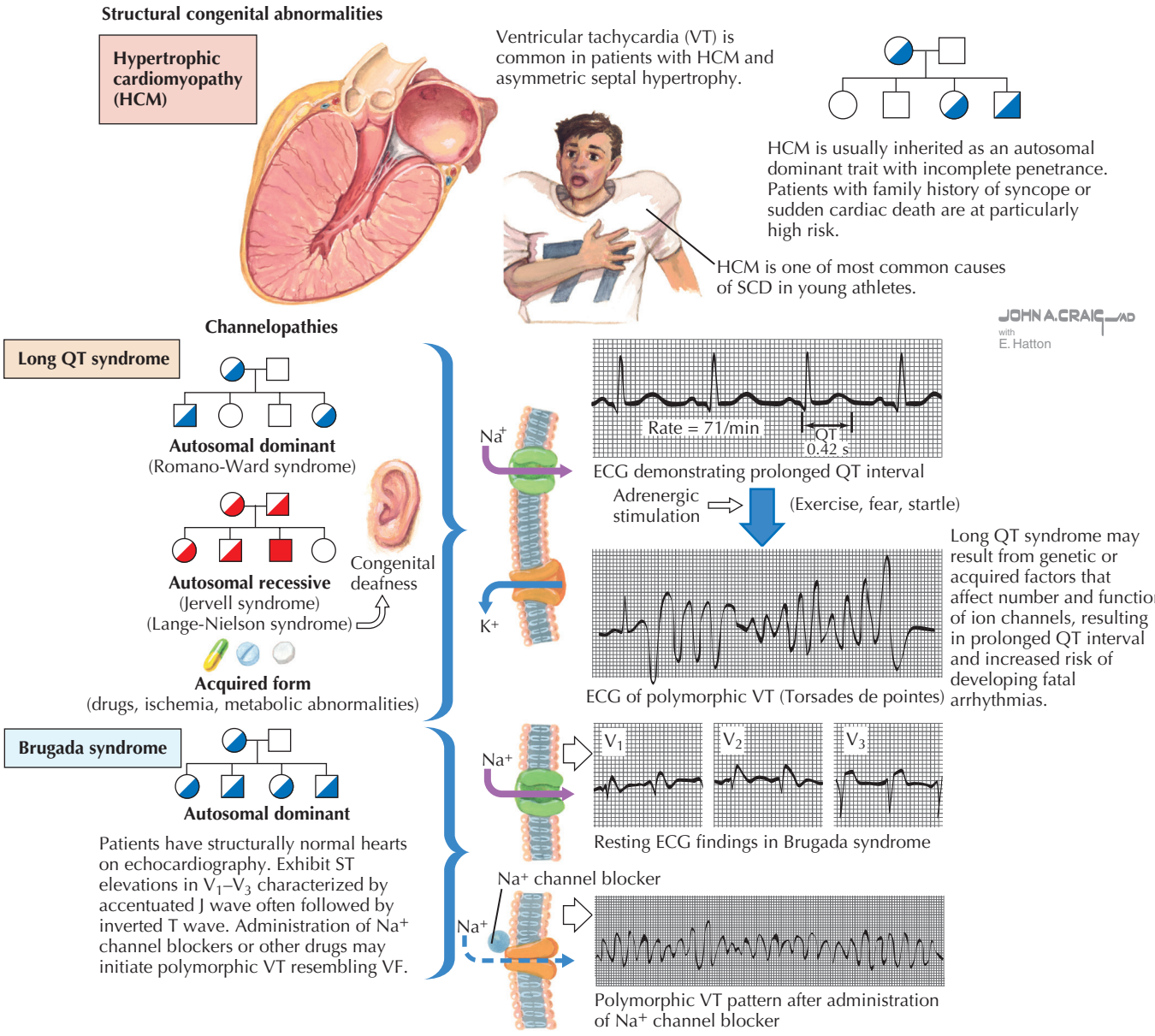


**Figure 3.1.** Sudden cardiac death.

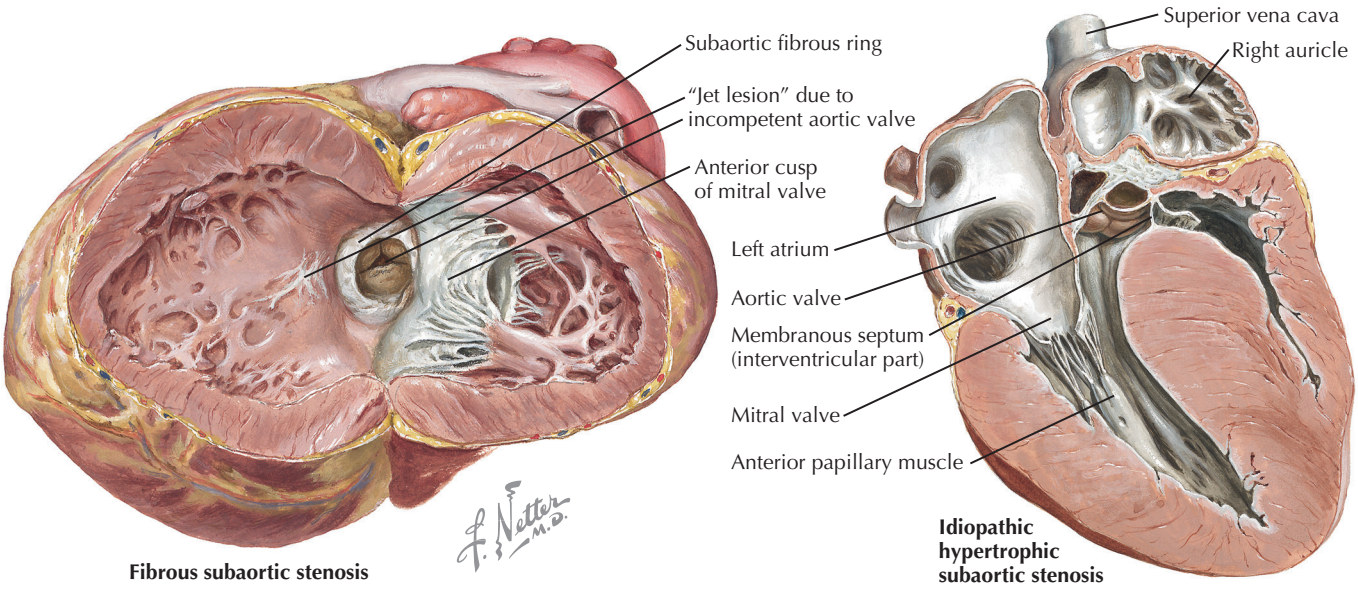


**Figure 3.2.** Hypertrophic cardiomyopathy.

## Head, Eyes, Ears, Nose, and Throat (HEENT)

- Optical examination is important: check visual acuity in all athletes, pupils for anisocoria, conjunctiva for anemia.
- Certain athletes may have predilection for ear issues (e.g., swimmers [otitis externa], scuba divers [otic barotrauma], and wrestlers [auricular hematoma]).
- Allergy sufferers and athletes with history of nose trauma need nasopharyngeal examinations.
- Smokeless tobacco users need oropharyngeal examinations.

## Cardiovascular Assessment

Cardiovascular assessment is essential for both initial PPPE and annual reevaluations (see Box 3.1).

### BOX 3.1 STANDARD COMPONENTS OF THE PREPARTICIPATION PHYSICAL EVALUATION

- Height
- Weight
- Eyes: visual acuity and pupil size
- Oral cavity
- Ears
- Nose
- Lungs
- Cardiovascular system: blood pressure, femoral and radial pulses, and heart rate, rhythm, and murmurs
- Abdomen: masses, tenderness, and organomegaly
- Skin: rashes and lesions (infectious)
- Musculoskeletal system: contour, range of motion, and symmetry of neck, back, shoulder/arm, elbow/forearm, wrist/hand, hip/thigh, knee, leg/ankle, and foot

Modified from *Preparticipation Physical Evaluation,* 4th ed. Elk Grove Village, Ill.: American Academy of Pediatrics; 2010.

**Brachial artery blood pressure measurement (with appropriate cuff size and ideally in both arms):** if elevated, recheck after the athlete rests quietly for 15 minutes and later, if needed (see Table 3.3). The following are classification categories of hypertension in children and adolescents (see www.nhlbi.nih.gov/guidelines/hypertension/hbp_ped.htm):
- Normal (<90th percentile for age, sex, and height)
- High-normal (90th–94th percentile for age, sex, and height)
- Hypertension (95th–99th percentile for age, sex, and height)
- Severe hypertension (>99th percentile for age, sex, and height)

**Palpate radial and femoral pulses:**
- Decreased or nonpalpable femoral pulses should raise suspicion for coarctation of aorta.
- Irregular pulse should raise suspicion for arrhythmia and requires ECG evaluation.

**Heart auscultation in supine and standing positions:** note the presence and character of any murmurs
- **HCM murmur:** systolic murmur heard best at lower left sternal border in the standing position; increases with maneuvers that decrease venous return to the heart
- Provocative maneuvers help to differentiate functional murmurs from pathologic murmurs.
  - To decrease venous return: Valsalva maneuver and squat-to-stand
  - To increase venous return: deep inspiration, stand-to-squat, and isometric hand grip
- General recommendations for murmurs requiring further evaluation before the athlete can participate:
  - Any systolic murmur grade ≥III/VI in severity
  - Any diastolic murmur
  - Any murmur that gets louder with Valsalva maneuver

**Marfan syndrome stigmata** (Fig. 3.3): Tall stature, arachnodactyly, kyphoscoliosis, anterior chest deformity, arm span greater than height, decreased upper body length to lower body length ratio, heart murmur or midsystolic click, ectopic lens, thumb sign (the thumbs protrude from the clenched fists), wrist sign

### TABLE 3.3 CLASSIFICATION OF HYPERTENSION

| Age and Phase | 90th–94th Percentile† High Normal* Prehypertensive‡ | 95th–99th Percentile† Significant HTN* Stage 1 HTN‡ | >99th Percentile† Severe HTN* Stage 2 HTN‡ |
|---|---|---|---|
| **6–9 y** | | | |
| Systolic† | 104–121 | 108–129 | >115–129 |
| Diastolic† | 68–81 | 72–89 | >83–89 |
| **10–12 y** | | | |
| Systolic† | 112–127 | 116–135 | >123–135 |
| Diastolic† | 73–83 | 77–91 | >84–91 |
| **13–15 y** | | | |
| Systolic† | 117–135 | 121–142 | >128–142 |
| Diastolic† | 76–85 | 80–93 | >87–93 |
| **16–17 y** | | | |
| Systolic† | 121–140 | 125–147 | >132–147 |
| Diastolic† | 78–89 | 82–97 | >90–97 |
| **≥18 y** | | | |
| Systolic‡ | 120–139 | 140–159 | ≥160 |
| Diastolic‡ | 80–89 | 90–99 | ≥100 |

*HTN,* Hypertension.

*American Academy of Pediatrics, Task Force on Blood Pressure in Children: Report of the Second Task Force Control on Blood Pressure in Children—1987. *Pediatrics.* 1987;79:1-25. Updated in 1996;98(4 Pt 1):649-658.

†The BP range is obtained from the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. For specific BP levels by age, gender, and height, see http://www.nhlbi.nih.gov/files/docs/guidelines/child_tbl.pdf.

‡From the Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure, 2003.

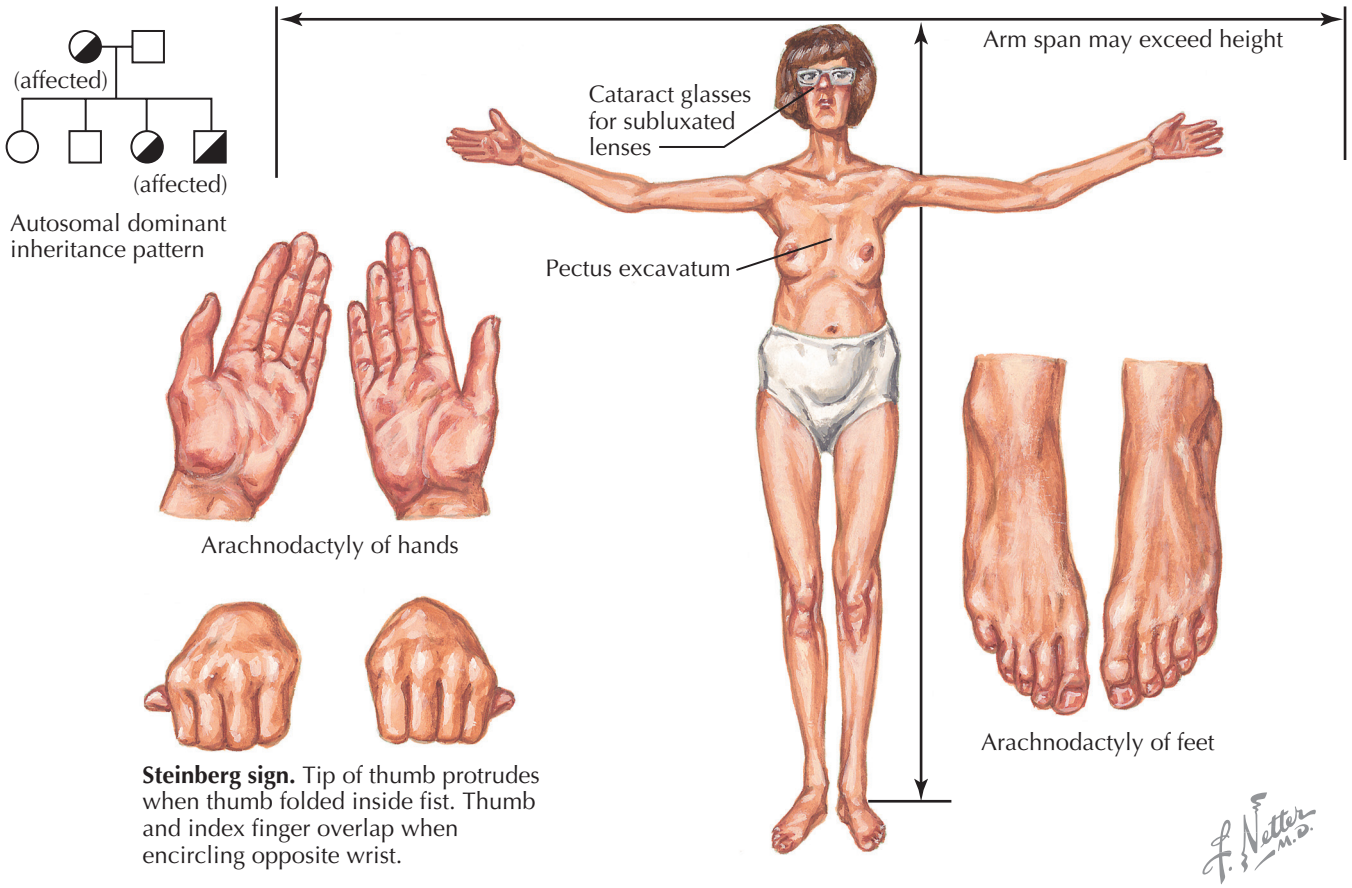


**Figure 3.3.** Marfan syndrome.

(the distal phalanges of the first and fifth digits of one hand overlap when wrapped around the opposite wrist), and family history of Marfan syndrome

## Pulmonary Assessment

- Focus on detecting abnormal breath sounds: wheezes, crackles, rubs, and abnormal inspiratory to expiratory ratio
- Asthma screening (e.g., exercise challenge test) has been suggested but is impractical in most preparticipation settings.

## Abdominal/Gastrointestinal Assessment

Should be performed with athlete in the supine position. Examples of problems requiring further evaluation before participation include organomegaly (liver and spleen), masses, bruits, tenderness, and/or rigidity and possible pregnancy in females.

## Genitourinary Assessment

- Male: The 4th PPPE Monograph recommends routine male genital examinations, but there is insufficient evidence for or against screening genital examinations for boys playing sports. If warranted by history or other findings, examine in a private setting looking for undescended testes, absence of testicle, hernia, or mass.
- Female: routine examination is not recommended; if warranted by history or other findings, examine in a private setting
- Tanner staging is no longer recommended in PPPE monograph because its use for injury prevention and psychological benefits is controversial; however, it may be useful for counseling on growth and development in boys aged 11–17 years.

## Musculoskeletal Examination

- Musculoskeletal examination is important to identify musculotendinous, bone, or joint problems that may limit athletic participation or predispose to acute injury or long-term complications (e.g., shoulder instability, anterior cruciate-deficient knee, unrehabilitated ankle sprain, or juvenile rheumatoid arthritis).
- General screening examination is most efficient for asymptomatic athletes with no prior musculoskeletal injuries (Fig. 3.4).
- Joint-specific examination: recommended for problematic areas; most accurate and most time consuming
- Back flexion is recommended in screening for thoracolumbar deformities such as scoliosis (Fig. 3.5).
- Sport-specific examination: some advocate need for focusing on commonly injured or stressed areas in particular sports (e.g., shoulder examination for throwers, tennis players, and swimmers and knee examination for basketball, football, and soccer players); also includes measures of endurance, strength, and flexibility in orthopedic screening but is time consuming and requires in-depth knowledge of particular sports
- Consider screening for flexibility (e.g., back, hamstrings, and Achilles tendon) because clinical anecdotal evidence suggests that increasing flexibility reduces risk of overuse problems (e.g., mechanical back pain, patellofemoral pain, and medial tibial stress syndrome); however, no study has supported a decreased risk of acute injury (e.g., sprains, strains, and dislocations).

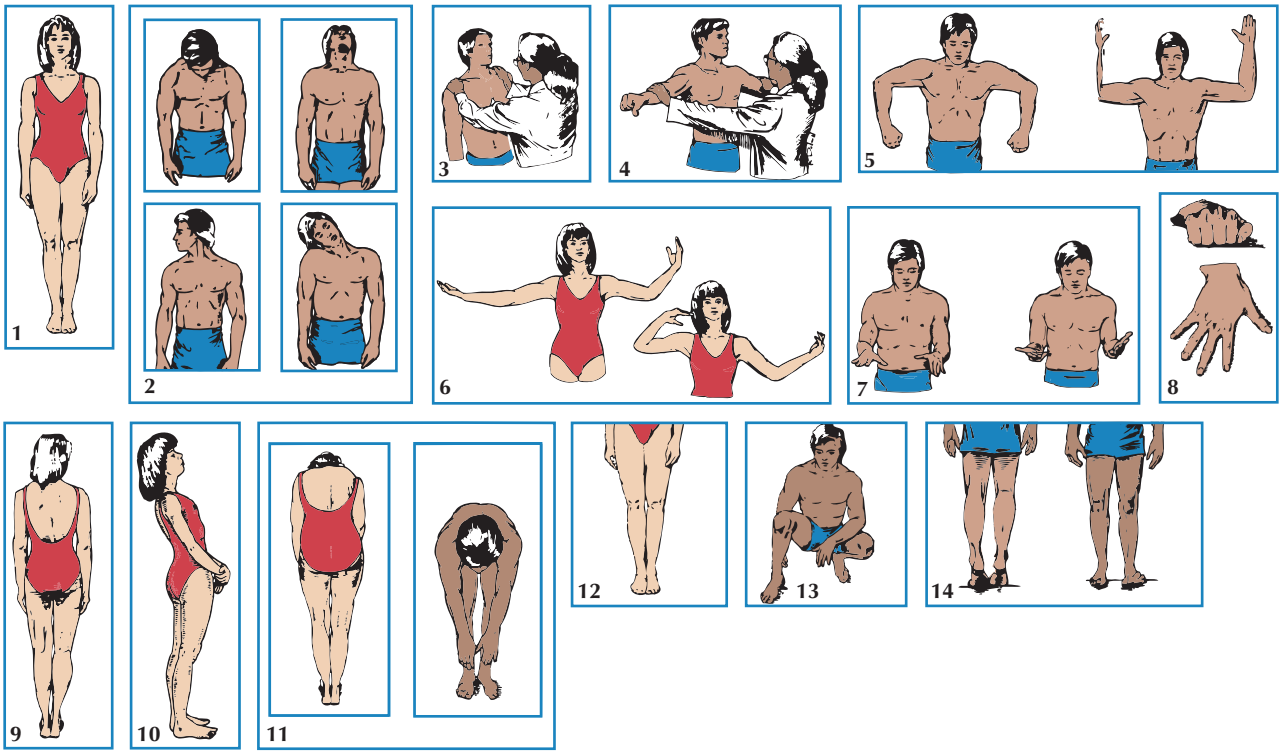


**Figure 3.4.** General musculoskeletal screening examination comprising the following: (1) inspection, athlete standing and facing the examiner (symmetry of trunk, upper extremities); (2) forward flexion, extension, rotation, and lateral flexion of neck (range of motion, cervical spine); (3) resisted shoulder shrug (strength, trapezius); (4) resisted shoulder abduction (strength, deltoid); (5) internal and external rotation of shoulder (range of motion, glenohumeral joint); (6) extension and flexion of elbow (range of motion, elbow); (7) pronation and supination of elbow (range of motion, elbow and wrist); (8) clench fist and then spread fingers (range of motion, hand and fingers); (9) inspection, athlete facing away from the examiner (symmetry of trunk, upper extremities); (10) back extension with knees straight (spondylolysis/spondylolisthesis); (11) back flexion with knees straight, facing toward and away from the examiner (range of motion, thoracic and lumbosacral spine; spine curvature; hamstring flexibility); (12) inspection of lower extremities, contraction of quadriceps muscles (alignment, symmetry); (13) "duck walk" four steps (motion of hip, knee, and ankle; strength, balance); (14) standing on toes and then on heels (symmetry, calf; strength; balance). (© Rebekah Dodson.)

- Functional Movement Screening (FMS) may be added to PPPE to assess movement quality based on the sport, accessibility, and available resources.

### Neurologic Assessment

Neurologic assessment (gross motor) is generally performed through musculoskeletal evaluation. Perform a more comprehensive neurologic examination in athletes with unexplained strength deficits, paresthesia, history of burners/stingers, history of head injury/concussion, or any focal or generalized neurologic deficit.

- NCAA and others have recommended baseline symptom score, cognitive examination, and balance assessment for all athletes; an example of this is the Sideline Concussion Assessment Tool-3 (SCAT-3).
- Consider baseline assessment of vestibular ocular motor movement screening (VOMS) for athletes participating in contact and collision sports.

### Other Assessment

Assessment for other problems such as lymphadenopathy, thyromegaly, physical findings of eating disorders, and skin conditions should be considered on an individual basis.

### Fitness and Performance Evaluation

- Secondary (ideal) objective of PPPE
- Performed more often in the group screening-station format
- Measures any or all of the following parameters:
  - Body composition (e.g., skinfold, underwater weighing, and circumferences)
  - Flexibility (e.g., sit-and-reach and goniometry)
  - Strength (e.g., manual muscle testing, hand or leg dynamometer, bench press or leg press, push-ups, pull-ups, or sit-ups)
  - Endurance (e.g., 12-minute and 1.5-mile run)
  - Power (e.g., vertical and standing broad jump)
  - Speed (e.g., 40-yard dash) and agility (e.g., agility run)
  - Balance (e.g., stork stand, balance beam walking, and Balance Error Scoring System [BESS] included in SCAT-3)
- Vision performance testing and/or VOMS has been added in certain settings:
  - Dynamic visual acuity
  - Depth perception
  - Visual tracking or pursuit
  - Eye–hand and eye–body coordination

## SCREENING TESTS

### Difference Between Routine Screening and Diagnostic Testing

A complete blood count in an asymptomatic female athlete is a screening test, whereas a complete blood count in a female athlete with poor eating habits, heavy menstrual periods, fatigue, and pale conjunctiva becomes a diagnostic test.

- Does the burden of suffering resulting from the condition warrant screening?

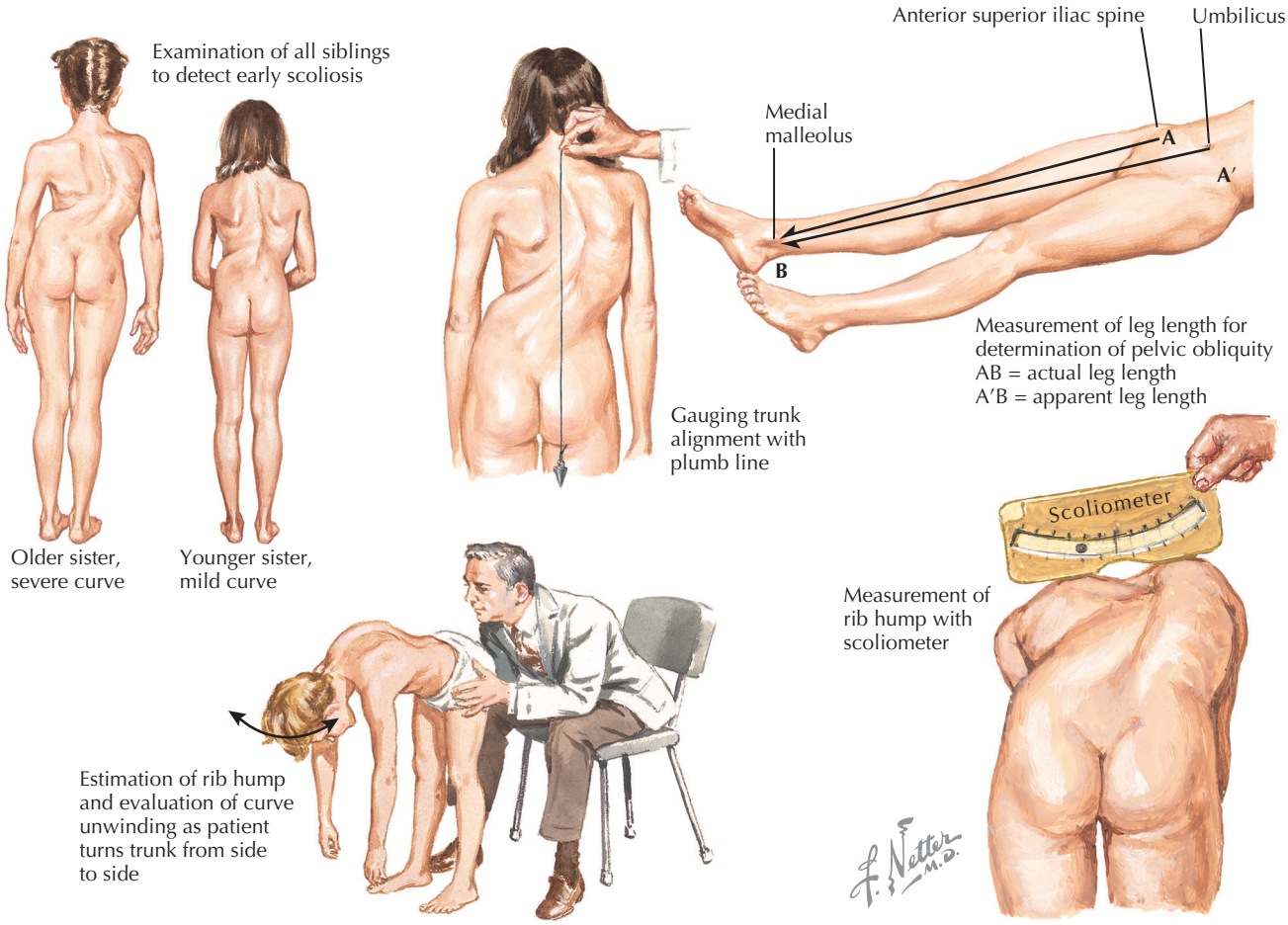


**Figure 3.5.** Clinical evaluation of scoliosis.

- If the answer is "yes," ask the following:
  - What is the sensitivity of the proposed screening test?
  - Are the potential risks and cost of the test acceptable?
  - If the screening test identifies the condition, are proven and acceptable treatments available? Is there a clear advantage of initiating such treatment during the asymptomatic phase of the condition?

## Routine Screening Tests

Routine screening is not recommended, but specific screening tests (e.g., sickle cell screening required by NCAA and hemoglobin and ferritin for distance runner) are recommended as indicated.

**Laboratory tests:** urinalysis, complete blood count, chemistry profile, lipid profile, ferritin, sickle cell trait, sexually transmitted disease, infectious hepatitis, and urine drug screening

**Radiographs:** chest, cervical spine, and joint radiographs

**Cardiopulmonary tests:** ECG, echocardiogram, exercise stress test, spirometry, exercise spirometry, and other exercise challenge tests. Although certain studies suggest that using new ECG screening protocols (e.g., Seattle criteria) may decrease false-positive screens, recommendation of ECG screening for athletes still remains controversial, even in select populations. In 2014, the AHA published *Assessment of the 12-Lead ECG as a Screening Test for Detection of Cardiovascular Disease in Healthy General Populations of Young People (12–25 Years of Age)*. It states:

- Screening with 12-lead ECG in association with comprehensive history-taking and physical examination to identify or raise suspicion of genetic/congenital and other cardiovascular abnormalities may be considered in relatively small cohorts of young healthy people aged 12–25 years but not necessarily limited to athletes (e.g., in high schools, colleges/universities, or local communities), provided that close physician involvement and sufficient quality control can be achieved; if undertaken, such initiatives should recognize known and anticipated limitations of 12-lead ECG as a population screening test, including the expected frequency of false-positive and false-negative test results, as well as the cost required to support these initiatives over time (Class IIb; Level of Evidence C).
- Mandatory and universal mass screening with 12-lead ECG in large general populations of young healthy people aged 12–25 years (including on a national basis in the United States) to identify genetic/congenital and other cardiovascular abnormalities is not recommended for athletes and nonathletes alike (Class III, no evidence of benefit; Level of Evidence C).

## The "Italian Experience"

In Italy, a systematic, state-subsidized national program for mandatory annual PPPE of all athletes aged 12–35 years has been in place for approximately 35 years. Minimum annual tests include a general examination and 12-lead ECG. Elite competitive athletes undergo a more comprehensive medical and physiological evaluation that includes routine ECG, findings of which are as follows:

- Detection of few definitive examples of potentially lethal cardiovascular abnormalities remains the norm.

- 2.2%–2.5% of athletes are disqualified (approximately 51% because of cardiovascular abnormalities).
- A study by Basso and colleagues suggests that the Italian national screening program can decrease the incidence of SCD among young athletes. During the study period, SCD occurred in 55 screened athletes versus 265 nonscreened nonathletes.
- Right ventricular dysplasia causes more athletic deaths than HCM. Reasons for the discrepancy in North American data (where HCM causes more deaths in athletes aged <30 years) are unresolved. In addition, possible disqualification of athletes through screening may contribute to this discrepancy.

With the rarity of potentially lethal cardiovascular abnormalities in young athletes and the overwhelming number of sports and athletic participants in the United States, screening of the Italian magnitude would be challenging in most settings.

### ESC and IOC

The European Society of Cardiology (ESC) and International Olympic Committee (IOC) recommend combining noninvasive testing (e.g., 12-lead ECG) with the standard history-taking and physical examination for cardiovascular screening in large populations of young trained athletes.

### ECG and/or Echocardiogram

ECG should be considered in athletes with any significant cardiac symptoms or abnormal findings on examination or with a family history of sudden death (unknown cause), SCD, or other cardiac conditions that are known predisposing factors for SCD (e.g., right ventricular dysplasia, HCM, long QT syndrome, and Marfan syndrome) in a family member aged <50 years, particularly a first-degree relative. Approximately 90% of people with HCM exhibit abnormal ECG findings.

### Baseline Neuropsychological (NP) Testing

Baseline NP testing may be considered and recommended, if available, for athletes in sports considered to have a risk of head injury. The utility of neuropsychological testing as a stand-alone test as well as the need for baseline testing remains controversial. Nevertheless, baseline NP testing is valuable as part of a comprehensive concussion protocol injury (see Chapter 45: Head Injuries).

### Patients With Disabilities

The PPPE and clearance to participate for athletes with disabilities is important and should be performed with an understanding of the medical and musculoskeletal issues that are present for the condition as well as understanding the sport-specific demands for the athlete (Preparticipation Physical Evaluation: The athlete with special needs form available at https://www.aap.org/en-us/professional-resources/practice-support/Documents/Preparticipation-Physical-Exam-Form.pdf, accessed March 2016; for additional information related to athletes with disability, see Chapter 14: The Physically Challenged Athlete). A common example is athletes with Down syndrome, with possible atlantoaxial instability.

#### Down Syndrome

Patients with Down syndrome and their parents should be closely questioned regarding signs or symptoms of atlantoaxial instability. Cervical radiographs, including flexion and extension views, may be considered (Fig. 3.6).

- In asymptomatic patients, neurologic signs or symptoms may be more predictive of risk of injury progression than radiographic abnormalities.
- The American Academy of Pediatrics acknowledges potential but unproven value of lateral plain radiographs of cervical spine but does not recommend routine screening radiographs.
- Special Olympics requires cervical spine radiographs before athletic participation in all patients with Down syndrome participating in judo, equestrian sports, gymnastics, diving, pentathlon, butterfly stroke, and diving starts in swimming, high jump, Alpine skiing, snowboarding, squat lift, and soccer.
- However, there is a lack of general agreement on the criteria for exclusion from sport.

## CLEARANCE FOR PARTICIPATION

- Clearance falls into **four categories:**
  1. Full participation without restrictions
  2. Participation pending further testing/evaluation
  3. Participation just in certain sports
  4. Disqualification
- Differentiation of categories is important.
- Familiarity with demands of a specific sport is essential; use of classification system for sports by contact and strenuousness is helpful in this regard (Table 3.4 and Fig. 3.7).
- **Published guidelines for medical conditions and sports participation** are helpful, but clinical judgment should be used in applying general guidelines to individual athletes (Table 3.5).
- Additional considerations:
  - How does the condition/illness affect the athlete's risk of morbidity or mortality?

**TABLE 3.4 SPORTS ACCORDING TO RISK OF IMPACT AND EDUCATIONAL BACKGROUND**

| | Junior High School | High School/College |
|---|---|---|
| **Impact Expected** | American football<br>Ice hockey<br>Lacrosse<br>Wrestling<br>Karate/judo<br>Fencing<br>Boxing | American football<br>Soccer<br>Ice hockey<br>Lacrosse<br>Basketball<br>Wrestling<br>Karate/judo<br>Downhill skiing<br>Squash<br>Fencing<br>Boxing |
| **Impact May Occur** | Soccer<br>Basketball<br>Field hockey<br>Downhill skiing<br>Equestrian<br>Squash<br>Cycling | Field hockey<br>Equestrian<br>Cycling<br>Baseball/softball<br>Gymnastics<br>Figure skating |
| **Impact Not Expected** | Baseball/softball<br>Cricket<br>Golf<br>Riflery<br>Gymnastics<br>Volleyball<br>Swimming<br>Track and field<br>Tennis<br>Figure skating<br>Cross-country skiing<br>Rowing<br>Sailing<br>Archery<br>Weightlifting<br>Badminton | Cricket<br>Golf<br>Riflery<br>Volleyball<br>Swimming<br>Track and field<br>Tennis<br>Cross-country skiing<br>Rowing<br>Sailing<br>Archery<br>Weightlifting<br>Badminton |

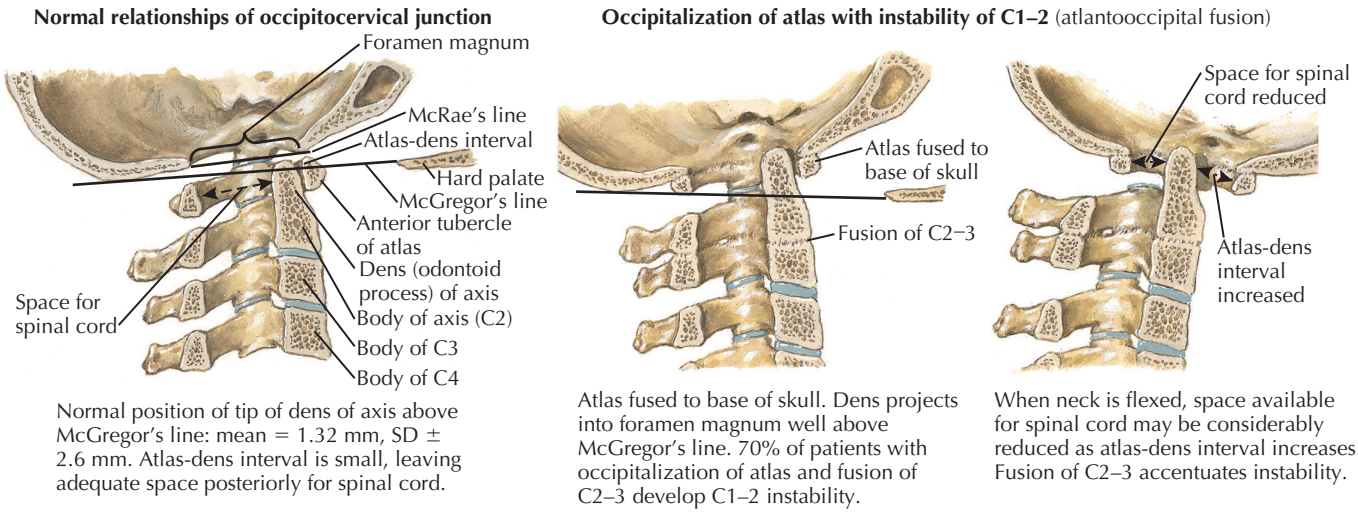


**Figure 3.6.** Congenital anomalies of occipitocervical junction.

- How will the condition/illness affect other participants?
- Are there any limitations or modifications within the sport that allow the athlete to continue participation despite the injury or illness? If so, is it reasonable to allow participation with limitations until the condition resolves?

- Most athletes are cleared for full participation without restriction or with minimal additional evaluation (e.g., reassessment of visual acuity, blood pressure, or ligamentous instability; correction of improper rehabilitation; and additional musculoskeletal consultation).
- Explain reasons for further evaluation to athlete and parent/guardian (if athlete is <18 years old).
- Extensive updated information about a medical condition and the risk of participation often requires attention to specifics of the medical problem and the individual athlete as well as a breakdown of sport-specific requirements.
- Decisions to disqualify may require additional specialist consultations as well as one or two "second expert opinions." A total of three opinions is suggested.

## AREAS OF CONCERN

### Ethical Issues Associated With PPPE

Ethical issues to consider as a team physician include:

- Confidentiality (e.g., compliance with Health Insurance Portability and Accountability Act [HIPAA], Family Educational Rights and Privacy Act [FERPA], disclosing medical information, and drug testing results)
- Informed consent (e.g., discussion of all treatment options and balancing athletes' desire to participate versus providing optimal medical treatment)
- Influence of third parties (e.g., pressure from teammates, coaches, administrators, parents/guardians, or other people from the community)
- Drug use (e.g., using pain medications to allow participation; pressure to supply, administer, or provide counsel regarding illegal; and illicit or performance-enhancing drugs)

### Medicolegal Issues Associated With PPPE

**Right to participate:**

- Under enactments such as the Americans with Disabilities Act and the Federal Rehabilitation Act, an athlete may have a legal right to participate against medical advice.
- If athletes choose to participate against medical advice, **an exculpatory waiver** or **prospective release** is highly recommended. Despite questions of validity, these forms of written contracts are intended to demonstrate that the athlete was fully informed of his or her condition as well as the potential risks of participation. "Guidelines" for various medical conditions (e.g., Concussion Guidelines and AHA/American College guidelines [formerly Bethesda Guidelines]) "are not intended to establish absolute mandates" and are better considered as recommendations for the team physician to consider when providing clearance decisions. Occasionally, these guidelines can provide support for the decision to restrict participation, and in others, the team physician may deviate from the recommendations based on individualized factors that might allow an athlete to participate. It is important that the physician fully informs the athlete of the potential risks and has it acknowledged in a

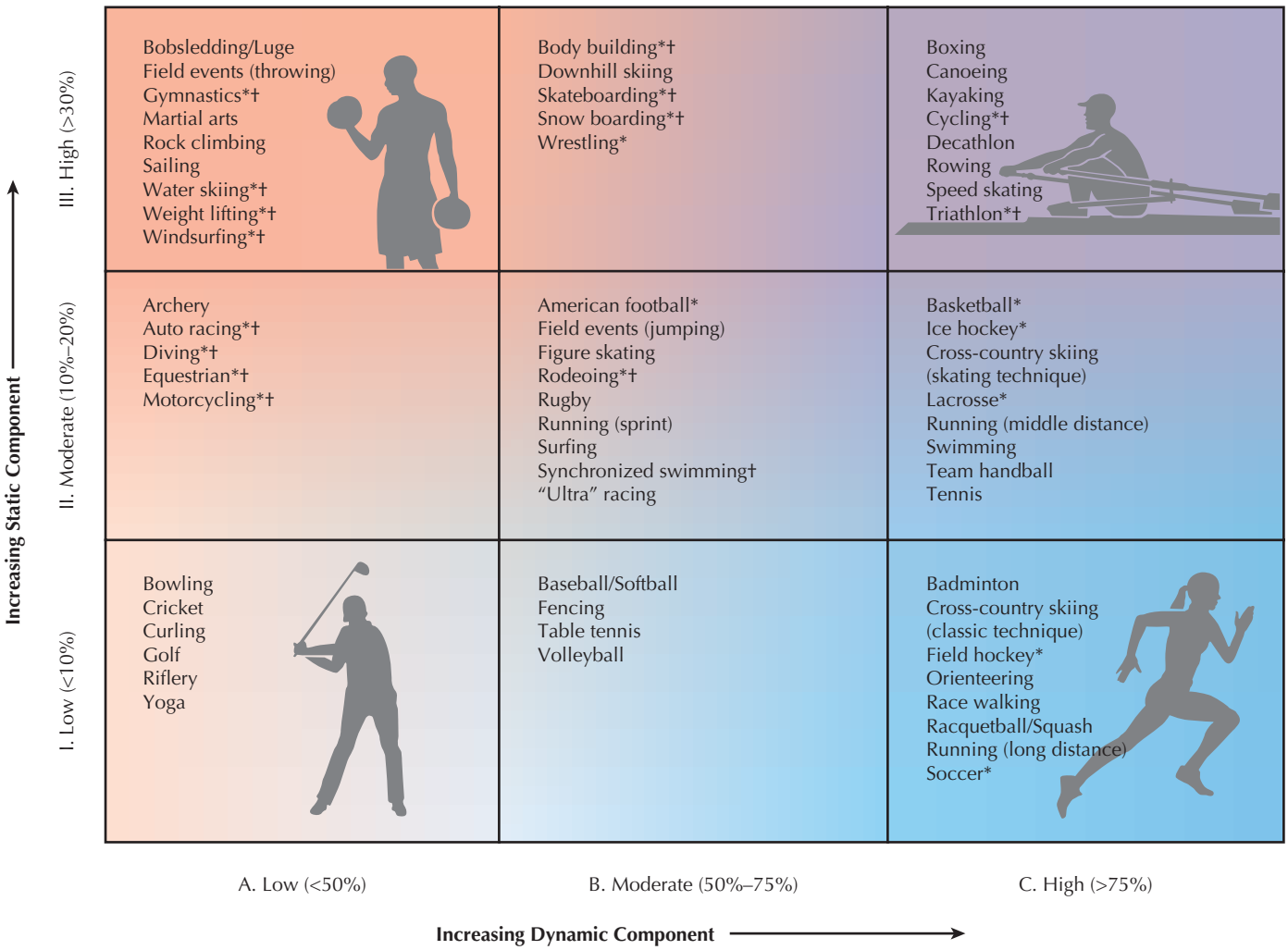


**Figure 3.7.** Classification of sports based on peak static and dynamic components achieved during competition; however, higher values may be reached during training. The increasing dynamic component is defined in terms of the estimated percentage of maximal oxygen uptake ($\dot{V}O_2max$) achieved and results in an increasing cardiac output. The increasing static component is related to the estimated percentage of maximal voluntary contraction reached and results in an increasing blood pressure load. The lowest total cardiovascular demands (cardiac output and blood pressure) are shown in the palest color, with increasing dynamic load depicted by increasing blue intensity and increasing static load by increasing red intensity. Note the graded transition between categories, which should be individualized on the basis of player position and style of play. *Danger of bodily collision (see Table 3.4 for more detail on collision risk). †Increased risk if syncope occurs. (Modified from Mitchell et al. with permission. Copyright © 2005, Journal of the American College of Cardiology. From Recommendations for Competitive Athletes with Cardiovascular Abnormalities: Task Force 1: Classification of Sports: Dynamic, Static, and Impact: A Scientific Statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132[22]:e262-266.)

## TABLE 3.5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION

| Condition | May Participate |
|---|---|
| **Atlantoaxial Instability (Joint Instability Between Cervical Vertebrae 1 and 2)**<br>*Explanation:* Athlete needs evaluation to assess risk of spinal cord injury during sports participation, particularly while using a trampoline | Qualified yes |
| **Bleeding Disorder**<br>*Explanation:* Athlete needs evaluation | Qualified yes |
| **Cardiovascular Diseases** | |
| **Carditis (Inflammation of the Heart)**<br>*Explanation:* Carditis may result in sudden death with exertion | No |
| **Hypertension (High Blood Pressure)**<br>*Explanation:* Those with severe hypertension (>99$^{th}$ percentile for age plus 5 mm Hg) should avoid heavy weight and power lifting, body building, strength training, and high-static component sports. Those with sustained hypertension (>95$^{th}$ percentile for age) need evaluation. | Qualified yes |

**TABLE 3.5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION—cont'd**

| Condition | May Participate |
|---|---|
| **Congenital Heart Disease (Structural Heart Defects Present at Birth)**<br>*Explanation:* Consultation with a cardiologist is recommended. Those with mild forms may participate fully, while those with moderate or severe forms or who have undergone surgery need evaluation. | Qualified yes |
| **Dysrhythmia (Irregular Heart Rhythm-Long-QT Syndrome, Malignant Ventricular Arrhythmias, Symptomatic Wolff–Parkinson–White Syndrome, Advanced Heart Block, Family History of Sudden Death or Previous Sudden Cardiac Event, or Implantation of a Cardioverter-Defibrillator)**<br>*Explanation:* Consultation with a cardiologist is advised. Those with symptoms (chest pain, syncope, near-syncope, dizziness, shortness of breath, or other symptoms of possible dysrhythmia) or evidence of mitral regurgitation on physical examination need evaluation. All others may participate fully. | Qualified yes |
| **Heart Murmur**<br>*Explanation:* If the murmur is innocent (does not indicate heart disease), full participation is permitted. Otherwise, athlete needs evaluation (see Structural Heart Disease, particularly hypertrophic cardiomyopathy and mitral valve prolapse). | Qualified yes |
| **Structural/Acquired Heart Disease**<br>*Hypertrophic cardiomyopathy*<br>*Coronary artery anomalies*<br>*Arrhythmogenic right ventricular cardiomyopathy*<br>*Acute rheumatic fever with carditis*<br>*Ehlers–Danlos syndrome, vascular form*<br>*Marfan syndrome*<br>*Mitral valve prolapse*<br>*Anthracycline use*<br>*Explanation:* Consultation with a cardiologist is recommended. Most of these conditions carry a significant risk of sudden cardiac death associated with intense physical exercise. Hypertrophic cardiomyopathy requires thorough and repeated evaluations because disease manifestations may change during later adolescence. Marfan syndrome with an aortic aneurysm also can cause sudden death during intense physical exercise. Athlete who has received chemotherapy with anthracyclines may be at an increased risk of cardiac problems because of cardiotoxic effects of such medications, and resistance training in this population should be approached with caution; strength training that avoids isometric contractions may be permitted. Athlete needs evaluation. | Qualified no<br>Qualified no<br>Qualified no<br>Qualified no<br>Qualified no<br>Qualified yes<br>Qualified yes<br>Qualified yes |
| **Vasculitis/Vascular Disease (Kawasaki Disease or Pulmonary Hypertension)**<br>*Explanation:* Consultation with a cardiologist is recommended. Athlete needs individual evaluation to assess risk on the basis of disease activity, pathologic changes, and medical regimen. | Qualified yes |
| **Cerebral Palsy**<br>*Explanation:* Athlete needs evaluation to assess functional capacity to perform sports-specific activities. | Qualified yes |
| **Diabetes Mellitus**<br>*Explanation:* All sports can be played with appropriate attention and adjustments to diet (particularly carbohydrate intake), blood glucose concentrations, hydration, and insulin therapy. Blood glucose concentrations should be monitored before exercise, every 30 min during continuous exercise, 15 min after completion of exercise, and at bedtime. | Yes |
| **Diarrhea (Infectious)**<br>*Explanation:* Unless symptoms are mild and athlete is fully hydrated, no participation is permitted because diarrhea may increase the risk of dehydration and heat illness. See Fever below. | Qualified no |
| **Eating Disorders**<br>*Explanation:* Athlete with an eating disorder needs medical and psychiatric assessment before participation. | Qualified yes |
| **Eyes**<br>Functionally one-eyed athlete, loss of an eye, detached retina, high myopia, connective tissue disorder, previous eye surgery, or serious eye injury<br>*Explanation:* A functionally one-eyed athlete is defined as having best-corrected visual acuity worse than 20/40 in the poorer-seeing eye. Such an athlete would suffer considerable disability if the better eye were seriously injured as would an athlete with loss of an eye. Specifically, boxing and full-contact martial arts are not recommended for functionally one-eyed athletes because eye protection is impractical and/or not permitted. Some athletes who previously underwent intraocular eye surgery or had a serious eye injury may be at an increased risk of injury because of weakened eye tissue. Availability of eye guards approved by the American Society for Testing and Materials and other protective equipment may allow participation in most sports, but this must be judged on an individual basis. | Qualified yes |
| **Conjunctivitis (Infectious)**<br>*Explanation:* Athlete with active infectious conjunctivitis should be excluded from swimming. | Qualified no |
| **Fever**<br>*Explanation:* Fever can increase cardiopulmonary effort, reduce maximum exercise capacity, increase the risk of heat illness, and increase orthostatic hypotension during exercise. Fever may rarely accompany myocarditis or other infections that may make exercise dangerous. | No |

*Continued*

## TABLE 3.5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION—cont'd

| Condition | May Participate |
|---|---|
| **Gastrointestinal**<br>**Malabsorption Syndromes (Celiac Disease or Cystic Fibrosis)**<br>*Explanation*: Athlete needs individual assessment for general malnutrition or specific deficits resulting in coagulation or other defects; with appropriate treatment, these deficits can be adequately treated to permit normal activities. | Qualified yes |
| **Short-Bowel Syndrome or Other Disorders Requiring Specialized Nutritional Support, Including Parenteral or Enteral Nutrition**<br>*Explanation*: Athlete needs individual assessment for collision, contact, or limited-contact sports. Presence of central or peripheral, indwelling venous catheter may require special considerations for activities and emergency preparedness for any unexpected trauma to the device(s). | |
| **Heat Illness (With History)**<br>*Explanation:* Because of the likelihood of recurrence, athlete needs individual assessment to determine the presence of predisposing conditions and behaviors and to develop a prevention strategy that includes sufficient acclimatization (to the environment and to exercise intensity and duration), conditioning, hydration, and salt intake as well as other effective measures to improve heat tolerance and reduce heat injury risk (e.g., protective equipment and uniform configurations). | Qualified yes |
| **Hepatitis (Primarily Hepatitis C) and HIV Infection**<br>*Explanation:* Because of the apparent minimal risk to others, all sports may be played based on the athlete's health status. In all athletes, skin lesions should be appropriately covered, and athletic personnel should use universal precautions when handling blood or body fluids with visible blood. However, certain sports (such as wrestling and boxing) may create a situation that favors viral transmission (likely bleeding plus skin breaks). If viral load is detectable, then athletes should be advised to avoid such high-contact sports. | Yes |
| **Kidney: Absence of One**<br>*Explanation:* Athlete needs individual assessment for contact, collision, and limited-contact sports. Protective equipment may sufficiently reduce the risk of injury to the remaining kidney to allow participation in most sports, providing such equipment remains in place during activity. | Qualified yes |
| **Liver: Enlarged**<br>*Explanation:* If the liver is acutely enlarged, then participation should be avoided because of the risk of rupture. If the liver is chronically enlarged, then individual assessment is needed before participation in collision, contact, or limited-contact sports. Patients with chronic liver disease may have changes in liver function that affect stamina, mental status, coagulation, or nutritional status. | Qualified yes |
| **Malignant Neoplasm**<br>*Explanation:* Athlete needs individual assessment. | Qualified yes |
| **Musculoskeletal Disorders**<br>*Explanation:* Athlete needs individual assessment. | Qualified yes |
| **Neurologic**<br>**History of Serious Head or Spine Trauma or Abnormality, Including Craniotomy, Epidural Bleeding, Subdural Hematoma, Intracerebral Hemorrhage, Second-Impact Syndrome, Vascular Malformation, and Neck Fracture**<br>*Explanation:* Athlete needs individual assessment for collision, contact, or limited-contact sports. | Qualified yes |
| **History of Simple Concussion (Mild Traumatic Brain Injury), Multiple Simple Concussions, and/or Complex Concussion**<br>*Explanation*: Athlete needs individual assessment. Research supports a conservative approach for concussion management, including no athletic participation while symptomatic or when deficits in judgment or cognition are detected, followed by graduated return to full activity. | Qualified yes |
| **Myopathies**<br>*Explanation*: Athlete needs individual assessment. | Qualified yes |
| **Recurrent Headaches**<br>*Explanation*: Athlete needs individual assessment. | Yes |
| **Recurrent Plexopathy (Burner or Stinger) and Cervical Cord Neuropraxia With Persistent Defects**<br>*Explanation*: Athlete needs individual assessment for collision, contact, or limited-contact sports; regaining normal strength is an important benchmark for clearance to return to play. | Qualified yes |
| **Seizure Disorder, Well Controlled**<br>*Explanation:* Risk of convulsion during participation is minimal. | Yes |
| **Seizure Disorder, Poorly Controlled**<br>*Explanation:* Athlete needs individual assessment for collision, contact, or limited-contact sports. The following noncontact sports should be avoided: archery, riflery, swimming, weightlifting, power lifting, strength training, and sports involving heights. In these sports, occurrence of a seizure during activity may pose a risk to self or others. | Qualified yes |

**TABLE 3.5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION—cont'd**

| Condition | May Participate |
|---|---|
| **Obesity**<br>*Explanation:* Because of the increased risk of heat illness and cardiovascular strain, obese athlete particularly needs careful acclimatization (to the environment and to exercise intensity and duration), sufficient hydration, and potential activity and recovery modifications during competition and training. | Yes |
| **Organ Transplant Recipient (and Those Taking Immunosuppressive Medications)**<br>*Explanation:* Athlete needs individual assessment for contact, collision, and limited-contact sports. In addition to potential risk of infections, some medications (e.g., prednisone) may increase the tendency for bruising. | Qualified yes |
| **Ovary, Absence of One**<br>*Explanation:* Risk of severe injury to the remaining ovary is minimal. | Yes |
| **Pregnancy/Postpartum**<br>*Explanation*: Athlete needs individual assessment. As pregnancy progresses, modifications to usual exercise routines will become necessary. Activities with high risk of falling or abdominal trauma should be avoided. Moreover, scuba diving and activities posing risk of altitude sickness should be avoided during pregnancy. After birth, physiologic and morphologic changes of pregnancy take 4–6 weeks to return to baseline. | Qualified yes |
| **Respiratory Conditions**<br>**Pulmonary Compromise Including Cystic Fibrosis**<br>*Explanation:* Athlete needs individual assessment, but generally, all sports may be played if oxygenation remains satisfactory during graded exercise test. Athletes with cystic fibrosis need acclimatization and good hydration to reduce the risk of heat illness. | Qualified yes |
| **Asthma**<br>*Explanation:* With proper medication and education, only athletes with severe asthma need to modify their participation. For those using inhalers, recommend having a written action plan and using a peak flowmeter daily. Athletes with asthma may encounter risks when scuba diving. | Yes |
| **Acute Upper Respiratory Infection**<br>*Explanation:* Upper respiratory obstruction may affect pulmonary function. Athlete needs individual assessment for all but mild disease. See Fever. | Qualified yes |
| **Rheumatologic Diseases**<br>**Juvenile Rheumatoid Arthritis**<br>*Explanation:* Athletes with systemic or polyarticular juvenile rheumatoid arthritis and history of cervical spine involvement need radiographs of vertebrae C1 and C2 to assess the risk of spinal cord injury. Athletes with systemic or HLA-B27-associated arthritis must undergo cardiovascular assessment for possible cardiac complications during exercise. For those with micrognathia (open bite and exposed teeth), mouth guards are helpful. If uveitis is present, risk of eye damage from trauma is increased, and ophthalmologic assessment is recommended. If visually impaired, guidelines for functionally one-eyed athletes should be followed. | Qualified yes |
| **Juvenile Dermatomyositis, Idiopathic Myositis, Systemic Lupus Erythematosus, and Raynaud Phenomenon**<br>*Explanation:* Athlete with juvenile dermatomyositis or systemic lupus erythematosus with cardiac involvement requires cardiology assessment before participation. Athletes receiving systemic corticosteroid therapy are at a higher risk of osteoporotic fractures and avascular necrosis, which should be assessed before clearance; those receiving immunosuppressive medications are at a higher risk of serious infections. Sports activities should be avoided when myositis is active. Rhabdomyolysis during intensive exercise may cause renal injury in athletes with idiopathic myositis and other myopathies. Because of photosensitivity with juvenile dermatomyositis and systemic lupus erythematosus, sun protection is necessary during outdoor activities. With Raynaud phenomenon, exposure to cold presents a risk to hands and feet. | |
| **Sickle Cell Disease**<br>*Explanation:* Athlete needs individual assessment. In general, if status of the illness permits, all but high exertion and collision/contact sports may be played. Overheating, dehydration, and chilling must be avoided. Participation at high altitude, particularly when not acclimatized, also poses a risk of sickle cell crisis. | Qualified yes |
| **Sickle Cell Trait**<br>*Explanation:* Athletes with sickle cell trait generally do not have an increased risk of sudden death or other medical problems during athletic participation under normal environmental conditions. However, when high exertional activity is performed under extreme conditions of heat and humidity or increased altitude, such catastrophic complications can rarely occur. Athletes with sickle cell trait, like all athletes, should be progressively acclimatized to the environment and to the intensity and duration of activities and should be sufficiently hydrated to reduce the risk of exertional heat illness and/or rhabdomyolysis. According to National Institutes of Health management guidelines, sickle cell trait is not a contraindication to participation in competitive athletics. | Yes |

*Continued*

**TABLE 3.5 MEDICAL CONDITIONS AND SPORTS PARTICIPATION—cont'd**

| Condition | May Participate |
|---|---|
| **Skin: Herpes Simplex, Molluscum Contagiosum, Verrucae (Warts), Staphylococcal and Streptococcal Infections (Furuncles [Boils], Carbuncles, Impetigo, and Methicillin-Resistant Staphylococcus Aureus [Cellulitis and/or Abscesses]), Scabies, and Tinea**<br>*Explanation:* During contagious periods, participation in gymnastics or cheerleading with mats, martial arts, wrestling, or other collision, contact, or limited-contact sports is not allowed. | Qualified yes |
| **Spleen, Enlarged**<br>*Explanation:* If the spleen is acutely enlarged, then participation should be avoided because of risk of rupture. If the spleen is chronically enlarged, then individual assessment is needed before collision, contact, or limited-contact sports are played. | Qualified yes |
| **Testicle: Undescended or Absence of One**<br>*Explanation:* Certain sports may require a protective cup. | Yes |

(Adapted from Rice SG. American Academy of Pediatrics Council on Sports Medicine and Fitness. Medical conditions affecting sports participation. *Pediatrics.* 2008)

written format so that there is no doubt that a discussion took place.
- Compliance with school and governing body guidelines, standards, rules, regulations, and policies
- Compliance with privacy laws (HIPAA and FERPA) and local state and/or federal rules, regulations, and laws
- Documentation

**"Good Samaritan" statutes:** certain states have made an effort to protect volunteer examiners (PPPE) under "Good Samaritan" statutes.

**Sexual harassment:**
- Athlete expectations, lack of privacy during examinations, and inappropriate examinations (e.g., breast or gynecologic examination in nonprivate settings) may contribute to such allegations.
- Clear communication with athletes, respect for their privacy, and presence of a chaperone (in certain situations) during examinations minimize potential problems.

## Confidentiality

- Good communication with other healthcare providers, parents, athletic trainers, and coaches is important, but it must take place with respect for the athlete's confidentiality.
- The newest PPPE form includes multiple pages—clearance considerations and recommendations are available as pages separate from the detailed history and examination.

## Team Physician Versus Personal Physician

- Team physicians may perform the PPPE but have minimal control over evaluation of specific problems uncovered in the PPPE, particularly in high schools, smaller colleges, and managed care settings.
- Space is provided near end of the recommended PPPE form to write specific recommendations for further evaluation before clearance (see Preparticipation Physical Evaluation: Clearance Form available at https://www.aap.org/en-us/professional-resources/practice-support/Documents/Preparticipation-Physical-Exam-Form.pdf, Accessed March 2016). Communication and documentation is essential for the physician and the athlete (and parent/guardian, if applicable) as well as with other physicians.

## Communication

- The team physician's **primary responsibility is toward the athlete, and parent/guardian if the athlete is younger than 18 years, in terms of discussion and decision making.** Secondary responsibility is toward the university, school, or organization. This distinction is often critical at the professional level. It is important, at times, to explain primary and secondary responsibility to coaching staff and administration in terms of sharing information. Emphasis must be placed on concerns regarding long-term safety and health.
- The team physician must respect the athlete's confidentiality with some understanding that if he or she is unable to participate in practice or play, this information should be shared with the coach and other medical care providers. Communication with athletic trainers, administrators, parents, and others, including the press, may be important. First, discuss with the athlete what information must be disclosed.
- The team physician can arrange for follow-up communication. Have a plan for follow-up care and establish it in writing, if appropriate. Arrange for additional evaluation and final clearance once additional testing requirements are met. Arrange for further rehabilitation, functional testing, return to play with modifications, and progression, as necessary. Inform the athlete regarding the risks and concerns of continued participation, and do not assume that the athlete will choose to participate. Follow-up care is essential.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SIDELINE PREPAREDNESS AND EMERGENCIES ON THE FIELD

4

Robert J. Dimeff • Munawar Hayat

## GENERAL PRINCIPLES

- Sideline preparedness is the recognition and formation of medical services in order to promote athletic participation, provide adequate administration of medical care, and reduce risk of injury.
  - It is achieved by having a unified system with qualified medical staff, pre-event preparation, game-day preparation, and post-event evaluation.
  - Many factors influence the type of injuries and emergencies that occur, and it is critical that the director of medical services is knowledgeable about the demands of the competition. Medical services will vary widely among a multiday competition, a 1-day Ironman race, an American football game, and a gymnastic meet.
  - During any sporting event, availability of medical services for participants, volunteers, and spectators is extremely vital. The extent of services is often part of a signed contractual agreement.
  - The medial team may consist of one individual physician or athletic trainer or may include several different healthcare professionals. The medical staff may be paid for their services, provided in-kind services, or unpaid volunteers with varying levels of knowledge, degrees, specialties, and experience.
- Pre-event planning and practice are imperative to improve safety measures, reduce risks to athletes and spectators during event participation, and provide appropriate medical care.
- Game-day planning is essential as it streamlines medical care for those injured on site.
- Post-event evaluation is also critical in order to provide continuing care for the injured and to provide strategic information for improvement in future events.

## MEDICAL STAFF

- Director of medical services
  - Is usually a physician but may be an athletic trainer or other healthcare provider; physician should be qualified MD or DO with an unrestricted medical license along with training and certification in sports medicine and knowledge of common sports injuries and illnesses
  - Responsibilities include:
    - Making decisions regarding the health of all players
    - Assembling the medical staff
    - Creating and rehearsing an emergency activation plan (EAP) and chain of command for practice and game environments
    - Being available at all times or have back-up coverage
    - Coordinating care among others associated with the team
    - Responsibility for medical transfer of injured or ill athletes and for clearing athletes to play
    - Safety assessment of the practice facility, event environment, and playing conditions
    - Providing appropriate documentation of medical care
    - Possible involvement in the development of drug testing protocols, treatment, and prevention programs
    - Communication with athletes, parents, administrators, coaches, athletic directors, general managers, owners, media, sports agents, legal experts, and others regarding any health issues related to sports participation
- Associate physicians
  - Should also be MD or DO with unrestricted license to assist the medical director; this may include various orthopedic surgeons and primary care physicians with interest and training in sports medicine
- Additional physicians
  - May be required by league rules and can include trauma surgeons, neurologists/neurosurgeons, cardiologists, plastics surgeons, ophthalmologists, maxillofacial surgeon, dentists, dermatologists, and others
- Head athletic trainer
  - Certified, with an unrestricted license, and is a critical member of the healthcare team
  - Works closely with the team physicians
  - Provides all aspects of medical care
- Other key members who may be included in successful sideline preparedness are assistant athletic trainers, exercise physiologists and strength and conditioning coaches, physical therapists, nurses, psychologists, dietitians, chiropractors, massage therapists, paramedics, and emergency medical technicians (EMTs).

## MEDICAL EQUIPMENT

- Medical equipment needs are substantially variable and are dependent on several factors such as the type of medical personnel in attendance; timing, duration, and location of the event; available on-site medical facilities; number of participants and spectators; access to medical facilities; expense; and league policy.
- Pre-event communication among medical providers will better ensure that all equipment needs are met, and unnecessary duplication of equipment is minimized.
- The medical director needs to know what supplies are available on site and what is available at local medical facilities.
- On-site supplies may be provided by the physician; an athletic trainer or physical therapist; the organization or team; and/or the paramedic or EMT squad.
- Required and/or essential and recommended and/or desirable supplies are listed in Table 4.1.

## PRE-EVENT

- The medical staff should be aware of common injuries and illnesses that occur during the sporting event for which they are to provide medical coverage; this may be accomplished by reviewing epidemiologic data, injury reports from previous similar events, published research, sports medicine textbooks and literature, and previous personal experiences. Having this knowledge will assist the medical staff in the creation of strategies and policies to promote athlete health, optimize medical care, and prevent any injury that may occur during the sporting event.
- The medical staff may be required to clear athletes with medical disorders and identify those at a risk of health issues related to participation.

**TABLE 4.1 MEDICAL EQUIPMENT**

| | Required and/or Essential | Recommended and/or Desirable |
|---|---|---|
| **Medical Bag** | **General**<br>Alcohol/betadine<br>Scissors<br>Bandages<br>D50W/instant glucose<br>Disinfectant<br>Exam gloves<br>Angiocatheters<br>Local anesthetics<br>Needle/syringes<br>Pen and paper<br>Sharps box<br>Suture kits<br>Wound irrigation<br>Thermometer<br>**Cardiopulmonary**<br>Airway and mask<br>BP cuff<br>Stethoscope<br>Cricothyrotomy kit<br>Epi 1:1000<br>Beta agonist MDI<br>**Head and Neck**<br>Dental kit<br>Eye kit<br>Ophthalmo/otoscope/pen light<br>Pin/sharp object<br>Reflex hammer | Benzoin<br>Blister care<br>Contact lens solution<br>Cautery<br>Instruction sheets<br>Emergency phone numbers<br>Mirror<br>Nail clippers<br>Nasal tampons<br>Paper bag<br>Rx pad<br>Razor and cream<br>Rectal thermometer<br>Scalpel<br>Skin lubricant<br>Skin stapler<br>Tongue depressor<br>Topical antibiotics<br>Preferred medications<br>ACLS medications<br>Normal saline IVF kit |
| **Medical Supplies** | AED/defibrillator<br>Extremity splints<br>Cervical collar<br>Radio/phone access<br>Spine board<br>Sling and swath<br>Ice<br>Oral fluids<br>Plastic bags<br>Facemask removal equipment<br>Medical waste bags | Blanket<br>Mylar sheets<br>Crutches<br>Mouth guards<br>Sling psychrometer<br>Tape cutter<br>Concussion assessment tools<br>Glucometer<br>I-Stat |

- The medical staff should visit the course/venue to inspect for safety risks, location of training rooms and medical treatment facilities, optimum positioning of medical staff during the event, and ambulance/emergency egress routes.
- The event EAP and injury protocols must be written, rehearsed, and practiced with all members of the healthcare team, and everyone should have a clear understanding of their roles and responsibilities in the event of a medical emergency.
- Successful care during a sideline emergency is achieved with regular practice drills of emergency situations.

## EVENT COVERAGE

- Game-day responsibilities will vary with the type of sporting event being covered and the venue where it is being held.
- The medical staff should arrive early, at least an hour before the start of the event. This will allow time to meet with other members of the medical staff, coaches, and administrators; opposing team medical personnel and administrators; game officials; paramedics/EMTs; security and law enforcement members; and other volunteers.
- Dress appropriately for the event and make sure to have appropriate medical credentials. Standardized clothing will allow for easy identification.
- It is critical to ensure that every member of the team understands what role they play in case of an emergency, and also allow time to rehearse and review the EAP.
- The medical team should meet with the opposing team's medical staff to ensure they have adequate medical coverage, know how to activate the EAP, and rehearse injury scenarios.
- Determine who will be the first providers to tend to an injured athlete and the means of communicating with other staff to assist with care. In addition, determine who will be providing care to spectators, officials, coaches, volunteers, and others in attendance.
- The medical director should review medical equipment and supplies, assess the sports venue and examination and treatment sites, evaluate environmental and weather risks, determine the closest medical facilities, and ensure that appropriate medical communication systems are in working order.
- May be required to determine final clearance for injured athletes to return to play
- Will assess and manage game-day illnesses and injuries, determine if the patient may be treated on site or requires urgent/emergency transportation, and if the athlete may return to game-day participation
- Physician expertise and experience have an impact on game-day return to play. One must consider if the disorder may be worsened by participation, predispose the athlete to other health risks, or place others at an increased risk of injury or illness.

## POST-EVENT

- Review injuries and illnesses that occurred during the event, follow-up participants who required emergency transportation, replenish medical supplies, and determine and correct deficiencies
- Communicate as necessary with appropriate nonmedical individuals and organizations such as parents, teachers, teammates, coaches, athletic directors, general managers, security staff, player agents, the event sponsor, the media, and others
  - HIPAA rules must be followed; however, these are often superseded by collective bargaining agreements, player contracts, scholarship rules, and other legal positions.
  - Nevertheless, it is critical to discuss medical issues and recommendations with the athlete first and foremost before communicating with others.

## EMERGENCIES ON THE FIELD

- Quick overall assessment if injury is life threatening (e.g., cardiac event, tracheal fracture, or laceration) or limb threatening (e.g., fracture dislocation with neurovascular compromise)
- Determine if there are multiple casualties that require further triage
- Secure safety of those attending to the injured athlete and prevent those injured from further injury
- Decide on the safest method to remove athlete from the playing field, where the athlete will be further assessed (sideline, training room, on-site medical facility, urgent care facility, emergency room [ER], or hospital), and the best means and timing of transportation
- If after on-site evaluation, it is determined that the athlete does not require emergent transportation, a decision should be made if it is safe for the athlete to return to play

## Cardiopulmonary Arrest

- Check for responsiveness; if not responsive, then activate the EAP
- Check the airway for spontaneous breathing, ensure that the athlete has a patent airway, and maintain cervical spine stability in neutral position, particularly if possible head and neck injury is a concern
- Remove facemask as necessary to gain access to and secure the oral airway
- Check pulse, remove clothing and protective gear, and start chest compressions at a depth of ≥2" and a rate of 100–120 compressions/minute
- If the patient is in ventricular fibrillation or exhibits unstable ventricular tachycardia, early defibrillation is necessary to restore normal electrical cardiac activity; this may be accomplished by use of an automated external defibrillator or manual cardiac monitor/defibrillator. Each minute of delay in defibrillation decreases the chance of survival by approximately 10%.
- Administer oxygen, secure intravenous access, and follow ACLS protocols while awaiting ambulance transport to a medical facility.

## Exercise-Induced Collapse

- Collapse during or after prolonged exercise bouts can be due to numerous common disorders. The medical staff must be able to provide prompt, accurate, on-site evaluation and treatment.
- Exercise-induced collapse (EIC) is defined as the inability to walk unassisted with or without exhaustion, nausea, vomiting, and cramps. There can be variable levels of consciousness; it is critical to record vitals including rectal temperature.
- Collapse before the finish line suggests a more serious disorder than collapse after completion of the event.
- Most common causes are postural hypotension, muscle cramps, dehydration, heat illness, hypoglycemia, hypothermia, and hyponatremia.
- Postural hypotension
  - Occurs after finish line and is due to blood pooling from gravity and loss of muscle pump
  - Treatment with elevation of legs and oral or intravenous (IV) fluids usually provides prompt recovery
- Muscle cramps
  - Often involves just lower legs, but whole body cramps may include thighs, abdomen, back, and upper extremities
  - Occurs in an unacclimatized, often hot and humid, environment
  - Likely due to a combination of dehydration, sodium or electrolyte depletion, muscle fatigue, and neural disturbance
  - Treatment includes stretching, oral salt and fluid intake, ice, massage, IV fluids, and IV benzodiazepines or magnesium
- Dehydration
  - May impair performance and lead to collapse due to reduced blood volume
  - Occurs in hot and humid environment, particularly if there is no cloud cover
  - Higher risk in those with acute illness and may increase the risk of hyperthermia
  - Treatment includes elevation of legs and oral or IV fluids
  - Prevention includes ensuring adequate fluid, electrolyte, and carbohydrate before, during, and after training and competition
- Heat illness
  - Ranges from heat exhaustion and syncope to life-threatening hyperthermia
  - Altered mental status with rectal temperature elevation (often 106°F–109°F)
  - Immediate cooling with cold water, fans, cold towels, and fluids and moving the athlete to a cool/shady environment are necessary.
  - Ice-water immersion is the most effective means of lowering core temperature and should be started once the disorder is recognized.
  - When core temperature reaches 102°F–103°F, ice immersion should be discontinued to prevent overshoot and hypothermia.
- Hypoglycemia
  - More likely to occur in long races in athletes with diabetes, eating disorders, and those with poor carbohydrate intake
  - This life-threatening state is diagnosed by history, examination, and blood sugar evaluation.
  - Corrected with administration of oral or IV glucose
  - If blood sugar measurement is not possible, administration of IV D50 is recommended.
- Hypothermia
  - Usually related to accidental prolonged exposure to cold damp weather during long-distance races and during the swim portion of triathlons
  - Usually presents with mild confusion and shivering that resolves when core temperature is <90°F
  - Athlete should remove cold, wet clothes, be moved to a warm environment, change into dry clothes, and use warm blankets and heaters to increase body temperature.
- Hyponatremia
  - Potentially fatal condition due to water overload from drinking hypotonic fluids or water during exercise
  - Sodium loss may be a contributing factor.
  - Occurs in those exercising for prolonged periods, often >4 hours, and may be more common in females
  - Athletes have normal vitals and temperature but gain weight during the event, appear bloated, and are confused, often with severe headache, nausea, and vomiting, and may develop seizures.
  - Once recognized and confirmed with serum sodium determination, treatment with observation, diuretics, and oral and IV hypertonic fluids may be required.

## Asthma

- Assess the severity by observing for increased respiratory rate, wheezing, cough, breathlessness, and use of accessory muscles
- Initial sideline management includes two puffs of albuterol with spacer 1 minute apart; may be repeated in 15–20 minutes while assessing for improvement; consider $O_2$ administration.
- If there is no change or worsening, transport to nearest facility for further care

## Anaphylaxis

- Severe, acute, life-threatening systemic reaction due to allergen exposure
- Characterized by flushing, urticaria, angioedema, chest tightness, cough, wheezing, and stridor progressing to pulmonary failure and cardiac arrest
- Epinephrine 0.3–0.5 mL of 1:1000 dilution into lateral thigh every 10–15 minutes (0.01 mg/kg in children; maximum, 0.3 mg)
- Transport to ER, supplemental oxygen, IV fluids, consider beta-agonist inhalers, $H_1$ or $H_2$ blockers, and systemic corticosteroids

## Acute Hemorrhage

- Internal bleeding, such as intracranial, abdominal, retroperitoneal, renal, and hepatic hemorrhages, is difficult to diagnose at the athletic venue; requires frequent assessment of vitals and urgent transportation to a hospital facility for emergency treatment
- External bleeding is common in sports due to abrasions and lacerations. For bleeding injuries, clean with water or saline to remove any foreign bodies and apply direct pressure with gauze. After administration of a local anesthetic with or without epinephrine, thoroughly assess lacerations for possible muscle, tendon, and neurovascular injury before closing the wound with appropriate sutures, staples, or surgical adhesive.
- Quikclot combat gauze is a commercial product that may be used to control severe, potentially life-threatening bleeding due to major vessel injury. Lacerations of carotid, brachial, femoral, and popliteal arteries should be treated with constant direct pressure and tourniquet, when applicable, until a vascular or trauma surgeon is able to do emergency vessel repair or ligation.
- For nosebleeds, keep the head elevated in a sniffing position and apply direct pressure at the nostrils followed by ice packs to control bleeding. Neosynephrine spray and nasal tampons may be helpful; if bleeding cannot be controlled, consider transfer to a medical facility for further evaluation and nasal packing.

## Head and Neck

- Head and neck injuries are common in contact and collision sports; most are benign and self-limited, some are serious and life threatening.
- Determine potential seriousness of the injuries, such as subdural/epidural hemorrhage, intracranial contusion, skull fracture, concussion, cervical fracture dislocation, spinal cord injury, and laryngotracheal fracture.
- Assume unconscious athlete has a cervical spine injury.
- Stabilize head and neck, secure airway access, backboard, and transport to designated medical facility. Helmet and shoulder pad removal must be practiced among the medical team.
- Current accepted best practice is that a concussed athlete should not return to play the same day. Any deterioration in mental status or neurologic examination requires transportation to a medical facility for further evaluation.

## Facial

- Commonly seen in all contact sports that do not require face masks or shields
- Ensure adequate airway control and clear cervical spine
- Inspect and palpate areas of swelling for crepitus, tenderness, or abnormal bony prominences and assess for normal range of motion of the jaw
- For nasal fractures, consider immediate reduction; monitor for uncontrolled bleeding and septal hematoma; also observe for clear rhinorrhea suggestive of skull fracture and cerebrospinal fluid leak
- For ear trauma, check for lacerations, tympanic membrane rupture, and clear otorrhea suggestive of cerebrospinal fluid leak

## Eye

- Type and severity of eye injuries depend on sport environment and mechanism of injury, i.e., blunt versus penetrating trauma
- Most eye injuries can be prevented with protective eyewear in high-risk sports.
- Assess visual acuity and visual fields, palpate periorbital tissues and eye, examine conjunctiva, cornea, anterior chamber, pupil size and reaction to light, and iris shape and continuity. Check extraocular movements after ruling out perforation; then consider anesthetic and fluorescein stain to evaluate for corneal abrasion
- Athletes with corneal irritations, abrasions and lacerations, subconjunctival hemorrhage, or foreign bodies may return to play after evaluation, irrigation, and removal of offending substance.
- Corneal laceration, significant eyelid laceration, hyphema, orbital wall fracture, ruptured or penetrated globe, and posterior segment injuries (i.e., retinal tear, detachment, edema, or hemorrhage) should be protected with eye shield and referred for emergent definitive ophthalmologic care.

## Oral

- Most common injuries involving the oral cavity are abrasions and lacerations of the lips, tongue, and buccal mucosa; most are easily assessed, treated, and sutured on site. Significant through-and-through oral lacerations may require transfer for appropriate and definitive treatment.
- Dental injuries include tooth fractures, subluxations, and avulsions.
- Bleeding and seepage around a tooth and simple finger-pressure test to check for pain, numbness, and excess movement associated with fractures and subluxations.
- Type I fractures involve enamel alone with no pain; may continue to play; delayed bonding for cosmesis.
- Type II fractures involve dentin and is painful; may continue to play; requires urgent bonding.
- Type III fractures involve pulp and significant pain; usually cannot continue to play; emergent treatment to preserve tooth, may require root canal.
- Lateral subluxations and extrusions should be gently manipulated into position followed by evaluation of bite alignment. Intrusions should be referred for urgent dental treatment. Subluxations require bracing with adjacent teeth to allow healing.
- Avulsion is complete displacement of tooth from its base. Handle by crown, gently cleanse with water or saline, avoid disturbing the attached soft tissues, replace in socket quickly, and clench teeth to hold it in place. If unable to replace, transport in saline, water, or commercial tooth-saver solution. Replacement of the tooth is time dependent; 90% salvage rate if replaced within 30 minutes; will require splinting to preserve tooth.

## Chest

- Emergency chest injuries are rare and occur primarily because of direct blow by a fast-moving object or another player.
- Life-threatening chest injuries include cardiac contusion and commotio cordis, tension pneumothorax, and damage to great vessels and airway.
- Chest-wall contusions and fractures may be complicated by brachial plexus injury, diaphragmatic rupture, pneumothorax, pulmonary contusion, and renal and hepatic injury.
- Assess vitals and $O_2$ saturation, observe for signs of respiratory distress and hemoptysis, tracheal deviation, distended neck veins, chest wall deformity, crepitus and tenderness, paradoxical chest movements, and abnormal breath sounds.
- If tension pneumothorax is suspected and patient is unstable, perform emergent needle decompression by using a 14–16-gauge needle above the third rib along the midclavicular line of the affected side to release air; administer $O_2$ and transfer to a medical facility for further imaging and care.
- Uncomplicated chest trauma may be treated with ice, analgesics, padding, and local anesthetic injection; may return to sport if tolerated; requires frequent reassessment

### Abdomen

- Abdominal muscle strains and contusions are the most common sports injuries and can be treated with ice, analgesics, compression wraps, and participation as tolerated, with frequent reassessment.
- Severe abdominal injuries causing internal organ damage are uncommon in sports with spleen, kidney, and liver being the most frequently affected organs.
- Signs and symptoms of contusion, hematoma, and laceration of abdominal structures are often delayed; thus, frequent reassessment including vital signs is necessary.
- Liver injury produces right upper quadrant pain and tenderness, referred pain to the right shoulder, and ecchymosis around the umbilicus (Cullen's sign).
- Splenic injury may cause left upper quadrant pain and referred pain to the left shoulder (Kerr's sign). Athletes with a recent history of infectious mononucleosis may be at a higher risk of splenic rupture.
- Kidney injury is often caused by trauma to the back and flank and may be associated with rib fractures. In addition, hematuria, flank ecchymosis (Grey–Turner's sign), and focal tenderness may be found.
- Potentially serious abdominal injuries can be difficult to assess on the sideline. If a serious abdominal injury is suspected, the athlete should not be given anything orally, have IV access established, and transferred to a hospital for further evaluation and treatment.

### Musculoskeletal

- The most common acute athletic injuries involve the musculoskeletal system: bone, cartilage, ligament, muscle, tendon, and neurovascular structures.
- Return to play is predicated based on diagnosis, pain control, ability to protect the athlete from further injury or injuring others, and completion of sport-specific demands.
- Acute bone injuries include contusions and fractures. Fractures may be difficult to diagnose without on-site imaging; minor to obvious deformity, focal tenderness, crepitus, and ecchymosis may be present. Check neurovascular status, attempt reduction, particularly if there is neurovascular compromise, splint to include the joint proximal and distal to the injury site, use ice and analgesics, and arrange for definitive imaging and treatment.
- Cartilage injuries are difficult to diagnose without advanced imaging; joint swelling, loss of motion, and tenderness may be present. Athletes may attempt return to play if they are able to complete functional testing.
- Ligament sprains may be contact or noncontact and affect virtually any joint, leading to instability and possible joint dislocation; mechanism of injury and physical examination are usually all that is necessary for an initial diagnosis. Sideline management will include attempted reduction of dislocated joints, particularly if there is neurovascular compromise, ice, analgesics, splinting, and taping, which may allow return to play in certain cases. Athletes should be then followed up for radiographic imaging and further definitive treatment.
- A trained physician can easily reduce most dislocations that occur on the field; however, if there is any difficulty, this should be attempted in the locker room or medical facility after further imaging and sedation.
- Acute muscle injuries include noncontact strains, direct contact contusions, and hemorrhage and hematomas. On-site treatment includes ice, compression, elevation, and analgesics. Return to play may be possible. Athletes should be monitored for the development of acute compartment syndrome that may be associated with fractures and muscle injury (severe pain that worsens with stretching, paresthesias, paralysis, pulseless, and pallor). Compartment pressures should be monitored, and emergency fasciotomy may be necessary.
- Tendon injuries range from minor 1° strains to complete 3° strains and may occur at muscle origin, muscle–tendon junction, mid-tendon substance, or tendon insertion. Most are eccentric noncontact injuries and are diagnosed by history and physical examination and radiologically confirmed by radiographs, musculoskeletal ultrasound, or MRI. Acute treatment includes rest, ice, compression, immobilization, and analgesics; surgical treatment may be necessary. Same-day return to play is often difficult; athletes should not return to play until function is restored.
- Neurovascular injuries must be fully assessed; athletes may return to play when they are pain free, and full motion, strength, power, and sensation have been achieved.

## SUMMARY

- Sideline evaluation and treatment of injuries related to sports participation, including safe return to play or transfer to a medical facility for further assessment, require extensive pre-event planning.
- Essential to have well-trained medical personnel, appropriate equipment, and communication when providing medical coverage for a sporting event
- Rapid assessment is critical to determine the need for life-saving treatments, such as CPR, defibrillation, airway management, and bleeding control.
- Frequent sideline reassessment of athlete may be necessary to determine the need for transfer to a medical facility for further evaluation and treatment.
- Most medical disorders and injuries can be managed on site by care providers with appropriate training and equipment and can allow for rapid and safe return to play.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


1. American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care, part 5: adult basic life support and cardiopulmonary resuscitation quality. *Circulation*. 2015;132(18 suppl 2):S414-S435.
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# 5 SPORTS NUTRITION

Jacqueline R. Berning

## ROLE OF NUTRITION IN ATHLETIC PERFORMANCE

- Exercise training and genetic makeup are major determinants of athletic performance.
- A healthy diet will not substitute for either factor, but making wise food choices will allow athletes to maximize their athletic potential by contributing to endurance, speed, and recovery of muscle tissue.
- Athletes usually lack nutrition knowledge regarding diet and performance, which is often not a priority and leads to misinformation regarding diet and exercise performance.
- In order to educate and assess the dietary practices of athletes, sports medicine professionals require an understanding of sports nutrition to be able to recommend eating patterns that allow athletes to perform at their full potential.

## ENERGY SOURCES FOR MUSCLES

- A basic understanding of how muscles use food as fuel is an important step in understanding an athlete's diet.
- The body cannot directly use the energy released from food; it must first convert the chemical energy found in food to adenosine triphosphate (ATP) (Fig. 5.1).
- A small amount of ATP can be found in resting muscle cells—just enough to keep the muscle working maximally for about 1–2 seconds.
- During extended periods of exercise, the body produces additional ATP by adding phosphocreatine to ADP, thereby producing more ATP for muscle contractions.
- Moreover, energy-yielding macronutrients such as carbohydrates, fats, and occasionally proteins are used as energy sources to produce more ATP (Table 5.1).

### Carbohydrate: Major Fuel for Short-Term, High-Intensity Exercise

#### Anaerobic vs. Aerobic Metabolism

- The primary pathway to provide energy for sporting events where exercise intensity is near maximal for 30–90 seconds is glycolysis or anaerobic metabolism.
  - During anaerobic glycolysis, lactate is produced; it accumulates in the muscles and increases acidity. As lactate increases, pH in the muscle lowers and is associated with the onset of fatigue. The muscle lactate produced from glycolysis is eventually released into the bloodstream and taken up by the liver, which resynthesizes the lactate into glucose. This pathway is called the Cori cycle and allows glucose to enter the bloodstream and be absorbed by cells to be used as energy.
- During anaerobic exercise, carbohydrate is the only substrate that can be used to resupply ATP.
- If oxygen is available to muscle cells and exercise is performed at a moderate or low intensity, then most of the pyruvate produced during glycolysis is shuttled into the mitochondria for ATP production via aerobic metabolism. Approximately 95% of ATP produced from carbohydrate metabolism is aerobically formed in the mitochondria.
- Aerobic metabolism supplies ATP more slowly than anaerobic pathways, but it releases greater energy and can be sustained for hours.

#### Glycogen

- Carbohydrates are stored in the form of glycogen in the body; it is stored both in the liver and the muscles.
- Through glycogenolysis, glycogen is broken down to glucose and metabolized via the anaerobic and aerobic pathways. Liver glycogen is used to maintain blood glucose, whereas muscle glycogen supplies glucose to working muscles.
- In events lasting <30 minutes, muscles primarily rely on muscle glycogen. As exercise time increases, muscle glycogen stores decline, and they begin to absorb glucose from the blood as an energy source (Fig. 5.2).
- Once glycogen stores are exhausted, exercise can continue, but the muscles can only work at 50% of maximal capacity; this state is often called "hitting the wall" or "bonking" because further exertion is hampered.
- In certain cases, before a competition, high-carbohydrate diets can be used to increase muscle glycogen stores to up to double the typical amount, thereby delaying the onset of fatigue and improving endurance.
- Maintenance of blood glucose becomes increasingly vital as exercise duration increases beyond 20–30 minutes. By maintaining blood glucose, the body saves muscle glycogen to use during sudden bursts of effort or for a second workout later in the day.
- A carbohydrate intake of 0.7 g/kg/hr during strenuous endurance exercise that lasts for approximately ≥1 hour can help maintain adequate blood glucose, which, in turn, results in delayed fatigue.

### Fat: Main Fuel Source for Prolonged, Low-Intensity Exercise

- At rest and during prolonged exercise at low to moderate intensity, fat becomes the predominant fuel source for exercising muscles. In fact, during very long activities such as ultra-marathons, fat supplies approximately 50%–90% of the required energy.
- The rate of fatty acid oxidation in the muscle is affected by training level. The better trained a muscle is, the greater its ability to use fat as a fuel. The more unfit a muscle is, the greater its reliance on carbohydrate as opposed to fat. Training increases the size and number of mitochondria and the level of fatty oxidative enzymes, thus allowing athletes to use fat more readily as a fuel source, eventually conserving glycogen.

### Protein: a Minor Fuel Source During Exercise

- Although protein can be used as fuel by muscles, its contribution is relatively small compared with that of carbohydrates and fat.
- Only approximately 5% of the body's energy needs is supplied by amino acid metabolism; however, proteins can contribute to energy needs during endurance exercise.

**TABLE 5.1 ENERGY SOURCES IN THE BODY**

| Energy Source | Storage Area | When Used | Activity |
|---|---|---|---|
| ATP | All tissues | All the time | Sprinting (0–3 seconds) |
| Phosphocreatine | All tissues | Short bursts | Shot put, high jump, bench press |
| Carbohydrate (anaerobic) | Muscles | High intensity lasting 30 seconds to 2 minutes | 200-meter sprint, 50-meter swim |
| Carbohydrate (aerobic) | Muscles and liver | Exercise lasting 2 minutes to ≥3 hours | Jogging, soccer, basketball, swimming |
| Fat (aerobic) | Muscles and fat cells | Exercise lasting more than a few minutes; greater amounts are used at lower exercise intensities | Long-distance running, marathons, ultra-marathons, day-long hikes |

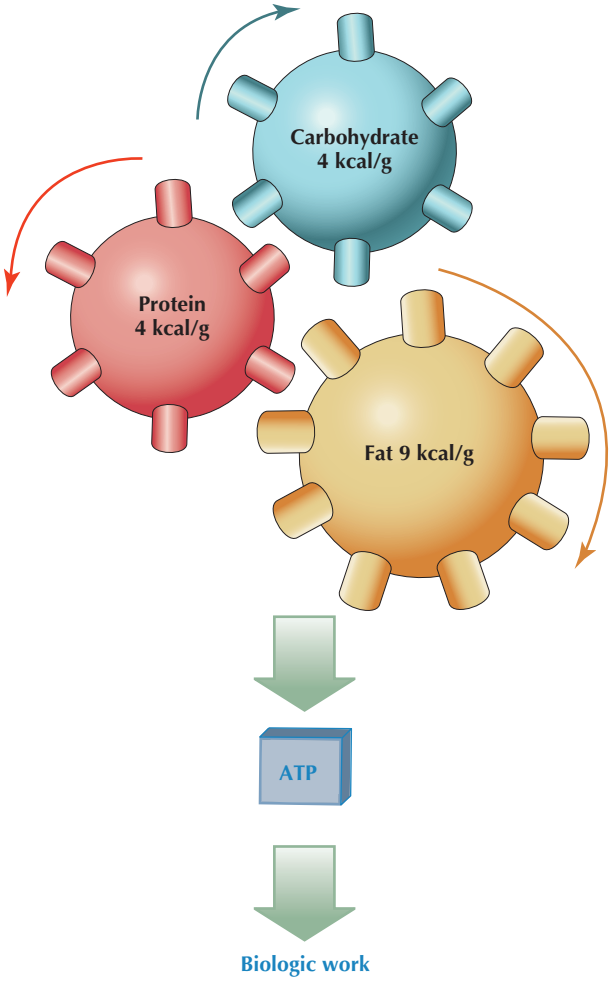


**Figure 5.1.** Sources of ATP for biologic work. ATP is harvested from macronutrients, carbohydrates, proteins, and fats to synthesize ATP for biologic work.

- If an athlete exhausts his or her muscle glycogen stores, then proteins can contribute as much as 15% of the energy expenditure for exercise.

## DIETARY RECOMMENDATIONS FOR ATHLETES

### Energy Needs

- Athletes need varying amounts of energy depending on their body size, body composition, and type of training or competition.
- Monitoring body weight is an easy way to assess the adequacy of caloric intake; however, several sport dietitians are now using the concept of energy availability to monitor caloric requirements.
  - Energy availability is the amount of energy left for bodily functions after the energy costs for training and competition have been calculated. Table 5.2 provides an example of how to calculate adequate energy availability for an athlete.
  - Typically, athletes with an energy availability of <30 kcal/kg of lean body mass have a greater risk of metabolic and hormonal disruptions, which could include reproductive functions as well.
- Regardless of the technique used to monitor caloric requirements, both an excess and inadequate intake of calories can result in fatigue, increased risk of injury, prolonged recovery period, and overall poor athletic performance.

**TABLE 5.2 SAMPLE CALCULATIONS TO DETERMINE ENERGY AVAILABILITY**

| | |
|---|---|
| Athlete's weight (kg) | 68 kg |
| Workout expenditure (kcal/d) | 900 kcal/d |
| Energy intake (kcal/d) | 3200 kcal/d |
| Percent body fat (%) | 13 |
| Lean body mass (kg) | 68 kg × .13 = 8.84 kg; 68 kg – 8.84 kg = 59.16 kg |
| Energy availability (kcal/lean mass) | 3200 kcal – 900 kcal = 1900 kcal/59 kg lean mass = 32.2 kcal/lean mass |

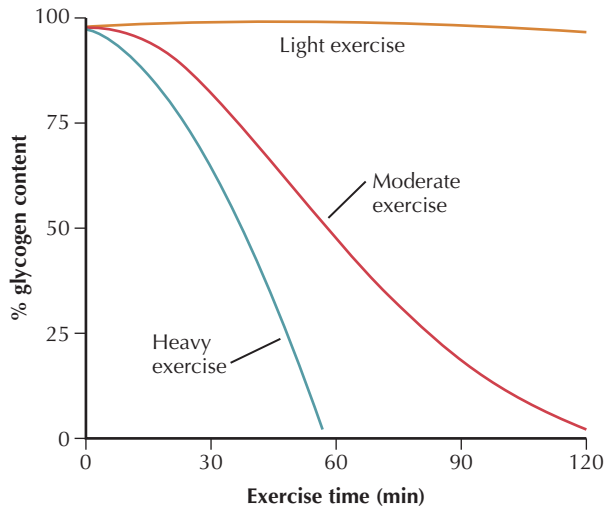


**Figure 5.2.** Rate of glycogen utilization in exercising muscle. (Data from Grisham CM, Garrett RG. *Biochemistry,* 3rd Ed. Brooks/Cole Publishing Company, Belmont CA; 2006.)

## Carbohydrates

- Carbohydrates are the primary source of energy for exercising muscle, particularly when exercise intensity reaches 65% or greater of the maximum oxygen consumption ($VO_2$ max).
- In the past, carbohydrate recommendations have often been expressed as a percentage of total calories; however, this percentage is poorly correlated to both the amount of carbohydrate actually consumed and the required fuel needed to support an athlete's training and competition; thus, the concept of carbohydrate availability is now being used.
  - Carbohydrate availability matches the athlete's carbohydrate intake to his or her fuel needs for training and competition.
  - If athletes fail to consume adequate carbohydrates and energy during daily training, then muscle glycogen levels decrease, and training and competition performance becomes impaired.
  - If athletes overconsume carbohydrates, the excess glucose is stored as fat in the body.
- In general, a wide variety of whole grains such as pasta, breads, and rice along with fruits and vegetables provide carbohydrates as well as essential vitamins, trace minerals, and fiber that athletes need.

### Carbohydrate Recommendations

- **Low-intensity exercise:** Carbohydrate intake ranges 3–5 g/kg of body weight
- **Moderate-intensity exercise:** Carbohydrate intake ranges 5–7 g/kg of body weight
- **Endurance exercise for several hours:** Carbohydrate intake ranges 6–10 g/kg of body weight
- **Extreme exercise lasting 4–5 hours:** Carbohydrate intake ranges 8–12 g/kg of body weight

### Application

- Carbohydrate intake is particularly important when performing multiple training bouts in 1 day, such as swimming practice or track and field events or in tournament play when multiple games may be played in 1 day. Consumption of multiple meals or snacks that contain carbohydrates throughout the day will ensure a fuel source for exercising muscles.

## Protein

- Protein consumption facilitates muscle synthesis and repair.
- Athletes need to consume more than double the protein amount required by a sedentary adult.
  - Most athletes can easily meet their protein requirements through a well-planned diet that includes high-quality proteins that are spread throughout the day rather than consumed in large amounts in one single meal.
  - Researchers have found that consuming >40 g of protein in a single meal has no additional benefits in stimulating more muscle protein synthesis (MPS), and any excess over this amount is simply oxidized by the body. Moreover, they have suggested that multiple meals containing 20–30 g of protein must be consumed throughout the day to stimulate muscle protein synthesis.
- While the amount of protein an athlete needs is important, so is the quality of the protein because it influences the body's ability to synthesize MPS.
  - Protein quality depends upon its essential amino acid profile, which includes both the specific amount and proportion of essential amino acids.
    - Essential amino acids (EAAs) are ones that the body cannot make and must therefore be consumed in the diet.
    - There are 9 EAAs, including 3 branched-chain amino acids (BCAAs).

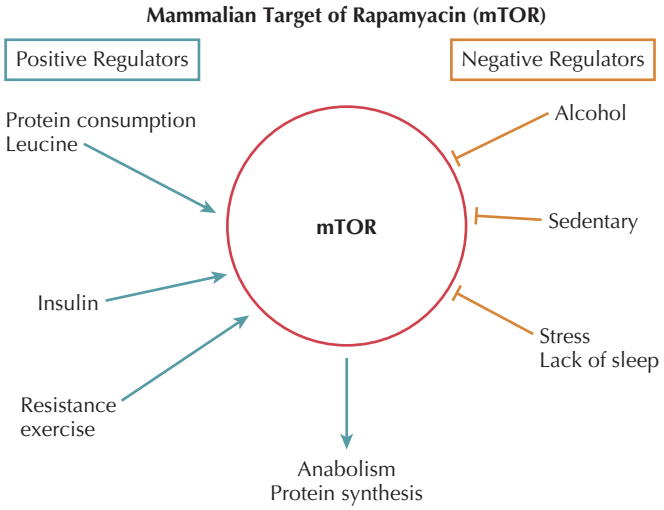


**Figure 5.3.** Regulators that influence the mTor pathway.

### BOX 5.1 CONCENTRATION OF LEUCINE IN FOOD AND DAIRY PRODUCTS

- Meats, fish, chicken, turkey, eggs, and dairy products contain leucine and the other BCAAs
- Effective dose of leucine is approximately 2–3 g/day
  - 12 ounces of milk = 2000 mg leucine
  - 3 egg whites = 990 mg leucine
  - 1 cup of 1% cottage cheese = 2880 mg leucine
  - 3 oz tuna = 1740 mg leucine
  - 3 oz chicken breast = 3690 mg leucine
  - 3 oz beef = 3005 mg leucine

### TABLE 5.3 PROTEIN RECOMMENDATIONS FOR ATHLETES

| Specific Athlete Group | Recommendation in g/kg of Body Weight |
|---|---|
| Endurance athletes | 1.2–1.4 |
| Strength athletes | 1.6–1.8 |
| Vegetarian athletes | 1.3–1.8 |
| Energy-restricted athletes | 1.5–1.7 |

- BCAAs are absorbed faster than small amino acids, and EAAs are absorbed faster than nonessential amino acids with the essential BCAAs leucine, isoleucine, and valine being absorbed the fastest. These physiologic properties give BCAAs the ability to stimulate the pathway that accelerates MPS.
- Research has shown that the amino acid leucine in combination with other BCAAs is critically important in stimulating MPS. Leucine is a key signaling protein of the mammalian target of rapamycin (mTOR) pathway, which is the pathway for stimulating MPS (Fig. 5.3). An effective dose of leucine is about 2–3 g/day. Box 5.1 lists food items that have a high leucine content.

### Protein Recommendations

- Ideally, a mix of animal and plant protein sources should be included in the diet of an athlete.
- Protein consumption for athletes is more than double the requirements for sedentary individuals; however, several athletes exceed their protein requirements by consuming additional protein in the form of shakes, powders, and protein bars. Table 5.3 lists the protein recommendations for athletes.

### Application for Vegetarian Athletes

- Vegetarian athletes appear to meet or exceed their protein requirements; however, since plant proteins have less bioavailability, vegetarian athletes should increase their protein intake by approximately 10%.
- A disadvantage of being a vegetarian athlete is low levels of muscle creatine, a nitrogenous compound found in muscles, which phosphorylates and produces phosphocreatine. Phosphocreatine then donates a phosphate group to adenosine diphosphate (ADP) to become ATP.
- Several studies have found that vegetarian athletes tend to have lower levels of muscle creatine compared with their omnivore counterparts; in addition, vegetarian athletes who consume creatine supplements had a greater potential for resistance training and greater lean tissue mass compared with a placebo group.
- Vegetarianism has several benefits for athletes as vegetarian athletes gain a greater proportion of their energy needs from carbohydrates, including more fruits and vegetables, which may minimize their potential for free-radical damage, which may be advantageous to an athlete's training and health.
- However, there is the potential for nutritional concerns for vegetarian athletes and include vitamin $B_{12}$, iron, zinc, and calcium deficiencies; deficiencies in any of these nutrients can result in poor athletic performance.
- Considering the variability of a vegetarian diet, a sports dietitian can play a key role in educating vegetarian athletes regarding menu planning, cooking, and food preparation in order to maximize their athletic potential.

## Fat

- The role of fats in the body is to supply a source of energy as well as essential fatty acids and fat-soluble vitamins that play important roles in an athlete's diet. Most athletes often meet and/or exceed their recommendations of dietary fat usually because of increased caloric intake.
- High-fat diets are not recommended for athletes primarily because of their adverse health effects. Conversely, athletes who consume very-low-calorie diets (<15% of total calories from fat) do not exhibit any additional performance benefits.
- Athletes who have increased abdominal fat and or a predisposition for diabetes and cardiovascular disease should consume healthier fats such as monounsaturated and polyunsaturated fatty acids and lesser saturated fatty acids and trans fats, along with the addition of more whole grains, fruits, and vegetables.

### Fat Recommendations

- In general, fat recommendations for athletes follow public health guidelines, such as the Dietary Guidelines for Americans, which recommends fat intake in the range of 20%–35% of total caloric intake; however, similar to carbohydrates, fat intake should be customized based on training levels and body composition.
- Some athletes believe that following a high-fat, low-carbohydrate diet can enhance rates of fat oxidation and improve endurance performance. Available literature suggests that high-fat, low-carbohydrate diets may match exercise capacity at moderate intensity but impair exercise at higher intensities.
- While high-fat diets may impair exercise intensity, low-fat diets (<20% of calories from fat) are not recommended because they tend to restrict intake of nutrients such as fat-soluble vitamins and essential fatty acids, particularly *omega-3* fatty acids. In addition, individuals who restrict their dietary intake of fats to <20% of their calories for weight-loss purposes found no advantage of further weight loss following a very-low-fat diet.

## Micronutrients

- Micronutrients play a specific role in facilitating the release of energy from food and in tissue synthesis.
- In general, if an athlete meets overall energy intake and incorporates a wide variety of nutrient-dense foods, vitamin and mineral recommendations can be met through the diet itself.
- Moreover, athletes tend to consume more calories owing to their energy expenditure during competition and training; therefore, they can meet their nutrient requirements with wise food choices. In addition, they may consume highly fortified "sport" foods and drinks, which provide several micro- and macronutrients.
- Certain athlete populations may be at risk of low or marginal intake of micronutrients. Athletes who compete in lean (distance running, gymnastics, swimming, and diving) or weight-restricted sports (wrestling and crew) are often at risk of low or marginal intake of micronutrients owing to their low-caloric diets.

### Iron

**Functions**: Iron is a part of oxygen-carrying transport proteins such as hemoglobin and myoglobin. Iron is also a component of cytochromes that carry electrons to molecular oxygen in the electron transport chain.

**Effects on performance**: Iron deficiency with or without anemia can impair performance and limit work capacity. Some athletes, at the start of training, experience sports anemia, wherein blood volume expands before the synthesis of red blood cells increases. This expansion results in dilution of the blood and a decrease in hemoglobin. Sports anemia is not detrimental to performance but is hard to differentiate from true anemia.

**Etiology**: Potential causes of iron-deficiency anemia can vary; as observed in the general population, female athletes are most susceptible to low iron status owing to monthly menstrual losses. Special diets followed by athletes, such as low-energy and vegetarian, particularly vegan, diets are likely to be low in dietary iron. Distance runners should pay special attention to iron intake because their intense workouts may lead to footstrike hemolysis or increased iron loss in sweat, feces, and urine, which can negatively influence the iron status.

Important interventions:

- **Screening:** Distance runners and all female athletes should have their iron status checked at the beginning of the season and midseason.
- **Serum ferritin**: Currently, there is no consensus on a specific serum ferritin level that corresponds to a possible level of iron depletion/deficiency in athletes. Suggested values range from >10 to <35 ng/mL.
- **Iron intake**: Athletes who are at risk should aim for an iron intake of greater than the RDA (>18 mg for women and >8 mg for men) (Table 5.4). Athletes at risk must follow dietary strategies that increase food sources of iron, such as eating iron-fortified breakfast cereals with a source of vitamin C to enhance iron absorption and cooking in iron skillets.
- **Supplements**: Decisions regarding iron supplementation are best made on an individual basis. It may take 3–6 months to reverse iron-deficiency anemia. While there is some evidence suggesting that iron supplements can improve performance in athletes with iron depletion without anemia, indiscriminate use of iron supplements is not advised and may cause gastrointestinal distress and toxicity.

### Calcium

**Function:** Calcium plays a major role in the formation and maintenance of bone. Athletes, particularly women trying to maintain a lean profile, can have marginal or low intakes of dietary

calcium, particularly if they restrict their caloric intake or avoid calcium-rich foods. Of still greater concern are female athletes who suffer from menstrual disturbances, low energy availability, and bone loss/osteopenia. Low calcium intake in such female athletes contributes to an increased risk of bone fractures during training and competition.

Important interventions:

- **Screening**: Female athletes should be asked about current and past intake of calcium-rich foods, calcium supplementation, as well as menstrual history.
- **Calcium intake**: The RDA for calcium is 1300 mg/day for female athletes aged 19–18 years and 1000 mg/day for athletes aged 19–50 years. Female athletes should be educated about highly bioavailable dietary sources of calcium and how to incorporate these sources in their daily diet (Table 5.5).
- **Supplements**: Calcium supplementation should be determined after a nutritional assessment of dietary calcium. In general, 1500 mg/day of calcium and 1500–2000 IU/day of vitamin D are recommended for athletes with compromised bone health owing to low energy availability and/or menstrual dysfunction.

## Hydration Guidelines

- Fluid loss of >2% of total body weight can compromise athletic performance and cognitive function, particularly in hot environments. Fluid loss ranging 3%–5% of body weight further disturbs athletic performance, particularly in sports that require specific skills. Additional fluid loss of 6%–10% of body weight has profound effects on physiologic functions, including cardiac output, blood flow, and sweat production, and increases the risk of exertional heat illness.
- Thirst is a late sign of dehydration and is not a reliable indicator of an athlete's need to replace fluid during exercise. An athlete who drinks only when thirsty is likely to take 48 hours to replenish fluid loss.
- To identify optimal fluid replacement strategies, athletes should know their individual sweat rates (Box 5.2); athletes should weigh themselves before and after exercise to identify sweat loss from exercise. The rule of thumb is for every pound lost during exercise, 2–3 cups of fluid should be consumed. Athletes should aim for a fluid intake that prevents weight loss during exercise as well as avoid consuming too much fluids resulting in weight gain during exercise.

**Fluid type**: Fluid from both food and beverages contribute to the overall hydration status. Water is an appropriate choice for most athletes; however, athletes who compete in multiple competitions, such as tournament play or those who have restricted dietary intake or skipped prevent meals, may benefit

### TABLE 5.4 COMMON FOOD SOURCES OF IRON

| Food | Serving Size | Iron (mg) |
|---|---|---|
| Fortified ready-to-eat cereals | 1 oz | 23.8 |
| Fortified instant cooked cereals | 1 packet | 4.9–8.1 |
| Soybeans, mature, cooked | ½ cup | 4.4 |
| Pumpkin and squash seed kernels, roasted | 1 oz | 4.2 |
| Lentils, cooked | ½ cup | 3.3 |
| Spinach, cooked from fresh | ½ cup | 3.2 |
| Beef, cooked | 3 oz | 3.0 |
| Kidney beans | ½ cup | 2.6 |
| Chickpeas, cooked | ½ cup | 2.4 |
| Lamb | 3 oz | 2.3 |
| Prune juice | ¾ cup | 2.3 |
| Refried beans | ½ cup | 2.1 |

Nutrient values from Agricultural Research Service (ARS) nutrient database for standard reference, Release 17

### BOX 5.2 CALCULATING SWEAT RATE IN ATHLETES

An athlete can calculate sweat rate by using this equation: Sweat Rate = (pre-exercise weight – post-exercise weight) + (fluid intake – urine volume)/exercise time in hours. This equation will determine an hourly sweat rate. Two examples are listed below:

$$(71\,kg - 70\,kg) + (4\,L - 1L)/2\,Hr = 1.5\,L/Hr$$

$$(125\,kg - 121\,kg) + (5\,L - 1.5\,L)/3\,Hr = 1.2\,L/Hr$$

### TABLE 5.5 CALCIUM CONTENTS OF DAIRY AND NONDAIRY FOOD SOURCES

| *Dairy Food Sources* | | | *Nondairy Food Sources* | | |
|---|---|---|---|---|---|
| **Food** | **Serving Size** | **Calcium (mg)** | **Food** | **Serving Size** | **Calcium (mg)** |
| Plain yogurt | 8 oz | 452 | Fortified cereals | 1 oz | 236–1043 |
| Romano cheese | 1.5 oz | 452 | Soy beverage | 1 cup | 368 |
| Fruit yogurt | 8 oz | 345 | Sardines | 3 oz | 325 |
| Swiss cheese | 1.5 oz | 336 | Tofu, firm | ½ cup | 253 |
| Ricotta cheese, part skim | ½ cup | 335 | Spinach | ½ cup | 146 |
| American cheese, processed | 2 oz | 323 | Soybeans, cooked | ½ cup | 130 |
| Cheddar cheese | 1.5 oz | 307 | Oatmeal, instant, fortified | 1 packet | 99–110 |
| Skim milk | 1 cup | 306 | Cowpeas | ½ cup | 106 |
| 2% reduced-fat milk | 1 cup | 285 | White beans, canned | ½ cup | 96 |
| Chocolate milk | 1 cup | 280 | Rainbow trout, cooked | 3 oz | 73 |

Nutrient values from Agricultural Research Service (ARS) nutrient database for standard reference, Release 17

from consuming a sport drink with a carbohydrate concentration of 6%–8%. In addition, the sodium found in sports drinks may help with fluid retention. For exercise bouts that last >60 minutes, sports drinks can help maintain hydration and performance by maintaining blood glucose and by sparing muscle glycogen.

## NUTRIENT TIMING

### Pre-event Nutrition

- The pre-exercise meal should be primarily composed of carbohydrates and be moderate in protein and low in fiber and fat; this recommendation is based on the digestion rates of these macronutrients. Carbohydrate is digested most rapidly, followed by protein and fat.
- The meal or snack consumed before exercise should be sufficient enough so that the athlete is not hungry during exercise but not so large as to leave undigested food in the stomach. The selection of food, water, or a sports drink to be consumed 1–4 hours before exercise should be based on the athlete's preference and competitive situation.
  - Consume smaller meals closer to the event to allow the stomach to empty and nutrients to be absorbed, while larger meals can be consumed after exercise. In general, a carbohydrate feeding of 1.0 g/kg (0.5 g/lb) is appropriate 1 hour before exercise, whereas 3.0–4.0 g/kg (1.5–1.8 g/lb) can be consumed 3–4 hours before exercise.
- Athletes' individual differences must be identified and needs met. Certain athletes can consume large amounts of food just before exercise, while others may need the full 4 hours before exercise to digest their meal and feel ready for performance. Athletes usually know what works or what does not work for them. If they want to try new foods, they should first experiment it in training before using the strategies during competition.

### During Exercise

- During brief exercise bouts lasting <45 minutes, no carbohydrates must be consumed.
- For optimal hydration during exercise, athletes should drink enough fluids to replace fluids that are lost in sweat (see Box 5.2). Fluids lost in sweat should not exceed >2% of body weight so as to not affect performance.
- Consumption of colder fluids may reduce core temperature and improve performance in hot environments.
- Depending on the length of the exercise or training bout, drinking a sports drink with 6%–8% carbohydrates can provide a source of fuel for the exercising muscles during exercise. For exercise, including stop-and-start sports lasting 1–2.5 hours, the recommended carbohydrate intake is 30–60 g/h, which can be consumed in the form of sports drinks or food based on the nature of the sport.
- For ultra-endurance exercise that lasts >3 hours, the recommended carbohydrate intake is up to 90 g/h. Research has found that a mixture of different sugars, such as glucose, sucrose, fructose, maltodextrins, or dextrose, may be most effective for carbohydrate oxidation.

### Recovery Nutrition

- The goal of postexercise recovery is to restore fluids and nutrients used during exercise and restore the body back to its pre-exercise status.
- Recovery of glycogen levels is a priority after exercise. To refill muscle glycogen, time and adequate carbohydrates are required. Glycogen resynthesis rate is the highest within the initial 4–6 hours after exercise; approximately 1–1.2 g of carbohydrates/kg/h should be consumed to maximize glycogen.
- Postexercise protein consumption, particularly after strength and endurance sports, has been shown to be beneficial in stimulating MPS.
- For practical purposes, combining carbohydrates and protein in a postexercise recovery snack will benefit both muscle glycogen restoration and MPS.

### Nutrition and the Injured Athlete

- Injury results in inflammation in an athlete. While inflammation is a necessary response for the healing process, chronic inflammation slows healing and may be damaging.
- Modifications to an injured athlete's diet may include a reduction in calories. Weight gain during an injury increases inflammation and may slow the healing process.
- An injured athlete needs to eat a variety of foods that focus on antioxidant-rich fruits and vegetables as well as foods high in omega-3 fatty acids. Table 5.6 lists the foods that reduce inflammation.

## SUMMARY

- Food and fuel recommendations for athletes has moved into the era of evidence-based science and the employment or consulting services of registered dietitians (RDs) who are board certified in sports nutrition (CSSD).
- All sports medicine teams should include a sports dietitian (RD, CSSD) to educate athletes about what foods to eat, how much to eat, and to provide recommendations regarding the timing of food for pregame and postgame meals, which are of prime importance for athletic performance.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

**TABLE 5.6 FOODS THAT DECREASE INFLAMMATION**

| Animal | Fruits/Vegetables | Oils | Nuts | Herb/Spice | Others |
|---|---|---|---|---|---|
| Salmon | Tomatoes | Olive oil | Almonds | Ginger | Dark chocolate |
| Mackerel | Spinach | Omega-3 fatty acids | Walnuts | Turmeric | Wine and alcohol |
| Tuna | Kale | | Pistachios | | Garlic |
| Sardines | Collards | | | | Onions |
| | Beets | | | | |
| | Blueberries | | | | |
| | Strawberries | | | | |
| | Tart cherries | | | | |

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# 6 SPORTS SUPPLEMENTS

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## PRODUCT OVERSIGHT AND MARKETING

### Dietary Supplement Health and Education Act of 1994 (DSHEA)

#### Food and Drug Administration (FDA)

- Regulates dietary supplements under separate regulations from those that cover "conventional" foods and drug products (prescription and over-the-counter)
- Under the DSHEA, dietary supplement manufacturers are responsible for ensuring that the product is safe before it is marketed.
- The FDA is responsible for taking action against any unsafe product after it reaches the market.
- Unlike other drugs, manufacturers need not register or get approval from the FDA before producing or selling dietary supplements.
- It is the manufacturer's responsibility to ensure that product label information is truthful and not misleading; the manufacturer also dictates product purity.
- The FDA has established a Dietary Supplements Guideline in 2007 for current Good Manufacturing Practices (cGMP) for dietary supplements. Established guidelines require that dietary supplements be produced in a quality manner, not contain contaminants or impurities, and have accurate labeling.
- Postmarketing responsibilities of the FDA include monitoring safety (voluntary dietary supplement adverse event [AE] reporting) and inspecting product information (claims, labeling, package inserts, and accompanying literature). These guidelines do not address the underlying safety of the supplement itself and remain nonbinding to the manufacturer.

#### Federal Trade Commission (FTC)

- Responsible for overseeing truth in dietary supplement advertising
- Requires that claims on products be symptom-specific and not disease-oriented; for example, statements such as "supplement X can stimulate immune system" are acceptable, but statements such as "supplement X can treat, cure, or resolve infections" are not.
- Despite the FTC requirement that claims on products be symptom-specific and not disease-oriented, one study that analyzed internet websites to assess the nature of marketing claims for the eight best-selling herbal products found that this rule is not always followed. The study revealed that most available information is derived from vendor sites and that half of these sites claim that these products can treat, prevent, diagnose, or cure specific diseases. Physicians should be aware that these claims appear on the first page of the most commonly used internet search engines.

#### Center for Food Safety and Applied Nutrition Adverse Event Reporting System (CAERS)

- The Center for Food Safety and Applied Nutrition (CFSAN) maintains an AE monitoring system known as CAERS.
- The primary reporting system established by the FDA is a voluntary reporting system; according to a report by the Office of the Inspector General, <1% of all AEs are reported through the CAERS.
- Dietary supplements are not evaluated for safety, and manufacturers are not required to prove safety. It is the FDA's responsibility to prove harmful consequences.
- In 2006, the Dietary Supplement and Nonprescription Act mandated reporting of serious AEs by supplement manufacturers (deaths or life-threatening events, initial hospitalizations or prolongation of stay, disabilities or permanent impairments, and congenital anomalies or birth defects), requiring supplement labels to include the manufacturer's contact information.
- In 2009, the FDA received only 596 serious and 353 mild or moderate AE reports during a 10-month period in 2008; however, according to the American Association of Poison Control Centers, the estimated annual number of AEs in the United States is closer to 50,000. Therefore, all supplement use should be closely scrutinized in patients who are pregnant or breastfeeding and in children, unless specifically noted.

### Other Oversight

- U.S. Pharmacopoeia (USP) has set standards for natural product potency ranges.
- Currently, only 6 brands of dietary supplements have been verified by the USP.
- ConsumerLab (www.consumerlab.com) is a helpful site that tests supplements from various companies and reports their potencies.

### Marketing

- Since the passage of the DSHEA in 1994, the number of dietary supplements sold in the United States (US) has increased from 4000 to >90,000 in 2014.
- Dietary supplements are estimated to be a US $104 billion industry worldwide (US $30 billion in the US).
- Supplements are marketed to athletes' fears. Several athletes believe that they have to use supplements to stay equal to competitors or to gain a "competitive edge." They frequently fear that competitors are using supplements. Over 50% of Olympic-caliber athletes stated that they would take a banned substance if it meant they would win every competition for the next 5 years, even if they would then die from adverse effects of the substance. Among elite athletes, performance differences are minuscule between first- and fourth-place winners; even minor enhancements may mean the difference between victory and defeat.
- Marketing companies rely heavily on testimonials of personal experiences, particularly from famous people and athletes. Many companies successfully sell unproven products using this approach. Supplement manufacturers often sponsor supplement studies, and negative findings may not be published. Word of mouth and hopes to gain a "competitive edge" help fuel sales.
- Sports supplements frequently have no "instant" effects; hence, companies often add stimulants to provide an "energy boost." Despite a new FDA label law to ensure accuracy in labeling of dietary supplements, there continues to be inaccurate labeling. In an FDA analysis of ephedra supplements, 6–20 other ingredients were identified. Cases have been reported of legal supplements containing trace amounts of illegal supplements. It is truly a "buyer-beware" market (Box 6.1).

**BOX 6.1 "BUYER-BEWARE" INFORMATION ABOUT SUPPLEMENTATION**

- Most ergogenic aids lack scientific proof.
- Most supplements have not been adequately tested for efficacy, purity, or safety.
- Be careful of misleading product information.
- "Natural" does not mean safe.
- "More" is seldom better.
- Nothing replaces a well-balanced diet that includes a variety of high-quality foods.
- Athletes use supplements at their own risk.

## COMMONLY USED ATHLETIC PERFORMANCE SUPPLEMENTS

- Optimal dose and long-term side effects of most supplements are not known.
- Manufacturers recommend doses and durations that have been tested and claim that side effects apply to these instructions.
- Several athletes may use higher doses than recommended and/or use them for longer periods of time, which raises concerns regarding unknown effects.
- Most supplements try to enhance the normal effects of exercise on the body.

### Arginine

**Claims:** Acutely improve exercise capacity; chronic effects result from stimulation of muscle protein synthesis and anabolism of muscle protein. Soy, sesame, and peanut proteins are an excellent source of arginine.
**Mechanism:** May promote secretion of endogenous growth hormone (GH); precursor in the synthesis of creatine; augments the production of nitric oxide
**Efficacy:** The scientific evidence available to support claims of promoting and increasing functional capacity in healthy, athletic participants is limited. Intravenous arginine increases GH, but oral arginine has not shown the same effect. It may increase nitric oxide production, but definitive studies are warranted to confirm the same. Moreover, it significantly increases muscle blood volume but does not affect strength performance. Effects on muscle protein synthesis are likely a net effect in combination with nitric oxide as well as concurrent elevation of other amino acids.
**Side effects:** None reported in short-duration studies for oral use; occasionally, flushing reported with IV administration
**Dosage:** 3–9 g/day or 250 mg/kg/day (as used in previous studies)

### Bovine Colostrum (BC)

**Claims:** BC supplementation may increase insulin-like growth factor-1 (IGF-1) levels. May positively influence exercise performance characterized by short bursts of activity; does not improve body composition in elite athletes but shows improvements in nonelite athletes; claims also include increased immune function after exercise
**Mechanism**: BC stimulates growth factors, including structurally identical IGF-1, which has an anabolic effect and is involved in the regulatory feedback of GH. GH stimulates hepatic production of IGF-1, which in turn provides negative feedback to reduce pituitary production of GH.
**Efficacy:** Limited studies have shown consistent beneficial effects of BC on recovery and exercise performance and improved immune function in special athletic populations.
**Side effects:** Occasional minor gastrointestinal (GI) complaints, including flatulence and nausea; a high proportion of participants complain about the "unattractive" taste of the beverage
**Dosage:** 20–60 g/day

### Branched-Chain Amino Acids (BCAAs)

**Claims:** Important source of energy in prolonged endurance exercise; proposed to increase endurance in long tennis matches, soccer, marathons, long-distance swimming, and cycling activities; may contribute to increased body fat loss and maintenance of a high level of exercise performance; claims to decrease chronic fatigue/overtraining symptoms
**Mechanism:** Replenishes loss of BCAAs used as fuel, increases protein synthesis and GH secretion, shifts leucine metabolism to fat metabolism, stimulates fat metabolism over glycogen in hypocaloric diets, and prevents decrease in plasma glutamine; inhibits dietary tryptophan transport across the blood–brain barrier, leading to decreased brain serotonin (associated with several brain regions that control central fatigue)
**Efficacy:** Most studies have reported neither beneficial nor detrimental effects of BCAA; studies on the effects of BCAAs in hypocaloric states are limited. BCAAs have not been shown to reduce chronic fatigue/overtraining symptoms, and preworkout/event BCAA loading has no effect on performance.
**Side effects**: Fatigue and ergolytic effects have been reported.
**Dosage:** Usually combined with other amino acids; range: 5–10 g/day (preferably before exercise)

### Carbohydrate Supplements

**Claims:** Used to restore muscle glycogen after exercise, maintain plasma glucose during endurance events (particularly those lasting >90 minutes), and to maximize muscle glycogen before significant glycogen-depleting activities (e.g., marathons or long-course triathlons); various sugars are used, including sucrose, glucose, fructose, and maltodextrin (popular among ultra-endurance athletes).
**Mechanism:** Increased blood glucose stimulates insulin production and GLUT-4 translocation in muscle, which results in increased glucose uptake and glycogen storage in muscle. Carbohydrates with a high glycemic index increase plasma glucose quickly and serve as a fuel source during sustained exercise.
**Efficacy:** Mixed reviews for pre-exercise supplementation and carbohydrate loading; benefits of supplementation after exercise and during long events (>90 minutes) are well supported.
**Side effects:** Individual GI tolerance varies with different types of carbohydrate supplements, and some athletes may experience dyspepsia and GI upset.
**Dosage:** Before exercise (benefits of carbohydrate loading controversial): 4 g/kg within 3 hours before and 1.1 g/kg 1 hour before; 10 g/kg/day of carbohydrates 3–7 days before a sport event. During exercise: 0.7–1.0 g/hour for events lasting >1 hour; sources include sports drinks (5–10 ounces every 15 minutes), sports gels or candies (2 gels and water), or gummy candy (a handful per hour and water). After exercise: 7–1.0 g/kg every 2 hours for the first 4 hours after exercise (first 90 minutes after exercise is the most important); best if started within 30 minutes of stopping exercise. Use a food source with a high glycemic index; addition of protein to the carbohydrate supplement increases glycogen production.

### Chromium

**Claims:** Trace mineral used for weight loss and for enhancement of glycemic control in the treatment of diabetes; proposed for the treatment of hyperlipidemia and hypercholesterolemia; used by athletes in attempts to gain muscle and lose fat

**Mechanism:** Functions in carbohydrate, protein, and fat metabolism as a cofactor that enhances action of insulin and uptake of amino acids into muscles; improves lipid profile and is theorized to sensitize insulin receptors in the brain, resulting in appetite suppression and down-regulation of insulin secretion; glycogen synthesis increases in chromium-deficient individuals. Exercise may result in loss of chromium, but athletes conserve chromium and probably do not develop deficiencies.

**Efficacy:** Possibly effective when used to reduce cholesterol, but probably ineffective for weight loss; mild hypoglycemic effect caused by a mechanism similar to metformin; considerable scientific evidence indicates that chromium has no effect on body composition when taken in the form of a supplement, and there are serious concerns regarding the potential adverse effects of chromium accumulation within the body, particularly with long-term use

**Side effects:** Chromium interferes with iron metabolism and zinc absorption. Prolonged use and abuse linked with serious side effects, including anemia, chromosomal damage, cognitive impairment, interstitial nephritis, GI intolerance, tremor, and insomnia; commercial preparations containing ephedrine restricted; low doses of the combined preparation have been found to cause hypertension, stroke, and death

**Dosage:** Chromium picolinate is more easily absorbed than other forms of chromium; chromium is complexed to picolinate to facilitate absorption. Common dose is 50–200 (mean, 120) mcg/day; similar dosing often found in multivitamins. Lower doses may be safer than higher doses.

## Creatine

Probably the most frequently used and most researched supplement consumed by athletes

**Claims:** Creatine may increase exercise performance in short repetitive bouts of high-intensity exercise offset by brief rest periods (30–120 seconds). Increase in exercise performance and work capacity probably leads to increased muscle mass in some athletes.

**Postulated mechanism:** Creatine is a low-molecular-weight, complex amino acid endogenously produced primarily in the liver and stored primarily in skeletal muscles. Hydrolysis of creatine phosphate results in rapid production of adenosine triphosphate (ATP), which is needed for muscle contraction. Maximal muscle stores of total creatine may enhance the ATP turnover rate and increase phosphocreatine resynthesis, resulting in shorter recovery periods and overall increased training load (volume/intensity). Creatine depletion is a limiting factor of anaerobic exercise. Free creatine may stimulate protein synthesis and cause muscle hydration, which results in increased muscle mass and strength.

**Efficacy:** Numerous studies have examined the effects of creatine supplementation on athletic performance. Despite some disagreement, general consensus is that creatine supplementation has a small, but real, beneficial effect on anaerobic activity, specifically during short-duration, repetitive, high-intensity exercises. Does not benefit aerobic training or performance and does not alter maximal force production; data that address chronic creatine supplementation, high-dose supplementation, and supplementation in young athletes are lacking. Reported to increase muscle phosphocreatine content by up to 20%, but this does not mimic physiologic changes related to training; apparently, there are responders and nonresponders to creatine; specifically, vegetarians who do not ingest primary exogenous sources of creatine (meat and fish) may benefit more from creatine supplementation.

**Side effects:** No serious side effects have been consistently documented from creatine supplementation when used for up to 6 months. Weight gain is a proven side effect, and areas of theoretical concern and anecdotal reports include the following:

- Renal
  - Creatine can spontaneously degrade to creatinine, and increases in both urine and serum creatinine levels have been reported. Elevations are likely brief and clinically insignificant.
  - Athletes with history of renal dysfunction or diseases that may lead to renal dysfunction (e.g., diabetes) should use creatine with caution.
  - Athletes using potentially nephrotoxic drugs (e.g., nonsteroidal anti-inflammatory drugs [NSAIDs]) may be at a higher risk of renal dysfunction.
  - Close monitoring of renal function should be considered as long-term effects of creatine on the kidney are unknown.
- Gastrointestinal
  - Nausea, bloating, cramping, and diarrhea have been reported by users, but effects are not supported by clinical studies.
  - No hepatic dysfunction reported
- Cardiovascular
  - The amount of creatine taken up by the myocardium is unknown. Animal studies that report no substantial myocardial uptake also report no skeletal muscle uptake.
  - Whether this is detrimental or possibly even beneficial remains unknown.
- Dehydration
  - Anecdotal reports of dehydration, particularly in hot, humid conditions; hence, adequate hydration in creatine users encouraged
  - May not represent true dehydration because creatine increases total body water proportion, but osmotic properties of creatine may increase third spacing, depleting intravascular fluid volume
  - High-dose creatine best avoided during periods of increased thermal stress
- Muscular
  - Anecdotal reports mention increased muscle cramping and strains but remains unproven
  - Fluid retention that may accompany creatine ingestion, particularly with loading doses, may theoretically increase compartmental pressure and predispose athletes to exertional compartment syndrome

**Dosage:** Some experts recommend loading doses of 20–30 g/day (5–7 g four times/day) for 5–7 days (an amount of creatine equal to the amount in 5–6 pounds of beef), followed by doses of 2–4 g/day to maintain intramuscular creatine stores. Other investigators have shown that intramuscular creatine stores reach the same level with a dose of 3 g/day, and these stores are maintained with as little as 2 g/day with no loading phase. Lower doses take longer to attain desired intramuscular creatine levels. Dosing may be based on body weight: loading dose of 0.3 g/kg/day with a maintenance dose of 0.03 g/kg/day. The "more is better" philosophy held by many athletes remains a concern because of the "ceiling" for muscle storage (5 g creatine/kg muscle mass) and excess creatine is not used by the muscles.

## Fluid Replacement Beverages

**Claims:** Used to prevent and treat dehydration; dehydration >3% decreases maximal aerobic power by 5%

**Mechanism:** Prevents dehydration/hypohydration and associated effects; prevents heat intolerance, maintain stroke volume, cognitive functioning, strength, and work capacity

**Efficacy:** Numerous studies have reported decreased performance in "hypohydrated" athletes. Recent studies have demonstrated that hypohydration decreases strength.

**Side effects:** Overhydration may cause hyponatremia, but for hyponatremia to occur, the hydration must be excessive and occur over a long period (e.g., endurance or ultra-endurance events that last >4 hours). GI upset may occur, particularly with fructose-containing fluid replacement drinks.

**Dosage:** American College of Sports Medicine recommends consumption of 400–600 mL of water 2 hours before exercise and 150–300 mL every 15–20 minutes of exercise. Additional consumption is needed in climates associated with high sweat rates. Drinking to thirst important; can also monitor urine color and volume and body weight (BW). Addition of carbohydrates is recommended for activities lasting >90 minutes. Hydration after exercise is also important. For athletes who need rapid recovery from dehydration, it is recommended to drink 1.5 L/kg of BW lost during exercise.

## Beta-Hydroxy Beta-Methylbutyrate (HMB)

**Claims:** Regulate protein metabolism, decreasing catabolism, increasing lean muscle mass and strength; often used to enhance esthetic and physical appearance in bodybuilding

**Mechanism:** Exact mechanism unknown; HMB is a metabolite of leucine, a BCAA, and may regulate enzymes responsible for protein breakdown, inhibiting breakdown of muscle during and after vigorous activity. HMB in liver and muscle cells is metabolized to HMG-CoA, which is then used in the synthesis of cholesterol, which increases the availability of cholesterol for cell wall synthesis. Localized deficiency of cholesterol for cell wall synthesis is postulated as a restriction to muscle hypertrophy, and increased local cholesterol stores could theoretically relieve this restriction. HMB may also undergo polymerization, stabilizing the cell membrane as it may be used as a structural component. Also proposed to increase muscle cell fatty-acid oxidation through unknown mechanisms and lead to a decrease in fat mass

**Efficacy:** Few scientific investigations published; may have additive effects when combined with creatine; studies have demonstrated certain benefits in untrained athletes but no significant improvement in trained athletes.

**Side effects:** No reported side effects, but research seems inadequate

**Dosage:** 1.5 g administered 1–3 times daily (2–3 g/day) in clinical trials

## Carnitine

**Claims:** Increase aerobic and anaerobic capacity and promote fat loss

**Mechanism:** Increases long-chain fatty acid oxidation in skeletal muscles during exercise

**Efficacy:** Clinical trials inconclusive and suffer from design limitations; small-scale studies have reported no alteration in L-carnitine levels in muscles following supplementation or after a single prolonged exercise session. Twenty years of research revealed no consistent evidence that carnitine supplements can improve exercise or physical performance in healthy individuals.

**Side effects:** Nausea, vomiting, abdominal cramps, diarrhea, and a "fishy" body odor; rare side effects include muscle weakness in uremic patients and seizures in individuals with known seizure disorder.

**Dosage:** 2–6 g/day in 2–3 doses with meals

## L-Glutamine

**Claims:** L-glutamine is the most abundant amino acid in the body. Is used in treatment of wound healing, immune function, and chemotherapy-induced stomatitis; athletes use it to prevent impaired immune responses following prolonged exercise.

**Mechanism:** Originally classified as a nonessential amino acid, glutamine is now considered essential for maintaining intestinal function, immune response, and amino acid homeostasis during times of stress. Is an important fuel for cells of the immune system (lymphocytes and macrophages); during prolonged exercise, as in other forms of chronic stress, plasma glutamine may decrease. Muscle glutamine may drop in an effort to sustain an anabolic state; if glutamine drops below critical levels, athletes may revert to a catabolic state.

**Efficacy:** Human and animal studies have reported conflicting results. Lack of reliable data for most proposed uses; preliminary data suggest that glutamine supplementation may enhance immune function. Glutamine has been shown to reduce upper respiratory infections in athletes after vigorous exercise, but additional research is warranted to confirm this finding. May be effective in treating chemotherapy-induced stomatitis

**Side effects:** No significant adverse reactions reported; may be safe at appropriate doses; occasional GI upset reported

**Dosage:** Typical dose: 20–30 g/day; tolerated without side effects at doses up to 40 g/day

## Nitric Oxide (NO)

**Claims:** Nitric oxide (NO) thought to increase muscular strength and endurance; has been shown to be beneficial in patients with cardiac disease and endothelial dysfunction; marketed to athletes with claim that vasodilation associated with NO improves muscular vascular perfusion and that increase in blood flow improves muscular gains with resistance training

**Mechanism**: Nitric oxide (NO) is produced in the body by an enzyme called *nitric oxide synthase*, which converts the amino acid L-arginine to nitric oxide and L-citrulline. Acts through vasodilatation, facilitating blood flow to muscle cells; also bactericidal, released by macrophages; a combination of these two effects observed in septic shock

**Efficacy:** Small subject numbers and lack of standardization of previous activity levels make current studies inconclusive.

**Side effects:** None identified in short-term studies

**Dosage:** Supplements rely on the conversion of an intermediate to NO, most often arginine, typically used at 3–6 g/day

## Protein Supplements

**Claims:** Protein supplementation above American Dietetic Association (ADA) recommendations (0.8 g/kg/day) is used to prevent negative nitrogen balance and to aid protein synthesis, particularly during high-intensity exercise. Numerous athletes, particularly weight lifters, use protein supplements to "bulk up" or to add muscle mass. Most frequently used varieties include whey, soy, or egg whites.

**Mechanism:** Protein supplements aid synthesis of new muscle proteins. Whey protein is a good source of BCAAs, which were discussed previously.

**Efficacy:** Whey protein is a soluble, easy-to-digest protein. Most studies show change in muscle synthesis with increased protein intake, but subjects were not tested for increased strength. Athletes require more protein than nonathletes (see Chapter 5: Sports Nutrition), and protein supplements may be used as dietary adjuncts.

**Side effects:** None documented at doses up to 2 g/kg/day in healthy individuals, but sustained use at this level is concerning. Caution recommended in athletes with renal insufficiency or failure as well as in individuals with lactose or dairy protein allergies; excessive protein intake stored as fat

**Dosage:** Recommended dose for recreational athletes is 0.8–1.0 g/kg/day, for endurance athletes is 1.2–1.4 g/kg/day, and for strength-trained individuals, 1.6–1.7 g/kg/day.

### Ribose

**Claim:** Several roles in human physiology; necessary substrate for synthesis of nucleotides and is a part of the building blocks that form DNA and RNA molecules; claims that it increases synthesis and reformation of ATP, improves high-power performance as well as recovery and muscle growth, and quickly restores energy levels in heart and skeletal muscles

**Mechanism:** Structural component of ATP, which is the primary energy source for exercising muscle; during intense muscular activity, total amount of available ATP is quickly depleted; estimated to take approximately 3 days to restore ATP levels to baseline; ribose helps to restore the level of adenosine nucleotides, and supplementation has been shown to increase the rate of ATP resynthesis following intense exercise.

**Efficacy:** Studies for various sports performance parameters; most evaluate effects on anaerobic cycle sprints, with limited studies on strength and endurance. Consistent ergogenic benefit not identified

**Side effects:** Headache, nausea, hyperuricemia, and hyperuricosuria; doses >200 mg/kg/h may cause diarrhea. Precautions should be taken with ribose and diabetes because all simple sugars increase insulin levels**.** Supplementation may cause hypoglycemia in patients with diabetes

**Dosage**: 5–10 g/day, but markedly different ribose dosages have been used in studies. Lack of consensus on recommended dose of ribose

### Tribulus Terrestris

**Claim:** Herb claimed to "naturally" increase testosterone levels

**Mechanism:** Postulated to increase release of luteinizing hormone (LH), indirectly stimulating testosterone release

**Efficacy:** Studies do not support claims of improved body composition or athletic performance. Elevation of serum testosterone may have anabolic effects.

**Side effects:** No reported side effects in humans; photosensitivity has been reported in animals grazing on tribulus terrestris. Potential anabolic side effects if it elevates testosterone

**Dosage:** 500–650 mg tablets for once-daily dosing frequently recommended; often combined with other "prohormones"

## LESS COMMONLY USED ATHLETIC SUPPLEMENTS

### Caffeine

**Claims:** Most widely used stimulant in the world and is present in beverages such as coffee, teas, colas, certain energy drinks, foods containing chocolate, energy bars, and over-the-counter medications sold to increase alertness; central nervous system (CNS) stimulant that increases alertness and can make intense exercise feel easier. Preworkout formulas usually contain caffeine; mildly elevates fat burning and metabolic rate**;** some claims state that mild caffeine consumption may decrease the risk of diabetes

**Mechanism:** Lipid-soluble compound metabolized by liver and through enzymatic action results in three metabolites: paraxanthine, theophylline, and theobromine; crosses the blood–brain barrier owing to lipid solubility; acts on the CNS as an adenosine antagonist but may also have peripheral effects on substrate metabolism and neuromuscular function

**Efficacy:** Potential improvement in endurance exercise, high-intensity team sports, and strength-power performance; studied in special force operations where military personnel routinely undergo training and real-life operations in sleep-deprived conditions; vigilance maintained or enhanced with caffeine in addition to run times and completion of an obstacle course; may also be ergogenic by enhancing lipolysis and decreasing reliance on glycogen utilization; effects of caffeine and exogenous carbohydrate intake during endurance exercise requires more research.

**Side effects:** Lethal dose in adult humans is estimated to be 10 g. Higher doses of caffeine may induce mild tremor, tachycardia, insomnia, GI upset, chest pain, arrhythmias, and nervousness at doses >600 mg/day.

**Dosage:** Typically used at 200 mg during late evening, overnight, and early morning periods over 3 days (up to 800 mg/day); single doses of approximately 200 mg improve cognitive function and doses of 2–6 mg/kg of BW can enhance physical performance.

### Ephedra

**Claims:** Synthetic form, pseudoephedrine, is a common ingredient in over-the-counter and prescription cold and allergy medications, now available "behind-the-counter"; claims to increase body fat loss (aid in weight loss), improve athletic performance, and improve concentration; not approved in the US for weight loss or to enhance athletic performance

**Mechanism:** The Chinese botanical ephedra, or ma-huang, is the common name for three species: *Ephedra sinica*, *Ephedra equisetina*, and *Ephedra intermedia*. The active compounds, present in the plant's stem, are the phenylalanine-derived alkaloids ephedrine, pseudoephedrine, phenylpropanolamine (norephedrine), and cathine (norpseudoephedrine). Ephedrine is a mixed sympathomimetic agent that enhances the release of norepinephrine from sympathetic neurons.

**Efficacy:** Ephedrine stimulates heart rate and increases cardiac output, causes peripheral constriction, increases peripheral resistance (leading to a rise in blood pressure), relaxes bronchial smooth muscle, and temporarily relieves shortness of breath caused by asthma. May act on the satiety center in the hypothalamus functioning as an anorectic; no studies have assessed the effect of dietary supplements containing ephedra and botanicals on athletic performance, and only a few studies have demonstrated a modest effect of ephedrine plus caffeine on short-term athletic performance in a select physically fit population.

**Side effects:** Ephedra alone is not sold in the US. Only side effects published are on ephedrine, ephedrine plus caffeine, or ephedra plus caffeine, which report a 2–3 times risk of nausea, vomiting, psychiatric symptoms (anxiety and change in mood), autonomic hyperactivity, and palpitations. Certain case reports have connected ephedra with serious adverse effects such as heart attack, stroke, seizures, and death.

**Dosage:** Usually combined with caffeine and occasionally with aspirin; ephedrine is consumed at a concentration of 20–24 mg (usually 3 times/day) in these compounds.

### Ginseng

**Claims:** The Chinese have used Ginseng for thousands of years as a stimulant, a diuretic, to promote menstruation, and to fight infection. Other claims address treatment of adrenal and thyroid dysfunction and aphrodisiac qualities. Athletes use it to enhance aerobic performance, energy level, and body resistance to stress.

**Mechanism:** Hypothesized to stimulate hypothalamic-pituitary-adrenal axis, which may result in increased resistance to various types of stress; may enhance myocardial metabolism, increase oxygen extraction by muscles, and optimize mitochondrial metabolism in muscle; during strenuous exercise, ginseng may mitigate catabolic effects of the stress hormone cortisol and enhance the body's ability to sustain muscle creatine phosphate levels, thereby decreasing lactic acid production.

**Efficacy:** No quality evidence supports the claim that ginseng supplementation enhances physical performance.

**Side effects:** Most common side effects are nervousness and excitability that may lead to insomnia with decrease in this effect after the first few days. Many people find its taste unpleasant. Hypoglycemia and difficulty in concentrating reported; because of estrogen-like effects, women who are pregnant or breastfeeding should not consume it. Occasional reports of more serious side effects, such as asthma attacks, increased blood pressure, and palpitations and uterine bleeding in postmenopausal women

**Dosage:** Doses ranging 0.3–6 g/day have been studied. The most commonly used dosages are 100–200 mg three times daily. Study of 50 commercial products revealed a level of active ingredient ranging 1.9%–9%, whereas 6 products showed no evidence of ginseng.

### Glutathione

**Claims:** Stimulation effects of glutathione may counteract free radical production due to extreme physical training

**Mechanism:** Hepatic glutathione production increases with exercise duration. Liver uses glutathione to remove vitamin E radicals and maintains vitamins C and E in their reduced (active) forms.

**Efficacy:** Effectiveness of aerosolized, intramuscular, or intravenous glutathione not well established; a randomized, double-blind, placebo-controlled clinical trial found no significant changes in biomarkers for oxidative stress with oral glutathione supplementation. Glutamine and N-acetylcysteine being studied to determine if they increase glutathione levels

**Side effects:** Not well studied; long-term glutathione linked to low zinc levels; inhaled glutathione may trigger asthma attacks

**Dosage:** Oral glutathione not bioavailable and standard dosages have not been established

### Glycerol

**Claims:** Oral ingestion may induce a state of "hyperhydration," resulting in superior athletic performance.

**Mechanism:** Simple polyol (sugar alcohol) that acts as an osmotic agent and increases water retention

**Efficacy:** Results equivocal regarding benefits of glycerol-induced hyperhydration on core temperature, plasma volume, and exercise tolerance; hyperhydration does not appear to offer additional benefits compared with euhydration; hyperhydrated athletes may be less likely to become dehydrated, particularly during exercise in extreme environmental conditions of high heat and humidity. Adverse effects of hyperhydration are concerning (e.g., electrolyte shifts), and practice should be to never replace appropriate oral hydration, appropriate acclimatization, and good sense during exercise in extreme conditions.

**Side effects:** Isolated reports of headache, bloating, and nausea after oral ingestion; otherwise data limited

**Dosage:** Typically 1–1.2 g/kg mixed with 1.5 L of fluid consumed 1–2 hours before competition

### Lysine

**Claims:** Used for prevention and treatment of recurrent herpes simplex virus (HSV) labialis and as a protein supplement to improve athletic performance

**Mechanism:** Essential amino acid that inhibits the growth of HSV in vitro; important in collagen synthesis and bone formation; proposed that lysine, arginine, and ornithine increase GH

**Efficacy:** Lysine reduces healing time, severity, and recurrence of HSV labialis. Essential amino acids, such as lysine, arginine, and ornithine, have shown no ergogenic benefit in resistance or aerobic exercise. Intravenous arginine before endurance activity insufficiently studied

**Side effects:** Lysine contraindicated with renal disease or hepatic impairment due to inability to eliminate large amounts of nitrogen produced from supplemented amino acid breakdown; hypercalcemia may result from increased gastric absorption and decreased excretion. No data to support its use in children or pregnant or breastfeeding women

**Dosage:** For recurrent herpes: 1000 mg/day for 1 year or 1000 mg three times daily for 6 months; no ergogenic dose established

### Pyruvate

**Claims:** Increase exercise endurance, facilitate weight loss, and reduce body fat content

**Mechanism:** BCAAs transfer an amine group to pyruvate to form alanine, which increases lipid oxidation and decreases carbohydrate oxidation. Pyruvate may also reduce free-radical production. Enhances leg exercise endurance capacity by increasing glucose extraction by muscle

**Efficacy:** Pyruvate not well absorbed orally; studies have failed to note increase in blood pyruvate levels with supplementation. Evidence from randomized clinical trials to support efficacy in weight loss unconvincing; increased time to exhaustion with endurance exercise noted in untrained participants; additional studies are needed before a recommendation can be made.

**Side effects:** GI upset including gas, bloating, and diarrhea; one death reported after intravenous administration. Limited evidence addressing safety

**Dosage:** Typically 22–44 g/day

### Sodium Bicarbonate

**Claims:** Increase time to exhaustion and decrease sprint time during 400–1500-meter races

**Mechanism:** Buffers blood, which leads to metabolic alkalosis, decreasing the lactic acid effects

**Efficacy:** Mixed reviews published; in some studies, positive effects seen regarding power output for events lasting >1 minute and <7 minutes, while other studies showed no improvement in sprint-related performance

**Side effects:** GI symptoms of belching, bloating, and flatulence; overdose a serious risk because of electrolyte shifts associated with a large sodium intake

**Dosage:** Dosage used in studies is 200–300 mg/kg consumed 60–90 minutes before exercise.

## GENERAL HEALTH SUPPLEMENTS

### Chondroitin Sulfate

**Claims:** Used as chondroprotective agent against progressive osteoarthritis (OA), a non-cyclooxygenase inhibitor, and an anti-inflammatory agent

**Mechanism:** Unknown; in animal models, reduces inflammatory response and experimental cartilage destruction; in vitro studies show stimulation of chondrocytes to replace or repair damaged proteoglycans in the joint

**Efficacy:** Not as well studied as glucosamine; a few randomized controlled trials have shown a decrease in NSAID use for pain relief in patients with OA; however, these studies lacked a placebo arm. No evidence that the natural history of OA is altered or that surgical intervention is prevented. Large chondroitin sulfate molecules poorly absorbed with approximately 10% bioavailability

**Side effects:** Few, if any, side effects noted with use; people with shellfish allergy may experience allergic reactions because chondroitin is made from marine exoskeleton

**Dosage:** 1200 mg/day in three times daily divided dosages

## Ginkgo Biloba

**Claims:** Several proposed uses: to improve cognitive function in dementia, including Alzheimer's disease; to treat vascular insufficiency (central and peripheral), dysmenorrhea, and acute mountain sickness; and to improve sleep in depression
**Mechanism:** Free radical scavenger and inhibitor of monoamine oxidase; proposed to stimulate functional nerve cell populations and protect nerve cells from pathologic influences
**Efficacy:** May improve cognitive function in dementia, although mixed reviews on its efficacy in treatment of Alzheimer's or vascular dementia; 2009 systematic review of 36 trials on cognitive impairment concluded that evidence for clinically significant benefit was inconsistent. Possibly effective in treatment of claudication and acute mountain sickness but results from studies also inconsistent
**Side effects:** Therapeutic doses may cause GI upset, headache, dizziness, and allergic skin reactions. May increase risk of bleeding with warfarin and other anticoagulant or antiplatelet drugs
**Dosage:** 120–240 mg/day, depending on use

## Glucosamine

**Claims:** Chondroprotective against progression of OA; may have anti-inflammatory effects
**Mechanism:** In vitro, glucosamine stimulates cartilage cells to produce proteoglycans and glycosaminoglycans on a dose-dependent basis. Increases mRNA production; anti-inflammatory effects poorly understood; glucosamine is produced in the body by attachment of an amino acid group to glucose, which is acetylated to acetyl glucosamine
**Efficacy:** Glucosamine sulfate preferred form; often combined with chondroitin sulfate, but effects of combination have been inadequately studied. The two most commonly used bioavailable forms studied are glucosamine sulfate and glucosamine hydrochloride (HCl). Absorption rate approximately 80%–90%; some studies have reported decreased pain and stiffness associated with OA, but results of randomized trials variable; 2015 Cochrane review of 43 randomized controlled trials including 9110 patients with mostly knee OA revealed that chondroitin, either alone or in combination with glucosamine, resulted in statistically significant improvements in pain scores, although the effect was small. Additional well-designed trials needed
**Side effects:** None reported for glucosamine HCl; glucosamine sulfate may cause mild GI side effects, including nausea, heartburn, diarrhea, and constipation. Supplements are made from bovine and calf cartilage; possible but unlikely risk of exposure to bovine spongiform encephalopathy; increases serum glucose levels in some diabetes patients with OA
**Dosage:** 1500 mg/day in single or divided doses; intramuscular dosages of 400 mg twice weekly have been used experimentally. Dose may vary with product; dose equivalences have not been established.

## Melatonin

**Claims:** Sedative/hypnotic effects: assists in recovery from jet lag; improves sleep in blind patients and patients with other disorders that alter the sleep–wake cycle (e.g., shift workers); adjunct to chemotherapy and in treatment of depression; other claims include use as antioxidant, prevention of aging effects, increase in energy, boosting the immune system, adjunct in treatment of epilepsy, contraception, and prevention of cancer.
**Mechanism:** Naturally produced in pineal gland and promotes sleep; secreted in diurnal manner and release inhibited by light and stimulated by darkness; lowers alertness and body temperature; low levels of melatonin reported with insomnia
**Efficacy:** Certain systematic reviews have reported reduced jet lag, improved sleep, and improved circadian rhythm disturbances in blind, developmentally disabled, and autistic patients. Certain in vitro studies have supported its antioxidant effects. Insufficient data to support other claims
**Side effects:** Drowsiness; users should not operate motor vehicles or heavy machinery within 4 hours of oral ingestion. Theoretical concern about interference with gonadal development in children and adolescents; can also cause irritability, dysphoria, dizziness, and abdominal cramping; insufficient data regarding effects of long-term use
**Dosage:** Typical dosing for sleep disturbance is 0.5–6 mg at bedtime, with recommendations to start at a lower physiologic doses of 0.1–0.5 mg. Typical dosing for jet lag: 5 mg at nighttime for 3 days before flight

## Niacin (Vitamin B3)

**Claims:** Used with diet therapy to treat dyslipidemia, adjunct in treatment of peripheral vascular disease/coronary artery disease (PVD/CAD), and in prevention and treatment of niacin deficiency; claimed to augment energy during exercise; used by bodybuilders to increase superficial vascularity (causes vasodilation "flush") before bodybuilding contests; prevents niacin deficiency with isoniazid therapy
**Mechanism:** May increase homocysteine levels and modify abnormal coagulation factors that accompany PVD/CAD; B-complex vitamins such as niacin are involved in energy production during exercise via oxidative phosphorylation and the Krebs cycle.
**Efficacy:** Effective in lowering triglycerides and increasing high-density lipoproteins and may be effective for secondary prevention of heart attack or stroke; symptomatic treatment of OA
**Side effects:** Low-dose dietary supplementation can cause minor effects such as flushing, which may be accompanied by pruritus, rash, headache, and occasionally muscle pain. Higher doses associated with headache, dizziness, nausea, and vomiting; interactions may occur when combined with carbamazepine and HMG-CoA reductase inhibitors. Contraindicated in active liver disease, peptic ulcer disease, or arterial bleeding
**Dosage**: Limited data suggest that the need for B-complex vitamins may be twice the daily-recommended amount. Dietary supplement dosage is 10–20 mg/day, as an adjunct to isoniazid 20 mg/day; for hyperlipidemia, 1000–2000 mg at bedtime; for OA, 3 g/day in divided doses of niacinamide. Recommended daily allowance (RDA) is 16 mg/day and 18 mg/day for pregnant women.

## St. John's Wort

**Claims:** Used in the treatment of mild to moderate depression and obsessive-compulsive disorder; other claims include use for muscle spasms, ulcers, menstrual cramps, and as an expectorant
**Mechanism:** Reported as a weak inhibitor of monoamine oxidase-A and -B activity; inhibits reuptake of serotonin, dopamine, and noradrenaline (norepinephrine) with approximately equal affinity; 5-HT3 and 5-HT4 receptor antagonism; may act as a receptor antagonist at gamma aminobutyric acid (GABA)-A, GABA-B, glutamate, and adenosine receptor
**Efficacy:** Effective compared with placebo in short-term treatment of mild to moderate depression; possibly as effective as low-dose tricyclics and certain selective serotonin reuptake inhibitors (SSRIs; sertraline and fluoxetine); may be effective in treatment of secondary symptoms associated with depression, but studies in the US do not support its efficacy; data insufficient for treatment of obsessive-compulsive disorder; other claims lack sufficient supporting evidence

**Side effects:** Well tolerated and probably safe when used appropriately for short periods; the most common side effect is insomnia, which can be alleviated by taking it in the morning or by decreasing the dose. Other minor side effects include dry mouth, restlessness, agitation, headache, vivid dreams, dizziness, and paresthesia. Important concerns arise from drug–drug interactions, particularly involving other antidepressants. Use with SSRIs may be synergistic and might increase the risk of serotonin syndrome. Addictive effects with monoamine oxidase inhibitors (MAOIs) include hypertension, hyperthermia, confusion, and coma. Should not be used if MAOI has been used in the past 14 days

**Dosage:** Most trials have used St. John's wort with a hypericin content of 0.3% at a dose of 300 mg three times daily. Doses up to 1200 mg/day have been reported.

## Valerian

**Claims:** Purported sedative and anxiolytic effects, elevates mood, and improves concentration; other claims (from various websites) include beneficial use for treatment of tremors, epilepsy, attention-deficit hyperactivity disorder, rheumatic pain, muscle spasm, menstrual cramps, ulcers, and hypertension

**Mechanism:** Precise mechanism by which valerian root may cause sedation is not known. Some data suggest that interference with catabolism of GABA concentrations may result in its elevation.

**Efficacy:** Small-scale studies indicate that it may be beneficial in elevating mood and improving concentration and as a sedative. However, meta-analysis that included 11 randomized controlled trials studying valerian found no significant differences in any measure of insomnia compared with placebo. Insufficient data to support other claims

**Side effects:** Safe use has been documented in trials lasting for up to 28 days. Longer use associated with benzodiazepine-like withdrawal response; several reports of hepatotoxicity with longer-term use; potential side effects include drowsiness, headache, excitability, and cardiac disturbances

**Dosage:** Various doses and formulations studied and marketed; maximum dose, 15 g/day; tea, tincture, or extract in pill form

## Vanadyl Sulfate

**Claims:** Used for diabetes, hypoglycemia, and heart disease and to increase strength with weight training

**Mechanism:** Cofactor for various essential enzymatic reactions; may mimic the effects of insulin or potentiate its actions

**Efficacy:** Possibly effective in treatment of diabetes; however, systematic reviews have determined no rigorous evidence that oral vanadium improves glycemic control. No ergogenic effect in weightlifters compared with placebo. Insufficient scientific evidence to support other claims

**Side effects:** Possibly safe in small doses for short-term use; minor side effects include green discoloration of tongue and GI upset. Serious side effects have been reported with high doses, including leukocytosis and manic-depressive disorder. Potentiates warfarin and digoxin

**Dosage:** 10–60 mg/day with food; average diet provides 15–30 mg/day

## Vitamin C

**Claims:** Prevent or reduce the duration of upper respiratory infections, but lacks evidence; antioxidant properties claimed to prevent cardiovascular disease and cancer. Vitamin C used to treat hypertension, exercise-induced asthma, and osteoporosis. Claims also include that it speeds recovery from injury and improves absorption of iron from the GI tract.

**Mechanism:** Water-soluble vitamin, naturally found in citrus fruits and vegetables; plays a role in collagen formation and bone health; antioxidant properties led to speculation about role in treating diseases in which oxidative stress may play a role. Increases glucose and free fatty acid mobilization through epinephrine synthesis; enhances iron absorption

**Efficacy:** Promotes iron absorption from the GI tract and may have a role in preventing cancer when directly obtained from fruit and vegetable sources; same benefits not shown with supplement use; some evidence of modest decrease in duration of common cold symptoms, particularly in those exposed to vigorous activity in cold conditions; lowers systolic blood pressure when combined with conventional antihypertensives; and decreases exacerbations of exercise-induced asthma; is effective in the prevention of osteoporosis and may slow progression of OA (only when directly obtained from fruit and vegetable sources); decreases risk of complex regional pain syndrome after wrist fracture; does not appear to prevent common cold or aid treatment of cancer

**Side effects:** Uncommon and dose-related; hyperoxaluria, hematuria, crystalluria, hyperuricosuria, and predisposition to urinary stone formation may be related to vitamin C intake of >1 g/day. Other side effects include intestinal obstruction, other GI distress, headache, insomnia, fatigue, and flushing.

**Dosage:** RDA ranges from 65 to 120 mg/day. Tobacco use increases daily requirements. As a dietary supplement, typically taken at doses of 75–90 mg/day, with pregnant women and the elderly requiring up to 120 mg/day. Some common foods (e.g., guava, kiwifruit, broccoli, loganberry, and brussel sprouts) can supply 80–100 mg of vitamin C/100 g of food. Studies have shown that up to 25% of athletes consume <70% of the recommended daily dose. Doses of 1–3 g/day have been recommended for prevention and treatment of common cold.

## Vitamin D

**Claims:** Important in bone metabolism: without sufficient vitamin D, bones can become brittle or misshapen; prevents rickets in children and osteomalacia in adults while protecting older adults from osteoporosis; other roles include modulation of cell growth, neuromuscular and immune function, and reduction of inflammation

**Mechanism:** Fat-soluble vitamin naturally found in very few foods; flesh of fatty fish (salmon, tuna, and mackerel) and fish liver oils are best sources. Endogenously produced when ultraviolet rays from sunlight triggers vitamin D synthesis through the skin but is impaired by sunscreen; must undergo two hydroxylations in the body for activation through the liver, where it is converted to 25-hydroxyvitamin D (calcidiol) and then in the kidney, where it forms 1,25-dihydroxyvitamin D (calcitriol); promotes calcium absorption and maintains calcium and phosphate concentrations to facilitate normal bone mineralization; necessary for bone growth and remodeling by osteoblasts and osteoclasts

**Efficacy:** Most trials studying its effects on bone health include calcium; hence, isolating the effects of vitamin D is difficult. Postmenopausal women and older men show a small increase in bone mineral density throughout the skeleton with supplementation of vitamin D and calcium; however, no risk reduction of fractures or reduction of falls in the elderly. Laboratory and animal evidence suggests vitamin D deficiency may contribute to the risk of cancer in the colon, prostate, and breast, but further research necessary. Studies do not support supplementation, with or without calcium, to be effective in reducing the risk of cancer.

**Side effects:** Toxicity threshold for vitamin D dosage is 10,000–40,000 IU/day and for serum 25-hydroxyvitamin D levels is 500–600 nmol/L. Toxic doses may cause nonspecific symptoms

(e.g., anorexia, weight loss, polyuria, and heart arrhythmias) and more serious side effects from hypercalcemia that lead to vascular and tissue calcification and subsequent damage to the heart, blood vessels, and kidneys.

**Dosage:** RDA for vitamin D is 400–800 IU/day. Tolerable upper limits of intake levels for vitamin D are 1,000–4,000 IU/day.

### Vitamin E

**Claims:** Antioxidant, and similar claims as for vitamin C regarding prevention of cardiovascular disease and cancer. May be useful in treating diabetes and associated complications as well as Alzheimer's disease and other dementias; numerous other claims include treatment of asthma and various neuromuscular disorders, prevention of allergies, negative side effects of air pollution, signs of aging, and cataracts. Claims to reduce delayed onset of muscle soreness in extreme exercise

**Mechanism:** Fat-soluble vitamin naturally found in grains, meats, poultry, eggs, fruits, and vegetables; widely distributed in different foods, so true vitamin E deficiency is rare. Deficiency may occur with fat malabsorption syndromes or eating disorders. Primary function is as an antioxidant and protecting cell membranes from oxidation and destruction; proposed benefits are mostly related to this function. May have anti-inflammatory and immune enhancement effects and may inhibit platelet aggregation

**Efficacy:** Role in the prevention of cardiovascular disease controversial; controlled and blinded multicenter trials have shown no benefit, whereas other studies have shown benefits from vitamin E supplementation. May be efficacious for several different problems, including treatment of dementia, liver disease, and normalizing retinal blood flow in diabetics; no evidence that supplementation improves health outcomes in healthy children or adults or otherwise

**Side effects:** Dose-dependent but generally safe, even at doses exceeding RDA; high-dose or long-term supplementation may cause vitamin E toxicity. Data suggest a possible increase in mortality and in the incidence of heart failure with long-term use of vitamin E (≥400 IU), particularly in patients with chronic diseases. May increase the risk of bleeding; reported side effects include GI distress, fatigue, weakness, rash, gonadal dysfunction, and creatinuria.

**Dosage:** Recommendations are confusing: expressed in mg, but most products labeled in IUs. Conversion to IU of natural vitamin E, multiply # mg by 0.67; conversion to IU of synthetic vitamin E, multiply # mg by 0.45; RDA for older children and adults is 15 mg/day; with lactation, 19 mg/day. Doses vary for other claimed uses.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SPORTS PHARMACOLOGY OF PAIN AND INFLAMMATION CONTROL IN ATHLETES

7

Sourav K. Poddar • Heather L. Grothe

## OVERVIEW

The pharmacology of pain management in the athletic arena can be a critical component in returning an athlete to play. Several options exist, and choosing an appropriate intervention should involve careful consideration of treatment goals and potential adverse reactions.

## NONSTEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDs)

NSAIDs are frequently prescribed to athletes by sports medicine providers as a way to limit inflammation and pain and to subsequently facilitate return to play. Research regarding effects of these widely used medications has brought into question the role of these drugs in treating athletic injuries.

### Prevalence

- NSAIDs are one of the most commonly used medications. In the United States (US) population, >29 million adults are estimated to be regular users of NSAIDs.
- In addition, an estimated 16% of the US population aged >50 years use an NSAID at least 3 times a week for at least 3 consecutive months.

### Mechanism of Action

- NSAIDs work by primarily inhibiting the cyclooxygenase (COX) pathway and, to a lesser extent, the lipoxygenase pathway, thereby blocking the conversion of arachidonic acid to prostacyclins, prostaglandins, and thromboxanes.
- Through this mechanism, NSAIDs exert antipyretic, analgesic, and anti-inflammatory actions.
- Moreover, blocking the production of certain prostaglandins causes NSAIDs to exert an inhibitory effect on neutrophil aggregation and lysosomal enzyme release.
- NSAIDs are also thought to have nonprostaglandin effects on limiting leukotriene synthesis via inhibition of membrane-related processes.
- The two main forms of COX, COX-1 and COX-2, are thought to have different functions (Fig. 7.1).
  - COX-1 is presumably a constitutive enzyme involved in the synthesis of prostaglandins that regulate physiologic processes; it plays an important role in the function of the gastric mucosa, kidneys, vascular endothelium, and platelets.
  - COX-2, on the other hand, is primarily considered as an inducible isoform (although data show that it may have a role in certain constitutive processes): involved in the synthesis of prostaglandins that mediate inflammation, pain, and fever in response to tissue injury.
  - The concept behind the development of COX-2-specific inhibitors was to preserve the physiologic function of COX-1 while limiting the effects of COX-2 on tissue injury.
  - Nonselective NSAIDs block both isoforms and subsequently have a significant side-effect profile.

### Alternate Mode of Delivery

#### Topical

- Pharmaceutical compounding of NSAIDs is readily available in the US. These formulations are prescribed in the form of a gel, foam, spray, cream, or patch.
- Purported benefits of topical delivery of NSAIDs lie in the decrease in adverse systemic effects on the gastric mucosa, kidneys, and vascular endothelium.
- Serum concentrations of topical NSAIDs appear to be considerably lower than the levels measured after oral intake or intramuscular administration; this may result in fewer drug–drug interactions.
- Older adults report a higher incidence of adverse effects than younger individuals.
- The most common side effect is local irritation at the application site, although this is uncommon.
- Several trials have demonstrated efficacy of topical NSAIDs with improvement in subjective pain symptoms. A Cochrane review showed that for all topical NSAIDs compared with placebo, the number needed to treat to benefit (NNT) for clinical success, equivalent to 50% pain relief, was 4.5 (3.9–5.3) for a treatment period of 6–14 days.
- Topical NSAID compounding appears to provide an alternative mode of use in the setting of acute superficial soft tissue injury with limited side effects.

#### Injectable

- There are few NSAIDs available for use as intramuscular (IM) injections, and their use is controversial.
- Intramuscular ketorolac (Toradol) is widely used before athletic competitions in college and professional sports; however, there are limited data, and actual prevalence of use remains unknown.
- Ketorolac reaches its peak plasma concentration within 45 minutes when administered via an IM route compared to 20 minutes via the oral route.
- In general, it is recommended that ketorolac should not be used prophylactically as a means of reducing anticipated pain during practices or games.
- It should be given in the lowest effective therapeutic dose and should not be used in any form for >5 days as the side-effect profile is nearly equivalent to oral administration.
- General caution and discussion of risks and benefits with the athlete should be considered before administration of an IM NSAID such as ketorolac.

#### Types

Within the NSAIDs family, there are several subclasses that may provide subtle differences in metabolism and therapeutic effects (Box 7.1).

### Adverse Reactions

- Considering the significant side-effect profile of NSAIDs, physicians should be cautious when prescribing them.

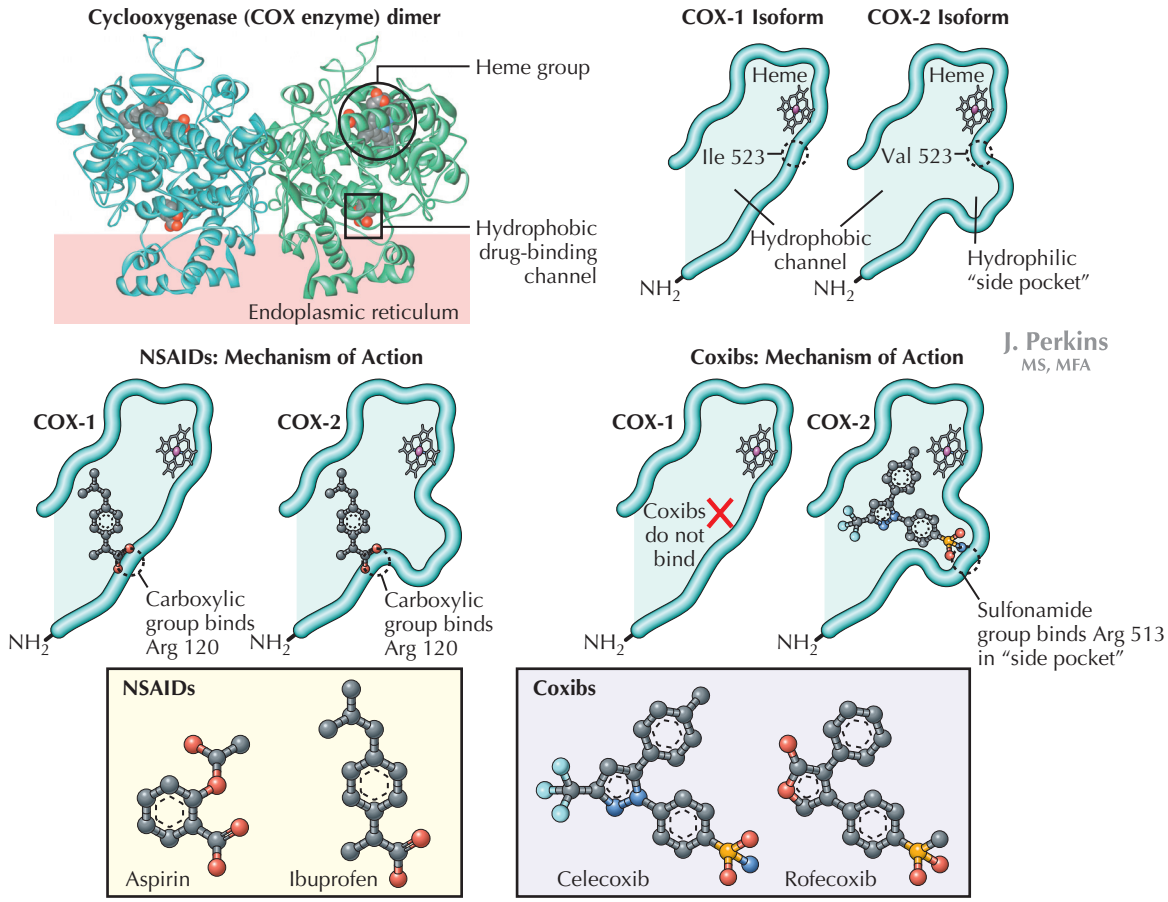



**Figure 7.1.** Nonopioids: NSAIDs, selective cyclooxygenase-2 inhibitors, and acetaminophen.

- Long-term use of NSAIDs may increase the risk of stroke, myocardial infarction, and thrombotic events.
  - Previously, COX-2 inhibitors have been linked to increased risk of myocardial infarction, but recent studies have suggested an increased risk of myocardial infarction with nonselective NSAIDs as well.
  - Shown to have a negative impact on blood pressure, increasing both systolic and diastolic pressures.
- The most common side effect associated with NSAID use is gastrointestinal (GI) bleeding:
  - Development of COX-2 inhibitors was supposed to ameliorate this problem, but data regarding an overall decrease in GI side effects are conflicting.
  - Use of prophylactic medications such as $H_2$-receptor antagonists and proton-pump inhibitors is common, but outcomes addressing risk reduction of stomach ulceration and GI bleeding are variable.
  - *H. pylori* eradication is recommended if long-term use of NSAIDs is being considered.
  - Increased risk of GI bleed, ulceration, and stomach or intestinal perforation may occur at any time without warning symptoms with both acute and chronic use.
  - Dosing for shorter intervals and consumption with food may reduce side effects.
- Effects of NSAID use on various musculoskeletal variables have also been studied:
  - Animal studies have demonstrated that NSAID use slows fracture healing and may contribute to malunion and/or nonunion.
  - Studies on NSAID use for ligament sprains and muscle strains have reported early improvement in symptoms and subsequent return to activity, but effects of long-term use on soft tissue healing remain unknown.
- NSAIDs block constitutive prostaglandins necessary for optimal kidney function.
  - Decrease sodium excretion and increase free water retention, particularly with long-term use
  - Avoidance with endurance events safest owing to potential renal morbidity
  - May also cause interstitial nephritis, regardless of duration of use

## Pharmacokinetics

- Rapidly and completely absorbed in the GI tract
- Although most NSAIDs are metabolized via the cytochrome P450 system through the enterohepatic circulation, excretion occurs through the kidneys.
- Half-lives of different NSAIDs vary considerably, ranging from a few to several hours.

## Therapeutic Recommendations

- Despite their significant side-effect profiles, judicious short-term use of NSAIDs in the athletic arena is justifiable. In settings of sprains, acute muscle strains, eccentric load injury to muscle, and acute tenosynovitis or tendonitis, a 3–5 day course may help decrease pain and facilitate quicker return to activity.
- Caution should be exercised regarding use of NSAIDs with acute fractures or stress fractures of areas at a high risk for of nonunion.

**BOX 7.1 SUBCLASSES OF NSAIDs**

**Salicylic Acid Derivatives**
- ASA
- Salicyl salicylate
- Diflunisal

**Heteroaryl Acetic Acids**
- Diclofenac
- Ketorolac
- Tolmetin

**Fenamates**
- Mefenamic acid
- Meclofenamic acid

**Alkanones**
- Nabumetone

**Indole Acetic Acids**
- Indomethacin
- Sulindac
- Etodolac

**Arylpropionic Acids**
- Ibuprofen
- Naproxen
- Ketoprofen

**Enolic Acids**
- Piroxicam
- Phenylbutazone

**COX-2 Inhibitors**
- Celecoxib

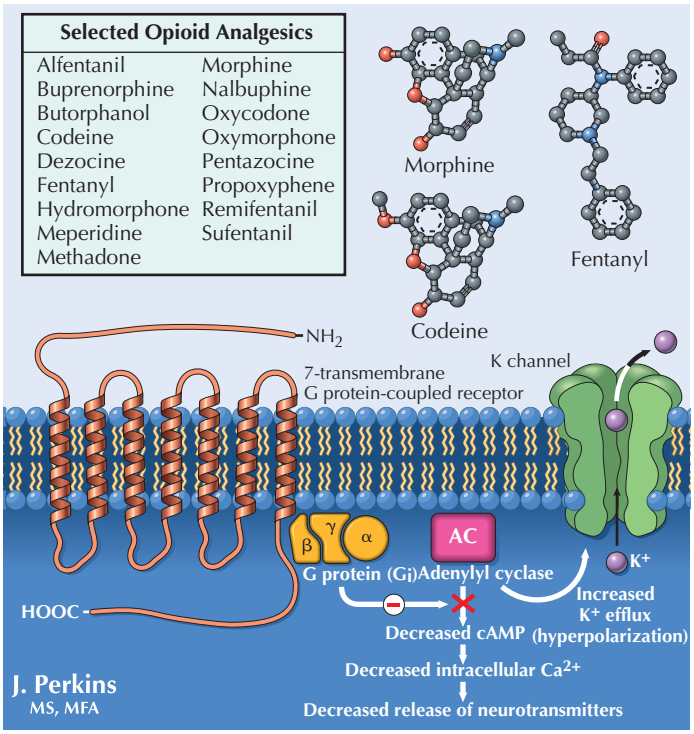



**Figure 7.2.** Opioids: receptor-transduction mechanisms.

## OTHER ANALGESICS

### Opioids

- Narcotic analgesics should be judiciously used. Significant side effects, such as sedation and subsequent deficits in coordination, cognition, and reaction time, make these poor choices for pain control before or during competition, and use during a sporting event cannot be justified (Fig. 7.2).
- Most opioids are banned by governing bodies of major sports unless prescribed for use by a physician with appropriate cause.
- Certain situations, such as a broken bone or severe acute trauma, may warrant the use of opioids; however, care must be taken to avoid extended use because physical dependency may readily develop.

### Acetaminophen

- One of the most common over-the-counter medications recommended by physicians, acetaminophen is considered as a first-line pain reliever according to numerous current guidelines.
- Exact analgesic mechanism is not known but has inhibitory effects on the COX-1 and -2 pathways of the body.
- Exerts antipyretic effects via direct action on the hypothalamic heat-regulating center
- Metabolism occurs via the cytochrome P450 system in the liver, and potentially toxic metabolites are then excreted in the urine.
- The maximal acetaminophen dosages should be reduced in athletes with renal impairment considering the potential accumulation of these toxic metabolites. Similarly, liver enzymes must be monitored with prolonged use in athletes with impaired liver function.
- The maximum recommended dose in healthy adolescents and adults is 3 g/day.

### Tramadol

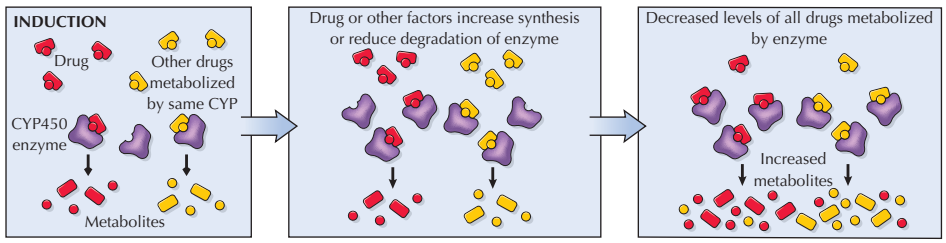
- Central opioid agonist: revised to a class IV controlled substance in 2014
- Produces its analgesic effect by binding to μ-opioid receptors and also by weakly inhibiting norepinephrine and serotonin reuptake
- Extensively metabolized in the liver by the cytochrome P450 system, has the potential for several drug interactions, and has an active metabolite that increases the half-life of the drug in the body (Fig. 7.3)
- Short-term use in athletes for moderate to severe pain associated with injury
- Risk of dependency and abuse if prescribed for long-term use

## CORTICOSTEROIDS

- Potent anti-inflammatory agents that produce multiple glucocorticoid and mineralocorticoid effects
- Use in athletes should be judicious owing to numerous systemic effects and drug interactions
- Available in oral formulations or administered as intra-articular injections for inflammation
- Consider tapering oral doses if used for >5–7 days
- Appropriate activity modification recommended with use of corticosteroids during the athletic season

### Mechanism of Action

- Corticosteroids are lipid-soluble molecules that work by binding to intranuclear receptors. They inhibit the chemotaxis of




| CYP | Inducers | Inhibitors |
|---|---|---|
| 1A2 | Smoking, charbroiled foods, cruciferous vegetables, insulin, modafinil, nafcillin, omeprazole, phenobarbital, primidone, rifampin | Amiodarone, anastrozole, cimetidine, ciprofloxacin, diltiazem, enoxacin, erythromycin, fluoroquinolones, fluvoxamine, grapefruit (juice), mexiletine, norfloxacin, ritonavir, tacrine, ticlopidine |
| 2A6 | Dexamethasone, phenobarbital | Methoxsalen, ritonavir, tranylcypromine |
| 2B6 | Cyclophosphamide, dexamethasone, phenobarbital, phenytoin, primidone, rifampin | Efavirenz, nelfinavir, orphenadrine, ritonavir, thiotepa, ticlopidine |
| 2C8/9 | Dexamethasone, primidone, rifampin, secobarbital | Anastrozole, amiodarone, cimetidine, diclofenac, disulfiram, fluconazole, fluvoxamine, flurbiprofen, fluvastatin, isoniazid, ketoprofen, lovastatin, metronidazole, omeprazole, paroxetine, phenylbutazone, ritonavir, sertraline, sulfinpyrazone, sulfonamides, sulfamethoxazole, trimethoprim, troglitazone, zafirlukast |
| 2C19 | Barbituates, rifampin | Cimetidine, ketonconazole, modafinil, omeprazole, oxcarbazepine, ticlopidine |
| 2D6 | Dexamethasone, quinidine, rifampin | Amiodarone, bupropion, celecoxib, chlorpromazine, chlorpheniramine, cimetidine, clomipramine, cocaine, doxorubicin, fluoxetine, fluphenazine, fluvoxamine, haloperidol, lomustine, metoclopramide, methadone, norfluoxetine, paroxetine, perphenazine, propafenone, quinidine, ranitidine, ritonavir, sertindole, sertraline, terbinafine, thioridazine, venlafaxine, vinblastine, vinorelbine |
| 2E1 | Acetone, ethanol, isoniazid | Disulfiram, ritonavir |
| 3A4 | Barbituates, carbamazepine, dexamethasone, efavirenz, macrolides, glucocorticoids, modafinil, nevirapine, oxcarbazepine, phenobarbital, phenylbutazone, pioglitazone, phenytoin, primidone, rifabutin, rifampin, St. John's wort, sulfinpyrazone, troglitazone | Amiodarone, anastrozole, chloramphenicol, cimetidine, ciprofloxacin, clarithromycin, clotrimazole, danazol, delavirdine, diltiazem, erythromycin, fluconazole, fluoxetine, fluvoxamine, grapefruit juice, indinavir, itraconazole, ketoconazole, metronidazole, mibefradil, miconazole, nefazodone, nelfinavir, nevirapine, norfloxacin, norfluoxetine, omeprazole, paroxetine, propoxyphene, quinidine, ranitidine, ritonavir, saquinavir, sertindole, troglitazone, troleandomycin, verapamil, zafirlukast, zileuton |

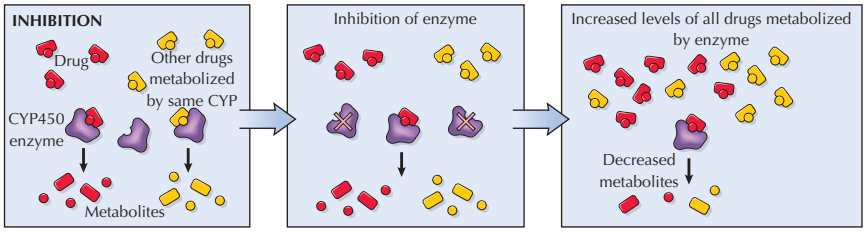



**Figure 7.3.** Metabolic enzyme induction and inhibition.

inflammatory cells and decrease lysosomal enzyme release as well as production of inflammatory mediators.
- Intra-articularly, corticosteroids decrease migration of neutrophils into arthritic joints that are inflamed; in addition, they reduce prostaglandin synthesis and decrease interleukin-1 secretion and subsequent leukocyte aggregation by the synovium.
- Studies have demonstrated the indirect increase of synovial fluid viscosity in a joint injected with corticosteroid preparation via an increased concentration of hyaluronic acid in the joint.

## Types

The original corticosteroid used for intra-articular injection was hydrocortisone (which explains the familiarity of several patients with the term "cortisone" injections). Subsequent research helped develop formulations with longer durations of effect, primarily mediated by solubility (Table 7.1).

## Uses

- Uses of corticosteroids in athletes include as intra-articular injections for chronic and acute inflammation; pain relief in small joints is greater than that in larger joints.
- Lack of inflammatory markers in tendinopathy make the use of corticosteroids of controversial value in treatment. However, tendon sheath injections have proven beneficial in the treatment of tenosynovitis of the wrist and proximal biceps. Moreover, some studies have demonstrated its role in the treatment of inflamed bursae.
- Single injection for adhesive capsulitis of the shoulder joint provides faster pain relief and earlier improvement of shoulder function and motion when compared with NSAIDs.

## Adverse Reactions

- Potential side effects of corticosteroids can be significant. Local effects include increased risk of tendon and ligament rupture secondary to inhibition of collagen synthesis. Injection into sites such as the Achilles or patellar tendons is contraindicated.
- Other potential adverse effects include skin atrophy at the injection site, fat atrophy, flare reaction, infection of the joint injected or injection site, and synovial calcification.
- Systemic effects of both oral and intra-articular administration of corticosteroids include inhibition of the hypothalamic-pituitary-adrenal axis at higher doses. Dose tapering is recommended to minimize the risk of adrenal insufficiency with prolonged or repetitive use.
- In individuals with diabetes, corticosteroids may result in short-term worsening of glycemic control (up to few weeks).
- Low-dose intra-articular injection of corticosteroid may have beneficial anti-inflammatory effects on distant joints.

# VISCOSUPPLEMENTATION

## Background

- The use of intra-articular viscosupplementation or hyaluronic acid (HA) injections in pain management in lower-extremity osteoarthritis (specifically knee and more recently ankle) has been established.

- HA is diminished in arthritic knees, which reduces the viscoelastic property of the synovial fluid; this increases stress and shear forces of articular surface and may lead to further damage.

## Mechanism of Action

- Exact mechanism of action of intra-articular viscosupplementation is unknown.
- At low shear forces in the joint, HA exhibits high viscosity and low elastic properties. Conversely, at high shear forces, the properties of HA are the opposite, demonstrating low viscosity and high elasticity.
- In addition, HA has both anti-inflammatory and analgesic properties:
  - Inhibits macrophage phagocytosis and neutrophil adherence
  - Reduces release of arachidonic acid (a precursor of inflammatory mediators) from fibroblasts in the synovium
  - Analgesic effects include possible direct inhibition of pain receptors.
  - Moreover, HA purportedly and indirectly binds substance P, decreasing pain signals.

**TABLE 7.1 DURATION OF EFFECT OF COMMONLY USED CORTICOSTEROIDS**

| Generic Name | Average Duration of Effect (Days) |
|---|---|
| Methylprednisolone sodium succinate | 4 |
| Dexamethasone sodium phosphate | 6 |
| Triamcinolone diacetate | 7 |
| Methylprednisolone acetate | 8 |
| Dexamethasone acetate | 8 |
| Hydrocortisone acetate | 8 |
| Betamethasone acetate | 9 |
| Prednisolone tebutate | 10-14 |
| Triamcinolone acetonide | 14 |
| Triamcinolone hexacetonide | 21 |

Adapted from Snibbe JC, Gambardella RA. *Clin Sports Med.* 2005;24:1-3.

## Properties

- Several properties contribute to its effectiveness and tolerability.
- Lack of immunogenicity limits the chances of local reaction after intra-articular injection.
- HA exhibits passive diffusion into the synovial fluid and a prolonged half-life within the synovium.

## Types

- Several different brands of viscosupplementation available in the US (Table 7.2); the number of injection series range from 1 to 5
- Each has different molecular weight and preparation of purified sodium hyaluronate.
- Molecular weight ranges from 500 to 6000 kDa.
- May be produced through bacterial processes of biologic fermentation or extraction of avian-derived molecules
- All have relative contraindications for use in patients with avian or avian-derived product allergies, except Euflexxa, which is a bioengineered, fermentation-derived product.

## Adverse Reactions

- 2%–4% per injection
- Local reactions such as warmth, swelling, and pain can last for 1–2 days.
- Granulomatous inflammation arising within 48 hours after injection has been reported with hylan G-F 20, which usually resolves within 1–2 weeks.

## Clinical Results

- Clinical efficacy well studied
- Most studies focus on knee arthritis, and despite numerous clinical trials, the efficacy of HA remains debatable with markedly diametric interpretations of the data.
- Improvement in pain and functionality versus placebo demonstrated in several studies
- Salutary effects may be similar to or better than intra-articular corticosteroid injections in the intermediate term.
- Small-scale studies have demonstrated benefits in the treatment of ankle arthritis.

**TABLE 7.2 BRANDS OF HYALURONIC ACID (HA) VISCOSUPPLEMENTS**

| Brand Name | Molecular Weight (kDA) | Comments |
|---|---|---|
| Hyalgan | 500–730 | Studies with 5 weekly injections show decrease in visual analog scale (VAS) pain scores at 26 weeks<br>Additional studies with 3 weekly injections show benefit over placebo at 60 days |
| Synvisc | 6000 | Cross-linked mixture of gel and fluid formulation of HA<br>Studies with 3 weekly injections show benefit versus placebo out to 26 weeks |
| Supartz | 620–1170 | Improvement in Lequesne index and VAS pain scale with 5 weekly injections followed up to 13 weeks |
| Euflexxa | 2400–3600 | Bioengineered HA that is not derived from purified rooster comb<br>Series of 3 weekly injections showed improvement of VAS WOMAC index similar to that of Synvisc at 12 weeks |
| Orthovisc | 1000–2900 | Improvement in WOMAC pain scores observed up to 22 weeks after 3 weekly injections |
| Synvisc One | 6000 | Combines the 3 Synvisc injections into a single 6-mL injection<br>Studies with a single injection show benefit versus placebo followed up to 26 weeks |
| Monovisc | 1000–2900 | Combines Orthovisc into a single 4-mL injection. Improvement in WOMAC pain scores observed up to 22 weeks after 3 weekly injections |
| Gel-One | Unknown | Single 3-mL injection of cross-linked hyaluronate<br>Improvement in WOMAC pain and function scores observed up to 13 weeks |

- Larger-scale studies involving ankle and other joints, including shoulder, are currently under way.
- Currently, no FDA approval for use in hip or shoulder
- Current literature focuses on studies in older adults; however, future studies should investigate its potential chondroprotective effects in younger athletes with early arthritis.
- In vitro studies suggest that early use of HA viscosupplementation in low-grade osteoarthritis may slow disease progression.

## BIOLOGICS

Advent of autologous growth factor therapies holds the potential to use the body's own healing ability to accelerate and improve tissue repair.

### Autologous Blood and Platelet-Rich Plasma Therapy

The concept of enhancing healing and accelerating an athletes "return to sport" ability remains an elusive goal in sports medicine. Autologous blood and platelet-rich plasma (PRP) are increasingly utilized owing to the presence of growth factors and the potential to induce additional release of local factors to enhance healing.

### Background

- Growth factors mediate the repair process, and higher concentrations of growth factors in injured tissues are theorized to enhance or accelerate the healing process.
- Autologous blood is utilized by injecting a small amount of patients' own whole blood back into injured tissues.
- PRP is prepared by taking patients' own whole blood and centrifuging it to extract plasma, which contains concentrated platelets and growth factors.

### Mechanism of Action

- Blood contains several nutrients and factors that are thought to promote healing.
- Platelets are one of the first cells to arrive on site after injury; they play an instrumental role in the normal healing response via local secretion of growth factors and recruitment of reparative cells.
- Platelet alpha-granules are activated to release bioactive molecules, which include adhesive proteins, clotting factors, membrane proteins, and growth factors.
- Growth factors, including TGF-β, VEGF, PDGF, FGF, HGF, IGF, and EGF, are released.
- Literature supports the concept that PRP regulates the local production of growth factors and alters the environmental milieu.

### Uses

- Autologous blood and PRP have been used in a variety of musculoskeletal injuries, which include muscle and ligament injuries, tendinosis, and enthesopathy.
- PRP is also currently being used and studied for cartilage defects and osteoarthritis and is also being used as an adjunct in orthopedic surgeries.

### Considerations and Outcomes

- PRP can be prepared in either leukocyte-rich or leukocyte-poor forms.
- Preliminary data show trends toward better clinical outcomes with leukocyte-poor PRP preparations used in treatment of osteoarthritis.
- Leukocyte-rich PRP shows promise in treatment of chronic tendinopathy.
- A wide variety of platelet concentrations used and optimum platelet concentration are not yet known.
- In-vitro lidocaine toxic to platelets and tenocytes
- Growth factor function is pH dependent
- Autologous blood shown to be an effective treatment for patellar tendinosis and elbow epicondylosis in a small number of case series, where it improved pain and function
- Autologous blood and PRP superior to corticosteroids in treatment of lateral epicondylosis
- Substantial evidence to support the use of PRP in chronic tendinopathy unresponsive to other conservative treatments
- Certain small-scale studies have shown that PRP may aid the natural repair process of muscle and decrease the healing time.
- Chondrocytes stimulated in vitro with PRP increase synthesis of proteoglycans and collagen.
- PRP and autologous blood may be used to treat musculoskeletal injuries; however, further research and standardization of protocols are needed.

## SUMMARY

- Management of pain and inflammation in athletes can be a challenging task. Side effects (both adverse and beneficial), drug interactions, and possible contraindications should be carefully considered.
- Although NSAIDs have long been a favorite in the armamentarium of the sports medicine practitioner, limiting its use to appropriate situations and shorter duration of therapy are prudent.
- Biologics and growth factors are an emerging treatment option.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SPORTS PHARMACOLOGY OF CHRONIC DISEASE

# 8

Amy P. Powell • Kyle Goerl

## INTRODUCTION

Adults are encouraged to engage in 150 minutes of moderate-intensity exercise weekly to maintain overall health and fitness. As the US population ages, many people are actively taking or have previously taken medications that may affect exercise performance. Managing athletes and patients on medications for chronic illnesses is an important skill for sports medicine physicians to master.

## LIPID-LOWERING AGENTS

### Statins

- Statins are some of the most commonly prescribed drugs worldwide, with atorvastatin alone reaching $12 billion in sales in 2015.
- Recent guidelines for statin use recommend pharmacologic management in most adults.
- Fifty-six million Americans aged 40–75 years are now considered candidates for statin use, and 87% of men aged over 60 years meet the eligibility criteria for statin treatment based on application of these guidelines.

#### Mechanism of Action

- Inhibitor of HMG CoA reductase, an enzyme important for cholesterol synthesis

#### Musculoskeletal Adverse Effects

- Myalgias
  - Muscle-related complaints are reported in 1.5% of patients in randomized controlled trials on statin and 10% of patients in observational studies.
  - Statin-induced myopathy exists on a spectrum. The mildest presentation is myalgia without elevation of creatine kinase. True myositis (defined as elevation of creatine kinase >10 times the upper limit of normal laboratory values) leading to rhabdomyolysis; death is the most severe presentation but very rare.
  - Symptoms typically begin within 6 months of treatment initiation and resolve 2–3 months after statin discontinuation.
  - Risk factors for statin-induced myopathy include higher medication dose, female gender, older age, low BMI, untreated hypothyroidism, and lipophilic statin use (lovastatin, simvastatin, and atorvastatin are the most lipophilic). Concomitant treatment with other medications metabolized through or genetic alterations of the cytochrome p450 system may also increase the risk of myopathy (e.g., fibrates, calcium-channel blockers, and azole antifungal agents).
  - Proximal muscles are preferentially affected.
  - Symptoms include muscle pain, stiffness, muscle fatigue, tendon pain, and nocturnal cramping. Symptoms tend to be worse with exertion and after exercise.

#### Impact on Exercise Performance

- Conflicting reports exist regarding exercise performance while on statins.
- Certain investigators have found decreased muscle strength and reduced aerobic exercise performance in patients treated with statins, but available literature in this area has mixed results.

### New Lipid-Lowering Medications

- Two pro-protein convertase subtilisin/kexin type 9 (PCSK9) inhibitors were approved for use in 2015: evolocumab and alirocumab.
- PCSK9 is an enzyme that binds to the LDL receptor and prevents the LDL receptor from appropriately removing LDL cholesterol.
- Inhibition of PCSK9 has been shown to be a powerful method of reducing LDL cholesterol, with initial trials showing 60% reduction in LDL levels after 12 months of treatment.
- PCSK9 inhibitors are administered by subcutaneous injection every 2–4 weeks.
- Annual treatment cost is $7000–$12000, which may limit its regular use.
- Neurocognitive side effects (e.g., confusion and inattention) have been reported in several clinical trials. Myalgias, while reported, were rare.
- PCSK9 inhibitors are likely to be considered for athletes who cannot tolerate statins drug due to their adverse effects.

## ANTIHYPERTENSIVE DRUGS

### Background

- Hypertension (HTN) is the most common cardiovascular condition affecting adults in the US. It is common in athletes of all ages and is a leading risk factor for cardiovascular morbidity and chronic kidney disease.
- HTN is associated with decreased exercise capacity in elite athletes.
- Systolic blood pressures continue to rise throughout life due to arterial stiffening, whereas diastolic pressures plateau in the sixth decade of life and subsequently decline.
- Approximately 55% of men and 65% of women will have HTN by the age of 60.

### Mechanism of Action and Side Effects

Multiple medication classes can be utilized to treat HTN. More typical medication classes are reviewed here:

- **Angiotensin-Converting Enzyme (ACE) inhibitors** (lisinopril, enalapril, and captopril) primarily act by suppressing the renin-angiotensin-aldosterone system via inhibition of conversion of angiotensin I to angiotensin II; this blocks the breakdown of bradykinin, which can lead to side effects such as cough and angioedema. Other side effects include hyperkalemia and elevated creatinine associated with renal artery stenosis.
- **Angiotensin Receptor Blockers** (ARBs: losartan, valsartan, and olmesartan) block binding of angiotensin II to angiotensin type I receptors, thereby effectively reducing the ability of angiotensin II to cause vasoconstriction, sodium retention, and aldosterone release. Side effects are similar to ACE inhibitors, but cough is rare.
- **Beta-Blockers:** Beta-1 receptor blockers (metoprolol and propranolol) inhibit sympathetic stimulation of the heart, thereby reducing heart rate. Others (labetalol and carvedilol) work by blocking alpha-1 receptor activity to cause peripheral vasodilation, and others have intrinsic sympathomimetic activity (acebutolol), which reduces systemic vascular resistance while maintaining heart rate and cardiac output.

- **Calcium-Channel Blockers (CCBs)**: Dihydropyridine CCBs (amlodipine and nifedipine) bind calcium channels in vascular smooth muscles leading to vasodilation. Nondihydropyridine CCBs (verapamil and diltiazem) bind to calcium channels in the sinoatrial and atrioventricular nodes leading to negative inotropic effects. Side effects may include lightheadedness, hypotension, and lower extremity swelling.
- **Diuretics**: Thiazide diuretics (hydrochlorothiazide and chlorthalidone) inhibit reabsorption of sodium and chloride, primarily in the distal tubules, which can lead to hyponatremia. Potassium-sparing diuretics (triamterene and spironolactone) inhibit sodium reabsorption in the distal tubules but are rarely used in monotherapy. Loop diuretics (furosemide, torsemide, and bumetanide) inhibit the reabsorption of sodium and chloride at the ascending loop of Henle and may commonly lead to hypokalemia.

### Treatment of Hypertension

- The eighth Joint National Committee (JNC8) recommends initiation of treatment for blood pressures >150/90 mmHg in adults aged ≥60 years and at 140/90 mmHg in anyone aged <60 years and patients with chronic kidney disease (CKD) and diabetes.
- Athletes should begin treatment with nonpharmacologic options including lifestyle modifications, such as decreased sodium intake, decreased fat intake, weight loss, decreased alcohol consumption, stimulant avoidance, limited NSAID use, smoking cessation, relaxation techniques, and aerobic exercise.
- Choosing HTN medications in athletes can be challenging: best choice of drug is one that reliably lowers blood pressure, has limited negative effects on exercise hemodynamics, and is legal for the athlete to use.
- Other existing medical comorbidities, drug–drug interactions, and response to previous medications must also be considered.
- ACE inhibitors, ARBs, and CCBs are typical first-line agents in athletes because they are well tolerated and have no major effects on energy metabolism or cardiovascular adaptation to exercise.
- Thiazide diuretics are also considered first-line agents but have certain negative effects that makes them less desirable choices in athletes.
- In patients with CKD, ACE inhibitors and ARBs should be the initial or first add-on choice of therapy.
- ACE inhibitors and ARBs should be cautiously used in women of reproductive age as they are contraindicated during pregnancy.
- African Americans typically respond better to CCBs and thiazide diuretics.

### Considerations in Athletes

- The prevalence of HTN in athletes is unknown but is thought to be half of that in the general population. However, in preparticipation cardiac screening, HTN is the most common abnormal finding. Among adolescent athletes with HTN, the blood pressure continues to remain elevated at the 1-year follow-up in approximately 80% of cases.
- Athletes at a greater risk for HTN include male athletes, strength-trained athletes, African Americans, obese individuals, athletes with diabetes, those with renal disease, and those with a family history of HTN. Collegiate and professional football players have higher rates of HTN compared to nonfootball athletes (19.2% vs. 7% and 13.8% vs. 5.5%, respectively).

#### Doping

- Certain medications are considered doping substances and may be banned by particular sport-governing bodies.
- Diuretics have been used as masking agents to dilute illegal substances; in addition, they have been used for rapid weight-loss, making them popular in sports such as wrestling and boxing.
- Beta-blockers are banned in precision sports such as archery, shooting, diving, and figure skating, where steadiness may give an athlete an unfair advantage.
- As with any medication, consultation with the banned substance list of an athlete's governing body should precede a prescription for an antihypertensive medication to ensure compliance.

#### Negative Effects on Performance

- Diuretics can decrease plasma volume and lead to electrolyte disturbances, which can ultimately result in muscle cramping, dehydration, and cardiac arrhythmias.
- Beta-blockers decrease aerobic output by reducing the athlete's maximal heart rate; moreover, they can reduce the amount of time athletes can spend at submaximal exercise intensity by approximately 20% when used with cardioselective medications (e.g., atenolol, metoprolol, and nebivolol) and by approximately 40% with nonselective medications (e.g., propranolol, nadolol, carvedilol, and sotalol).

## COPD MEDICATIONS

### Background

- Chronic obstructive pulmonary disease (COPD) affects approximately 7% of US adults and 10% of US adults aged ≥65 years; however, it is estimated to be actually higher due to underdiagnosis.
- COPD is the third leading cause of death in the US.
- The Global Initiative for Chronic Obstructive Lung Disease (GOLD) emphasizes both pharmacologic and nonpharmacologic treatments to control COPD.
  - Nonpharmacologic therapies include (i) smoking cessation, (ii) reduction of other risk factors, (iii) vaccination, (iv) oxygen therapy, and (v) pulmonary rehabilitation.
  - Pharmacologic therapy is typically added in a stepwise fashion based on the severity of the disease and may include anticholinergics, beta-2 agonists, and glucocorticoids.
- Goals for pharmacological treatment
  - Bronchodilation is necessary to increase exercise tolerance and decrease exertional dyspnea.
  - Long-term use of daily inhaled anticholinergic medication leads to improved inspiratory capacity, decreased thoracic gas volume, and improved health outcomes.

### Exacerbation Management

- Antibiotics are well supported for the treatment of acute infections and are indicated for increasing dyspnea, sputum production, or sputum purulence.
- Common pathogens in acute exacerbations include *S. pneumoniae*, *H. influenzae*, and *M. catarrhalis*. Antibiotic selection may include amoxicillin, doxycycline, trimethoprim/sulfamethoxazole, cefuroxime, azithromycin, and clarithromycin.
- Macrolides and fluoroquinolones combined with statins increase the risk of QT prolongation; hence, it is recommended to hold statin treatment while taking these antibiotics.

### Implications for Athletes

- Several medications used for COPD management have been defined as banned substances by governing bodies such as the WADA, the USADA, the USOC, and the NCAA. These include beta-2 agonists and glucocorticoids.

## ANTICOAGULANTS

### Common Uses

- Venous thromboembolism (VTE) is a major cause of morbidity and mortality. Treatment for VTE is the same for athletes as nonathletes, typically with anticoagulation and early mobilization.
- Athletes may have multiple risk factors for the development of a VTE, including orthopedic trauma, postinjury immobilization, frequent and prolonged travel, hemoconcentration after exercise, and polycythemia from altitude training or exogenous EPO use.
- Upper extremity deep venous thrombosis (UEDVT) has been well documented in overhead athletes, such as pitchers, and after heavy upper-body workouts.
- Other conditions requiring anticoagulation treatment include atrial fibrillation and pulmonary embolism.

### Treatment

- Anticoagulation is the mainstay for VTE treatment. Use helps maintain collateral patency, reduces further thrombus growth, and reduces the risk of embolization.
- Common medications used to initiate treatment include subcutaneous low-molecular-weight heparin (LMWH), fondaparinux, or intravenous unfractionated heparin (UFH) and simultaneous initiation of a vitamin K antagonist (VKA) such as warfarin.
- Typically, LMWH, fondaparinux, or UFH is used for about 5 days and then discontinued when the patient's international normalized ratio (INR) is in the therapeutic range of 2.0–3.0 (target, 2.5) for 24 hours.
- Monotherapy with factor Xa inhibitors are an alternative for patients who wish to avoid monitoring; those proven effective in monotherapy include rivaroxaban and apixaban.
- Treatment duration for a first-time provoked or unprovoked proximal DVT with a VKA or factor Xa inhibitor should be at least 3 months. If the risk factor(s) for the DVT is transient but persistent at the end of 3 months, then anticoagulation should be continued until the risk factor(s) is resolved.
- For a first-time provoked or unprovoked distal DVT, 3 months of anticoagulation is typically adequate.

### Return-to-Sport

- Athletes participating in contact/collision sports who are on anticoagulation should be restricted from participation until their anticoagulation treatment is complete.
- Athletes participating in noncontact sports may return to sports following an informed discussion and an appropriate return-to-play protocol for their respective sports, unless that sport is suspected to be the cause of the VTE.
- Athletes should be instructed to report any recurrent symptoms consistent with a VTE and signs of internal bleeding, blood loss, or bruising.

## FLUOROQUINOLONE ANTIBIOTICS

### Background

- Fluoroquinolones (FQs) are some of the most commonly prescribed antibiotics, and they are used to treat a broad spectrum of infections affecting the urinary, gastrointestinal, and respiratory tracts as well as bone, joint, and skin infections.
- FQs are used for their excellent gastrointestinal absorption, tissue diffusion, and long half-life.

### Mechanism of Action

- First- and second-generation FQs act by inhibiting DNA gyrase, and the newer-generation FQs inhibit topoisomerase IV, making it effective against anaerobes.

### Medical Adverse Effects

- Most adverse effects from FQs are mild to moderate in severity and self-limiting.
- More serious adverse effects include: C. difficile-associated diarrhea, hemolytic uremic syndrome, hepatotoxicity, hypoglycemia, phototoxicity, and QT prolongation.

#### Musculoskeletal Adverse Effects

- FQs are known to negatively affect tendon, bone, muscle, and cartilage.
- In pediatric patients, FQs are typically avoided except in life-threatening situations owing to irreversible cartilage damage reported in growing animals, as demonstrated by previous studies.
- Most commonly implicated second-generation medications in the descending order are perfloxacin, ofloxacin, norfloxacin, and ciprofloxacin.
- Over 100 cases of FQ-associated tendonitis and tendon rupture have been reported in the literature. Incidence of FQ tendinopathy is estimated as 0.14%–0.4%.
- The US Food and Drug Administration (FDA) issued a warning for FQ causing possible tendonitis in 1996 and then placed a boxed warning on FQs in 2008 for associated tendonitis.
- The risk of tendon rupture appears to be within the first month following FQ use in patients older than 60 years and in patients taking oral corticosteroids.
- Proposed mechanisms by which FQs negatively affect tendons are as follows:
  - FQs are shown to stimulate matrix-degrading protease activity in fibroblasts, thereby inhibiting cell proliferation and matrix ground substance synthesis.
  - FQs chelate cations like magnesium. Cations are used by integrins and transmembrane proteins for structural stability; hence, chelation of cations may weaken the cell and thus the tendon.
  - FQs stimulate oxygen radical production.
- Other anecdotal reports of fatigue, confusion, myalgia, arthralgia, and trigger digits

### Recommendations for Use in Athletes

- Hall et al. proposed guidelines for use of FQs in athletes.
  - Avoid FQs if possible.
  - Inform athlete's care team if FQs are prescribed.
  - Do not prescribe oral or injectable corticosteroids simultaneously.
  - Consider prescribing magnesium with FQs.
  - Alter training regimens while on FQs, followed by a graded return to activity.
  - Stop athletic activity at the onset of symptoms.
  - Monitor the athlete closely for 1 month after completion of treatment.

## CHEMOTHERAPEUTIC AGENTS

### Background

- Some athletes are survivors of various types of adult or childhood cancers.
- Several chemotherapeutic agents have unfavorable side effect profiles that affect the ability to exercise after a malignancy is treated.

### Medications a Sports Medicine Physician Should Know

#### Drugs With Potential to Cause Peripheral Neuropathy

- Includes:
  - Vincristine
  - Vinblastine

  - Cisplatin
  - Paclitaxel
  - Thalidomide
- These drugs are used to treat breast cancer, multiple myeloma, rhabdomyosarcoma, leukemia, lymphomas, ovarian cancer, nonsmall cell lung cancer, bladder cancer, and testicular cancer.
- Signs and symptoms include peripheral numbness, tingling, pain, and loss of reflexes.
- Dose reduction at the time of symptom onset (if possible) may prevent progression.
- Risk factors for development of peripheral neuropathy include higher medication doses and co-treatment with other chemotherapeutic agents known to cause neuropathy.

### Drugs With Potential to Cause Pulmonary Toxicity

- Includes:
  - Bleomycin
  - Carmustine
  - Methotrexate
  - Cyclophosphamide
- These medications are used to treat a variety of cancers, including head and neck squamous cell cancers, leukemia, lymphoma, brain tumor, multiple myeloma, and testicular, lung, breast, and ovarian cancers.
- Pneumonitis progressing to pulmonary fibrosis is a known complication with these drugs. Late-onset symptoms (>6 months after treatment onset) have a poorer prognosis than early-onset symptoms.
- Signs and symptoms of pulmonary toxicity include fatigue, exercise intolerance, dry cough, dyspnea, and wheezing.
- Risk factors for development of pulmonary toxicity related to chemotherapeutic agents include older age (>70 years), combination treatments, radiation therapy to the chest wall, and history of lung disease.

### Drugs With Potential to Cause Cardiac Toxicity

- Includes:
  - Anthracycline drugs, including doxorubicin, daunorubicin, epirubicin, idarubicin, and mitoxantrone
    - Used to treat multiple cancers, including leukemia, lymphoma, bone sarcomas, and breast, ovarian, and prostate cancers
    - The most severe myocardial toxicity associated with anthracycline drugs is potentially fatal congestive heart failure, which can manifest either during therapy or months/years after treatment discontinuation.
    - Prior to initiating anthracycline drugs, guidelines recommend completing a full history and physical examination, obtaining baseline ECG, and evaluating baseline LVEF with echocardiogram, MUGA, or cardiac MRI.
  - Cyclophosphamide
    - Various cardiac toxicities have been reported, including myocarditis, pericardial effusion resulting in tamponade, and potentially fatal congestive heart failure. Cardiotoxicity appears to be dose dependent.
- Risk factors for chemotherapy-related cardiotoxicity include older age, combination therapy, history of cardiac disease, and history of radiation therapy to the chest wall.
- Presenting symptoms include exercise intolerance, palpitations, tachycardia, dyspnea, fatigue, cough, and edema.

## HIV MEDICATIONS

Significant advances in antiretroviral therapy have led to improved life expectancy among those infected with HIV; thus, more chronic diseases are presenting in an aging HIV population. Sports medicine physicians need to be aware of interactions with antiretroviral medications and commonly used drugs in sports medicine.

### Medications Sports Medicine Physicians Should Know

#### Cobicistat

- Pharmacokinetic booster, increases the action of other antiretroviral medications, is a powerful inhibitor of cytochrome p450 3A enzymes including CYP3A4

### Protease Inhibitors

- Commonly used protease inhibitors (PIs) include atazanavir, darunavir, fosamprenavir, indinavir, lopinavir/ritonavir, nelfinavir, saquinavir, and tipranavir, and these are frequently included in combination preparations.
- Mechanism of action: prevent viral replication by selectively binding to viral proteases
- Metabolism of PIs is through the cytochrome P450 system, particularly CYP3A4. Co-administration of drugs also metabolized through CYP3A4 increases serum concentration of both substances.
- Side effects:
  - GI symptoms: nausea, vomiting, and diarrhea
  - Metabolic complications: dyslipidemia, hyperglycemia, glucose intolerance, and type II diabetes mellitus
  - Co-administration of PIs or cobicistat with steroid medications, even injections, can cause steroid excess, adrenal insufficiency, and adrenal crisis. Disruption of the hypothalamic-pituitary-adrenal (HPA) axis has been shown with oral, inhaled, intranasal, and injected corticosteroids.
  - Risk of HPA dysfunction has been shown to be higher in cobicistat or PI-treated patients who are older and who have received more than one steroid injection in a 6-month period.
  - Onset of symptoms is typically 30–90 days after steroid administration.
  - Recommendations:
    - Avoid corticosteroid injections in HIV patients on cobicistat or PIs in favor of alternate management strategies where possible.
    - Consult the patient's HIV specialist before administering corticosteroids.
    - After steroid administration, monitor the patient for signs/symptoms of steroid excess for up to 90 days.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SPORTS PHARMACOLOGY OF PSYCHIATRY AND BEHAVIORAL MEDICINE

# 9

Stephen R. Paul • Bevila John-Daniel • Bruce Helming

## INTRODUCTION

- Often, an optimal treatment regimen is determined through a collaborative approach, including team physicians, psychologists, psychiatrists, athletic trainers, academic advisors, coaches, teammates, parents, and administrative staff. The following nonprescription therapies have been shown to be helpful:
  - Psychotherapy
  - Over-the-counter herbal and dietary supplements
  - Light therapy (for seasonal affective disorder)
  - Electroconvulsive therapy (ECT) (treatment-resistant manic or mixed episodes; bipolar disorder; highly symptomatic, dysfunctional, or suicidal major depressive disorder)
- Athletes often self-treat their conditions with the following:
  - Overtraining
  - Avoidance
  - Self-help information
  - Self-medication with over-the-counter medications, alcohol, and illicit drugs
- Treatment with medications requires evaluation and monitoring:
  - Accurate diagnosis and identifying existing comorbidities
  - Suicidal ideation, self-harm
  - Side effects, effects on school/work/sport performance
  - Efficacy and compliance
  - Attention to potential side effects that may affect athletic performance:
    - Daytime sedation
    - Orthostasis
    - Tremors
    - Arrhythmias
    - Nausea
    - Weight gain or loss
- Table 9.1 lists syndromes associated with the medications reviewed in this chapter: neuroleptic malignant syndrome (NMS), serotonin syndrome (SS), and extrapyramidal symptoms (EPS).
- Use caution with medication selection in pediatric, pregnant, potentially pregnant, or geriatric patients.

## TREATMENT FOR MAJOR DEPRESSIVE DISORDER (MDD)

- Table 9.2 summarizes antidepressant classification and adverse effects.
- Psychotherapy is effective when compared with pharmacologic therapy and may be a safe and effective alternative for depressed patients in primary care.
- Second-generation antidepressants in this chapter include selective serotonin reuptake inhibitors (SSRIs), serotonin–norepinephrine reuptake inhibitors (SNRIs), and other medications that selectively target neurotransmitters with similar mechanisms of action
- Pharmacologic treatment is better than placebo in primary care practice settings (53% vs. 40%)
- Second-generation antidepressants have similar efficacy and response rates (60%), with primary differences in their respective side-effect profiles.
- There is insufficient evidence to evaluate the comparative risk of suicidal thoughts and behaviors or rare but severe adverse events, such as seizures, cardiovascular events, hyponatremia, hepatotoxicity, and SS.
- Treatment is administered in phases:
  - Acute phase: 4–8 weeks are needed for initial therapy with close monitoring
  - Continuation phase: continue to monitor, treat for 4–9 months
  - Maintenance phase: continue to try to reduce the risk of relapse, which is greater if there is a previous history of major depressive disorder (MDD) or chronic MDD
  - Discontinuation of therapy: taper over several weeks, choose optimal time of low stressors and risks or high outside support
- If family history of previous antidepressant use, consider that agent first
- Consider starting with an SSRI
- If failure, consider another SSRI
- If failure of two SSRIs, try an SNRI or another class of antidepressant
- If partial response, optimize or augment treatment with a different class of antidepressant
- For patients with associated insomnia, SSRIs have similar effectiveness; trazodone has been found beneficial for patients with associated insomnia.
- Tricyclic antidepressants (TCAs), particularly imipramine and clomipramine, have reasonable efficacy, but adverse effects limit their use.
- The only FDA-approved medications for adolescents are fluoxetine and escitalopram.

### The Black Box Warning

- Use of antidepressants in young patients under the age of 25 years should balance the risk of suicidal behavior and attempts along with the clinical need. Important considerations with the black box warning are as follows:
  - Mental health disease itself carries inherent risks of suicide, even without treatment.
  - Importance should be placed on frequent and thorough monitoring of the patient for their symptoms and response to medication, particularly in the first few weeks of treatment.

## TREATMENT FOR BIPOLAR DISORDER

- Table 9.3 lists the common medications used in bipolar disorder.
- Bipolar disorder is often managed by or in consultation with a psychiatrist. Hence, treatment modalities and certain salient features of the treatment may be helpful for the primary care physician, particularly to help stabilize patients waiting to consult with a psychiatrist.
- Treatment guidelines include the following:
  - Adjunct therapies like cognitive behavioral therapy (CBT)
  - Education of early warning signs and support for the caregivers
- Omega-3 fatty acids have been shown to be helpful as an adjunct for bipolar depression but not mania.

## TABLE 9.1 SYNDROMES ASSOCIATED WITH MEDICATIONS USED FOR PSYCHIATRIC DISORDERS

| | |
|---|---|
| **Serotonin Syndrome**<br>(Italics distinguish from NMS) | • Potentially fatal, caused by net increase in serotonin<br>• Occurs *rapidly* after medication change (usually within 24 hours)<br>• Symptoms include anxiety, agitation, disorientation, and delirium; autonomic (dilated pupils, tachycardia, hypertension, hyperthermia, diaphoresis, vomiting, and diarrhea); and neuromuscular *(hyperrigidity*, *myoclonus*, *hyperreflexia*, *tremor*, akathisia, *ataxia,* and *shivering)*<br>• Treatment: discontinue serotonergic drugs; supportive management with intravenous fluids, oxygen, blood pressure control, benzodiazepine to control agitation, and cyproheptadine (if other treatments fail) |
| **Neuroleptic Malignant Syndrome**<br>(Italics distinguish from serotonin syndrome) | • May be a life-threatening emergency<br>• *Often develops over days*<br>• Symptoms include mental status changes (confusion, agitated delirium, mutism, and catatonic); muscular *rigidity (lead-pipe rigidity/cogwheeling), bradyreflexia*, hyperthermia, tremor, diaphoresis, tachycardia, and labile blood pressure<br>• Treatment: hospitalization; discontinue offending agent; consider benzodiazepines, dantrolene, bromocriptine, or amantadine and supportive care |
| **Extrapyramidal Symptoms (EPS)** | • Range of movement disorders: dystonia (muscular spasms of neck, jaw, and back), akathisia (restlessness, nervousness, and anxiety), parkinsonism (rigidity, tremor, bradykinesia, shuffling gait, and masked facies), and tardive dyskinesia (involuntary muscle movements of distal extremities and face)<br>• Treatment: consider anticholinergic drugs, beta-blockers, benzodiazepines, and pramipexole |

## TABLE 9.2 MEDICATIONS USED FOR DEPRESSION AND/OR ANXIETY

| Name (Brand) | Mechanism, Adverse Effects (AEs), Comments |
|---|---|
| **Selective Serotonin Reuptake Inhibitor (SSRI)** | • AEs: Sexual dysfunction, drowsiness, weight gain, insomnia, anxiety, dizziness, headache, dry mouth, blurred vision, rash-itching, tremor, constipation, and stomach upset<br>• Caution with the use of nonsteroidal anti-inflammatories, gastrointestinal bleeding and serotonin syndrome |
| **Citalopram (Celexa)** | • Some antihistamine effect |
| **Escitalopram (Lexapro)** | • Best tolerated, fewest drug interactions |
| **Fluoxetine (Prozac)** | • Activating properties (not good with insomnia and anxiety)<br>• Lowest rate of withdrawal symptoms<br>• Long half life |
| **Paroxetine (Paxil)** | • Calming, good for anxiety |
| **Paroxetine CR (Paxil CR)** | • Highest rate of withdrawal due to AEs<br>• Highest rate of sexual AEs (16%) |
| **Sertraline (Zoloft)** | • Activating properties<br>• Diarrhea (common) |
| **Serotonin Noradrenergic Reuptake Inhibitor (SNRI)** | • Not interchangeable, have different levels of NE and 5HT action<br>• Common AEs: nausea, somnolence, dry mouth; dizziness, constipation, weakness; blurred vision, sweating |
| **Desvenlafaxine (Pristiq)** | • Hypertension may occur during titration<br>• Withdrawal reaction<br>• Headache, anxiety, insomnia, and diarrhea |
| **Duloxetine (Cymbalta)** | • Good for depression with chronic pain<br>• Less risk of hypertension than venlafaxine<br>• 67% higher discontinuation due to AEs<br>• Start twice daily then switch to once daily dosing |
| **Levomilnacipran (Fetzima)** | • Enantiomer of milnacipran<br>• Has the most noradrenergic action of SNRIs |
| **Venlafaxine (Effexor)** | • First-line SNRI, most often prescribed<br>• Hypertension may occur during titration<br>• Has 52% higher (nausea, vomiting) AE incidence than SSRIs as a class<br>• 40% higher risk of discontinuation due to AEs<br>• Highest rate of withdrawal symptoms<br>• XR form lower in AE |
| **Serotonin Antagonist and Reuptake Inhibitor (SARI)** | • Mechanism: antagonize serotonin receptor and inhibit reuptake of serotonin, norepinephrine, or dopamine |
| **Trazodone (Desyrel)** | • Priapism<br>• Lower dose increases somnolence (may improve sleep) |
| **Vilazodone (Viibryd)** | • Titrate dose per recommendations<br>• Diarrhea, nausea; sexual dysfunction; dizziness, insomnia, vomiting; dry mouth |

**TABLE 9.2 MEDICATIONS USED FOR DEPRESSION AND/OR ANXIETY—cont'd**

| Name (Brand) | Mechanism, Adverse Effects (AEs), Comments |
|---|---|
| **Other Mechanisms** | |
| **Mirtazapine (Remeron, Remeron SolTab)** | • Mechanism: noradrenergic and specific serotonergic antidepressant (NaSSA) has some alpha-adrenergic and serotonergic properties<br>• Weight gain, sedation<br>• Associated with weight gain (1.8–6.6 lb) after 6–8 weeks<br>• Faster onset of action<br>• Sedation decreases with increased dose |
| **Bupropion (Wellbutrin)** | • Mechanism: norepinephrine and dopamine reuptake inhibitor (NDRI), is both an active drug and precursor<br>• Weight loss<br>• Activating and energizing properties<br>• Low incidence of sexual dysfunction<br>• XR form available |
| **Buspirone (Buspar)** | • Mechanism: selective serotonin 5HT1A receptor agonist and dopamine agonist/antagonist<br>• Off label to augment major depressive disorder<br>• May have role in anxiety if unable to tolerate SSRIs or SNRIs. May not work as well if patient was on long-term benzodiazepine<br>• Multiple dosing may be a drawback |
| **Tricyclic Antidepressants** (e.g., amitriptyline, nortriptyline, and clomipramine) | • **Dangerous and lethal in overdose (QT prolongation leading to arrhythmias)**<br>• Mechanism increases neurotransmitter by inhibiting reuptake of primarily serotonin and norepinephrine<br>• Not used much due to dose-related AEs; potential cardiotoxicity; anticholinergic, antihistamine, and sedating effects; sexual dysfunction; diaphoresis; tremors; and acute hepatitis<br>• Decrease seizure threshold<br>• Evaluate for cardiac risk factors, consider ECG in patients aged >40 years<br>• Onset of action may take 4 weeks<br>• Taper to avoid withdrawal |
| **Monoamine Oxidase Inhibitors** | • Mild anticholinergic (AEs: sedation, agitation, orthostatic hypotension, dizziness, GI effects, weight gain, and suppressed REM sleep)<br>• Not often used due to diet restrictions, drug interactions, and AEs<br>• Risk of SS |
| **Benzodiazepines** (e.g., alprazolam, lorazepam, clonazepam, and diazepam) | • AEs: Sedation, nausea, syndrome of inappropriate antidiuretic hormone secretion, blood dyscrasia, hyponatremia, anterograde amnesia, agitation, depression, cognitive impairment, hyponatremia, and extrapyramidal syndrome<br>• Used in combination in acute settings of mania/hypomania, particularly to reduce agitation in patients with acute mania<br>• Risk of tolerance and addiction<br>• Monitor complete blood count and liver function for long-term use<br>• Contraindicated in patients with myasthenia gravis or acute narrow-angle glaucoma<br>• Caution in patients with substance abuse |

**TABLE 9.3 MEDICATIONS USED FOR BIPOLAR DISORDER**

| Medication | Adverse Effects (AEs), Comments |
|---|---|
| **Antidepressants** (see Table 9.2) | • Concern with using antidepressants as monotherapy for the potential risk of converting to manic state |
| **Typical Antipsychotics (First-Generation Antipsychotics)** (e.g., haloperidol and thorazine) | • AEs: Increased risk of dry mouth, sedation, EPS, NMS and tardive dyskinesia, restlessness, anxiety, headache, weight gain, insomnia, and depression<br>• Use: mania<br>• Increase risk of death in older patients with dementia<br>• Rarely used now with emergence of second-generation antipsychotics |
| **Atypical Antipsychotics (Second-Generation Antipsychotics)** | • AEs: EPS and tardive dyskinesia (but lower risk than typical antipsychotics), anticholinergic symptoms, sedation, metabolic effects (weight gain, hyperglycemia, dyslipidemia, and hyperprolactinemia), cardiac effects, hypotension, cataracts, cardiomyopathy, and sexual dysfunction<br>• Rare: NMS, seizures, agranulocytosis, and hypersensitivity reactions<br>• Before initiation: document, then monitor weight/BMI, pulse and blood pressure, lipid profile, fasting glucose, complete blood count, and consider ECG if cardiovascular risk factors are noted<br>• Target doses often achieved within first week |

*Continued*

**TABLE 9.3 MEDICATIONS USED FOR BIPOLAR DISORDER—cont'd**

| Medication | Adverse Effects (AEs), Comments |
|---|---|
| **Aripiprazole (Abilify)** | • Associated with akathisia and increased risk of EPS and tardive dyskinesia<br>• Less associated with dyslipidemia and sexual dysfunction<br>• Use: mania, mixed<br>• Effective in preventing relapse in mania<br>• Can be used as monotherapy or in combination with lithium or valproate<br>• **Only in class without risk of dyslipidemia**<br>• Can be used in adults and adolescents (aged 10–17 years) |
| **Olanzapine (Zyprexa)** | • Increased risk of weight gain, glucose intolerance, and diabetes<br>• Can be used in adults and adolescents (aged 13–17 years) for manic/mixed, in adults only for depression<br>• Used for agitation in bipolar mania<br>• Effective in preventing manic relapse<br>• Caution in obese patients |
| **Olanzapine With Fluoxetine Combination (Symbyax)** | • Can be used in adults with depressive symptoms<br>• Effective in preventing manic relapse<br>• Caution in obese patients |
| **Quetiapine (Seroquel)** | • Lower risk of EPS (less than others in the class) and tardive dyskinesia<br>• Risk of weight gain, diabetes, glucose intolerance (less than olanzapine), and sedation<br>• Use: mania, mixed, depression<br>• Can be used in adult and adolescents (aged 10–17 years) for acute mania<br>• Superior to monotherapy for maintenance if added along with lithium or valproate |
| **Risperidone (Risperdal)** | • Increased risk of EPS and tardive dyskinesia, monitor prolactin levels<br>• Risk of weight gain, diabetes, and glucose intolerance (less than olanzapine)<br>• Can be used as short-term therapy for acute manic or mixed states and in adults and adolescents (aged 10–17 years) |
| **Anticonvulsants** | • Monitor levels and check complete blood count and liver function |
| **Valproic Acid (Depakene)**<br>**Divalproex (Depakote)** | • AEs: Tremor, sedation, weight gain, nausea, diarrhea, alopecia, leukopenia, increase in liver enzymes, liver failure, pancreatitis, and PCOS<br>• Use: mania (rapid cycling), depression, mixed<br>• Caution with liver disease<br>• Not recommended for women of childbearing potential<br>• Polycystic ovary syndrome more common in women who start therapy before the age of 20 years<br>• More effective than lithium for mixed states |
| **Lamotrigine (Lamictal)** | • Dizziness, tremor, somnolence, headache, nausea, anticholinergic, rash, Stevens–Johnson syndrome, toxic epidermal necrolysis, leukopenia, thrombocytopenia, pancytopenia, and aseptic meningitis<br>• Use: mania, depression<br>• Weight loss associated with use in obese patients<br>• **Acceptable for pregnancy**<br>• Reduced risk of rash with slow titration<br>• Black box warning for increased risk of Stevens–Johnson syndrome |
| **Other (Mood Stabilizer)**<br>**Lithium** | • AEs: Excessive thirst, polyuria, sedation, tremor, nausea, loose stools, cognitive effects, weight gain, hypothyroidism, and diabetes insipidus<br>• Use: mania, depression<br>• Before initiation, check, then monitor: weight/BMI, electrolytes, estimated glomerular filtration rate, thyroid, complete blood count, and consider ECG if risk factors for cardiovascular disease are noted<br>• Monitor serum lithium levels 1–2 times in the first week and 1 week after dose change<br>• Toxicity is dose dependent<br>• Caution: renal disease, avoid NSAIDs and maintain hydration status<br>• Compared to valproate or carbamazepine, lithium lowers the risk of suicide<br>• Protective against dementia<br>• Depressive symptoms not improved by adding SSRI or bupropion |

*EPS,* Extrapyramidal symptoms; *NMS,* neuroleptic malignant syndrome.

- Treatment protocol depends on presenting symptoms (mania, hypomania, mixed state, depression, or maintenance), comorbidities, adverse effects, whether the patient (or a family member) has successfully been treated for bipolar disorder before, and the cost of medication.
- Fewer relapses have been noted with the combination of pharmacology and psychotherapy.

### Mania/Hypomania/Mixed States

- Acute mania with risk of harm to self or others is initially treated with hospitalization and psychiatric consultation.
- Severe mania is often treated with lithium/valproate and augmentation with an antipsychotic (haloperidol, olanzapine, quetiapine, or risperidone).
- Severe mania may not respond to single-drug therapy.

- ECT is considered for severe mania that is nonresponsive to multidrug therapy.
- If there is abrupt mood elevation with antidepressants, taper and discontinue its use of and adjust the dose of the mood stabilizer/antipsychotic.
- Avoid alcohol and drug abuse, caffeine, and nicotine.

### Bipolar Depression

- Use of antidepressants as monotherapy increases the risk of activating a manic spell.
- Consider lithium or lamotrigine as first-line therapy; other options include quetiapine, aripiprazole, or olanzapine.
- New-onset, untreated bipolar depression: consider quetiapine or lamotrigine as monotherapy or start with olanzapine combined with fluoxetine
- With a sleep disturbance, avoid using trazodone, which can induce mania (consider using benzodiazepines)
- If associated psychosis, add an antipsychotic

## TREATMENT OF ANXIETY DISORDERS

- Treatment approach for anxiety similar to depression (Table 9.2)
- Obsessive compulsive disorder may need higher doses of SSRIs.
- Athletes with anxiety disorders often welcome treatment; their symptoms may affect their athletic performance/participation.
- Several anxiety disorders will respond to counseling, but this may take time.
- Initiate treatment with SSRI
- Second-generation antidepressants have similar efficacy (60%) in the treatment of anxiety and depression.
- In a small-scale study of sport psychiatrists, buspirone was preferred for treatment in athletes but was considered as second-line therapy when compared with SSRIs (may be less effective in patients recently taking a benzodiazepine).
- Consider using benzodiazepines as a "short bridge" at acute onset while awaiting effects of treatment with SSRI; best to avoid or minimize benzodiazepine use during season due to side effects of sleepiness, balance and coordination problems, and potential for abuse
- TCAs (particularly imipramine and clomipramine) are rarely used due to adverse effects, common interactions with other medications, potential lethality with overdose, and increased risk of suicide.

## TREATMENT OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADD/ADHD)

- Stimulant medications are used as first-line therapy for attention-deficit/hyperactivity disorder (ADD/ADHD) in most cases and are similarly effective and safe in both children and adults (see Table 9.4 for stimulant medications).
- **Stimulant medications may be banned, prohibited in competition, or require specific documentation or formal permission from governing bodies; therefore, review the rules for athletic participation.**
- Dosing should be titrated (can be done quickly) to maximize response; increase dose over 3–7-day intervals.
- Consider goal of maximum tolerable dose rather than minimum effective dose (increased dose may yield additional benefits)
- Short- and long-acting medications are similarly effective, consider once-daily dosing for convenience and cost effectiveness

**TABLE 9.4 MEDICATIONS USED FOR ADHD**

| Stimulant Medications | Onset (Minutes) | Duration (Hours) |
|---|---|---|
| **Long-Acting** | | |
| Methylphenidate (Concerta, Daytrana, Metadate, Quillivant, Ritalin LA) | 90 minutes: capsule<br>30–60 minutes: tablet<br>60–270 minutes: patch | 7–12 hours: capsule<br>8–12 hours: tablet<br>10–12 hours: patch |
| Dextroamphetamine/amphetamine (Adderall XR) | 20–60 minutes | 8–10 hours |
| Dexmethylphenidate (Focalin XR) | 30–60 minutes | 8–12 hours |
| Lisdexamfetamine (Vyvanse) | 60–120 minutes | 10–14 hours |
| **Intermediate-Acting** | | |
| Methylphenidate (Ritalin, Metadate ER) | 60–180 minutes | 3–8 hours |
| Dextroamphetamine (Dexedrine) | 60–90 minutes | 6–10 hours |
| **Short-Acting** | | |
| Methylphenidate (Ritalin, Methylin) | 20–60 minutes | 3–6 hours |
| Dexmethylphenidate (Focalin) | 20–60 minutes | 3–6 hours |
| Dextroamphetamine/amphetamine (Adderall) | 20–60 minutes | 5–7 hours |
| Dextroamphetamine (Zenzedi, Procentra) | 20–60 minutes | 4–6 hours |
| **Selective Norepinephrine Reuptake Inhibitor** | • Nonstimulant, not restricted for athletes and not subject to controlled substance restrictions<br>• Effect size is smaller and onset of clinical response may be delayed (up to 4–8 weeks)<br>• Adverse effects: drowsiness, nausea, GI upset, and decreased appetite | |
| Atomoxetine (Strattera) | 1–2 hours | 24 hours |
| **Alpha-Adrenergic Agonists** | • Nonstimulant, used in pediatric patients<br>• Can take 1–2 weeks to achieve clinical effect<br>• Adverse effects: somnolence and dry mouth | |
| Guanfacine ER (Intuniv) | 30–45 minutes | 4–8 hours |
| Clonidine (Kapvay) | 30–60 minutes | 3–8 hours: tablet<br>4–7 days: patch |

- Flexibility in dose/formulations allows individualization to symptoms/schedule.
- No need for tapering to discontinue
- In children, approximately 75% will respond to first medication, and 90%–95% will respond to second, although response can be idiosyncratic.
- Monitor: medication use, side effects, response to medication, mood, weight, heart rate, and blood pressure
- Frequent side effects include appetite loss, weight loss, anxiety, irritability, and insomnia.
- In children, abdominal symptoms and a small reduction in height velocity may occur (total height loss of 1–2 cm).
- Medications may also benefit sports and daily life as well as academic/work environments; hence, assess function in all realms.
- One third will have comorbid psychological diagnosis; so screen for medical conditions causing similar symptoms (including substance abuse) and cardiac conditions.
- For coexisting depression, combination of SSRIs and stimulants is safe and effective; consider single-agent treatment with bupropion.
- For coexisting anxiety, stimulants are still indicated, consider in combination with SSRIs and/or CBT
- Use of stimulant medications does not appear to increase the incidence of substance abuse.
- Diversion and abuse may be less likely with longer-acting agents.

## PHARMACOLOGIC TREATMENT OF EATING DISORDERS

- Eating disorders are commonly associated with other psychiatric disorders such as anxiety, depression, obsessive compulsive and impulse control disorders, and substance abuse.
- Treatment is challenging and usually involves a multidisciplinary approach that involves psychosocial intervention and medications.
- Overall, combination of CBT and medication was found superior to medication alone

### Anorexia Nervosa

- Individuals who are severely underweight and medically compromised are best served in a more intensive program including hospitalization or an intensive outpatient program where focus is on weight restoration, psychotherapy, and nutritional counseling.
- Medications may be used as an adjunct but there is no clear consensus of efficacy; be sure to address comorbidities such as depression and anxiety
- SSRIs (Table 9.2) may help manage severe comorbid psychiatric disorders.

### Bulimia Nervosa

- SSRIs (Table 9.2) form the first-line pharmacologic treatment.
- Fluoxetine is the only FDA-approved medication for treatment.
- Emerging research on other medications with potential benefit

## TREATMENT OF INSOMNIA AND JET LAG

- Psychological and behavioral treatments aid in treatment of insomnia. CBT, sleep hygiene education, along with relaxation therapy are preferred over medications.
- Medication may improve sleep (both short- and long-term).
- Medications include newer hypnotics (zolpidem, zaleplon, eszopiclone, or ramelteon), benzodiazepines, and sedating antihistamines. Individual classes and agents have unique side effects.
- Reduce jet lag by adjusting circadian schedule before, during, and after travel.
- Melatonin reduces jet lag. Prescription medications can help with sleep while adjusting to change in time zones.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE PEDIATRIC ATHLETE

# 10

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## GENERAL PRINCIPLES

- A pediatric athlete can be any child or adolescent usually under the age of 18 years who participates regularly in sports activities.
- Understanding physical and physiologic differences between pediatric and adult patients aids in the prompt recognition and management of most medical and orthopedic conditions affecting a pediatric athlete.
- Activity type, skill level, and motivation for sports participation greatly varies at different ages and levels of maturity; therefore, it is best to understand young athletes in the context of their chronologic age, developmental stage, and physical maturity, coupled with an assessment of the nature and level of sports participation.
- It is important to understand both the child's and parent's motivation for sports participation and to capitalize on opportunities to educate parents, athletes, and coaches on healthy athletic participation and sports safety.
- Sports specialization is defined as intense activity in only one sport throughout the year. Avoiding early sports specialization may decrease the risk of injury, overtraining, and burnout.
- The ultimate goal of youth sports participation should be the promotion of life-long physical activity, pursuit of recreation, and enjoying the challenge of competition.
- Healthcare professionals face challenges at both ends of the physical activity spectrum: the sedentary obese child, who faces a lifetime of morbidity related to physical inactivity, and the highly competitive, overzealous, potentially undernourished young athlete are both at risk for a myriad of injuries associated with sedentary behaviors or excessive exercise. A successful pediatric athlete will lead a healthy, balanced lifestyle and integrate exercise, nutrition, and recreational pursuits with an adequate amount of rest and recovery.

## MEDICAL CONCERNS OF THE PEDIATRIC ATHLETE

- Several conditions that affect pediatric athletes are similar to those that affect adult athletes. Examples include cardiac conditions (e.g., cardiac arrhythmias), pulmonary conditions (e.g., asthma), mental health conditions (e.g., eating disorders and stress/anxiety/depression), endocrine disorders (e.g., diabetes and obesity), renal conditions (e.g., polycystic kidney disease), and infectious diseases (e.g., mononucleosis).
- Prompt recognition and management of these conditions lead to safe and early return to sports.
- A preparticipation physical examination (PPE) (refer to Chapter 3) is recommended for pediatric athletes before organized sports participation. It is usually a state-mandated legal requirement for participation in high school interscholastic athletics.
- The PPE is a helpful tool to help physicians identify medical conditions that may affect participation in sports and physical activity.

## EXERCISE AND THE PEDIATRIC ATHLETE

- According to the American Heart Association, children and adolescents should participate in at least 60 minutes of moderate to vigorous activity daily.
- Multiple small periods of activity, such as two 30-minute periods or four 15-minute periods of exercise, are acceptable alternatives.
- Suggestions to encourage physical activity:
  - Limit or reduce sedentary time (television, computer, video games, and phone) to 30 minutes/day.
  - Find fun activities that children enjoy.
  - Incorporate parent role models.
  - Emphasize the social aspect of participating in team sports.
  - Promote the use of activity trackers (step counters, wireless trackers, etc.).
- There are numerous benefits of exercise in pediatric patients (Box 10.1).

### Physicians, Patients, and Exercise

- A recent study found that approximately 47% of primary care physicians self-report the inclusion of exercise history during patient examinations.
- The activity prescription "MD FITT" is a useful tool for guiding and tracking physical activity (Table 10.1).

## CHILDHOOD OBESITY

- Obesity is the most important health concern among children in the United States (US).
- The prevalence rate of obesity in children is 11%–22%, and it has doubled in the past 20 years.
- Childhood obesity is increasing at an epidemic rate, particularly in economically disadvantaged areas and minority populations.

### Age Range: Preschool Through High School

- Preschoolers spend approximately 11% of their time in vigorous activities, 60% in sedentary activities, and an average of 3–5 hours/day watching television.
- Every hour of television is associated with a 2% increase in obesity risk.

### Risks of Adult Obesity

- 50% of children who are obese at the age of 6 years are likely to remain obese in adulthood.
- 70%–80% of children who are obese at the age of 10 years are likely to remain obese inadulthood.
- Additional risk is associated with concurrent parental obesity: 23% of all deaths in the US are associated with sedentary lifestyles that begin in childhood.

### Body Mass Index (BMI) in Children

- BMI = [weight (kg)]/[height $(m)^2$]
- A child with a BMI in the 85th to 95th percentile is considered overweight and at a risk of obesity.
- A child with a BMI in the 95th percentile and above is considered obese.
- Annual BMI calculation is recommended for children during routine and sports physical examinations and can be followed longitudinally. Pediatric growth charts based on age and

**BOX 10.1 COMMON BENEFITS OF PHYSICAL ACTIVITY IN CHILDREN AND ADOLESCENTS**

Weight control
Lowers blood pressure
Raises HDL or "good" cholesterol
Reduces risk of diabetes
Improves self-esteem

**TABLE 10.1 DESCRIPTION OF 'MD FITT' EXERCISE PRESCRIPTION**

| | |
|---|---|
| M-MODE | What type of activity (e.g., walking or biking) |
| D-DURATION | For how long does the patient exercise daily? |
| F-FREQUENCY | How often does the patient exercise (days/week)? |
| I-INTENSITY | How intense is the exercise (e.g., moderate)? |
| T-TIMELY FOLLOW-UP | How often the patient re-visits the clinician |
| T-THERAPY | Are there any concerns of injury or side effects? |

gender include BMI and are available online (www.cdc.gov/growthcharts). Numerous EMR systems calculate BMI when height and weight measurements are entered.

### Causes of Childhood Obesity

- Energy intake is greater than energy expenditure.
- Endocrine, hormonal, and genetic syndromes can each cause or contribute to obesity in children.

### Complications of Childhood Obesity

- Any and all organ systems in the body can be affected by childhood obesity: cardiac, orthopedic, endocrine, gastrointestinal, respiratory, and neurologic systems are among those most often affected.

### Treatment Recommendations

- Assessment of energy intake and output, physical examination, and laboratory evaluation to exclude other causes of obesity as well as providing nutritional and exercise education
- Nutritional interventions include changes in advertising, healthy school lunches, and adequate and varied healthy food choices in the home environment.
- Exercise recommendations include increased recreational activities, organized sports participation, preservation of adequate physical education time in school, and decreased sedentary screen time (e.g., computer and television).
- A meta-analysis of 30 randomized controlled trials in children aged 5–17 years found that low-intensity, long-duration exercise coupled with resistance training was highly effective in altering and improving body composition.

## GROWTH AND MATURATION AND THE YOUNG ATHLETE

- Concerns regarding potential negative effects of athletic competition on growth and maturation have existed for many years, particularly attributable to the trend of intense competition at younger ages.
  - The demands of sports require a certain level of physical and psychological maturity in order to participate. Feelings of insecurity, frustration, and failure may cause young athletes to quit because of burnout or inability to perform up to expectations.
  - While young athletes are struggling to master advanced sports-specific skills, their coaches may be less experienced and less educated in appropriate training techniques; these barriers can negatively affect a young athlete's enjoyment and participation in his or her sport(s).
- **A significant challenge for sports medicine healthcare providers** is the consideration of the development of the neurologic, cognitive, somatic, and psychological interdependent processes and the effects of each of these on the health and well-being of pediatric athletes.
  - An understanding of fundamental principles of normal child and adolescent growth and development is essential in providing quality healthcare for young athletes.
- Growth and maturation is a natural, fundamental, continuous process, with achievement of the same milestones in the same order.
  - The rate of progression varies greatly and seems predominantly genetically regulated.
  - Neuropsychological development often does not parallel physical development.
  - Growth refers to an increase in size of the body and its parts, including stature, body systems, and body composition.
  - Maturation refers to a biologically mature state of skeletal, sexual, and somatic development with variable timing and tempo.
  - Neurodevelopment is culturally mediated and is the acquisition and mastery of behavioral competence.
  - Quantitative milestones are easy to measure by the number of skills performed.
  - Qualitative milestones are harder to measure because they reflect mastery of specific skills.

### Neurodevelopmental Domains

**Motor:** fine and gross motor, strength, and endurance
**Visual:** acuity, discrimination, and tracking
**Cognitive:** attention, alertness, memory, comprehension, and solving complex problems or simultaneously performing multiple tasks
**Language:** receptive and expressive
**Auditory:** hearing acuity and processing, sound discrimination, and auditory cues
**Emotional and psychological:** relationships with teammates and coaches and regulation of emotions
**Motor:** fine and gross, visual–spatial discrimination, temporal sequencing, proprioception, sports-specific motor adaptive skills, muscular strength and endurance, and reaction time

### Motor Developmental Milestones in Various Age Groups

- Understanding developmental milestones from infancy through young adulthood is essential in successfully caring for the constantly growing pediatric athlete.
- It is difficult to "skip" major neuromuscular milestones during periods of growth; however, the rate at which young athletes progress is sometimes accelerated.
- Accelerated motor development can be problematic if the young athlete is not psychologically or emotionally ready to fully function at this new level of expectation and skill.
- Healthcare professionals should be familiar with the sequence of skill acquisition that is predictable among various young athletes.
- **Preschoolers (4–6 years)**
  - Ride bike without training wheels
  - Hop six times on one foot

- Catch a small ball thrown from 10 feet
- Run, gallop, and skip using alternating feet
- Broad jump up to 3 feet
- Throw a ball with a shift of their bodies at a target
- Move from parallel play to interactive play with others

- **Middle Childhood (6–11 years)**
  - Gender differences can be observed.
  - Girls excel at hopping, skipping, catching, and balance.
  - Boys excel at striking objects, jumping (vertical and long), kicking, and throwing and can run faster.

### Implications for Sports Participation in Young Athletes

- Coach and parent reaction with appropriate feedback is crucial in sports development.
- Confidence, self-esteem, and body awareness are all developing.
- Young athletes should be taught to think "I'm learning and improving" rather than "I can or can't".

### Gender Differences

- In preadolescents, there is little difference in strength, power, and endurance between boys and girls.
  - Girls are consistently more flexible.
  - Boys are consistently better throwers.
- Power and maximal oxygen uptake ($VO_2$ max) increase linearly with age until adolescence when it accelerates. However, in adolescent boys, accelerated rate gains far exceed than those seen in adolescent girls and are partly a result of increased muscle mass in such boys.

### Neuropsychological and Emotional Readiness

- Peer relationships with teammates involve the ability to take turns, attend to the game, focus, and participate in teamwork.
- The coach–athlete relationship requires the ability to follow rules, understand strategies, and control emotions.

### Implications for Organized Sports Participation

- Physical maturation is necessary to master sports-specific skills.
- Neurodevelopmental maturation allows simultaneous functional integration of multiple skills to meet the demands of the competition.
  - **Motor:** e.g., a soccer player needs to simultaneously run and kick in a coordinated fashion
  - **Visual:** monitor for position of teammates and defenders
  - **Auditory:** process instructions from coaches
  - **Language:** communicate with teammates and coaches
  - **Cognitive:** problem solving and implementing sports strategies
  - **Emotional:** possess the ability to process various emotions such as excitement, anxiety, elation of winning, and frustration of losing
- Athletes that are competing in sports at levels above their neurodevelopmental abilities will be more likely to experience negative feelings such as frustration, anger, and lower confidence and self-esteem. Moreover, they will be less likely to have fun and to enjoy the overall sports participation experience. Drop-out as well as injury rates may be higher in these situations.

### Psychological Concerns

- Following are the common mistakes made by parents that negatively influence young athletes:
  - Choosing sports participation based on what the parent wants and not what the child wants
  - Push their children to "over-train"
  - Criticize the performance of the young athlete
  - Promote a "win-at-all-costs mentality"
  - Allow early sports specialization, often in the parent's sport of choice
  - Serve as parent-coaches who either favor or disfavor their own children
  - Lack of knowledge about common overuse injuries

### Sports Safety

- Following modifications are appropriate for young, elementary-school-aged participants:
  - Use smaller fields and courts and small-sided games (e.g., 6 vs. 6 instead of 11 vs. 11) to encourage participation, activity, and skill acquisition
  - Use size- and weight-appropriate equipment
  - Shorten duration of games and practice sessions
  - Adequate number and length of breaks with opportunities for hydration
  - Monitor environmental conditions
  - Establish emergency action plans (EAPs) that must be implemented for emergencies related to injury, illness, or environmental conditions
  - Additional time during sports participation dedicated for teaching and enforcing rules and safety
  - Promote equal playing time and rotate positions
  - Avoid score keeping and win–loss records; reinforce "fun" as the goal of sports participation

## YOUTH STRENGTH TRAINING (ALSO SEE CHAPTER 15)

- Muscle strength development is an important topic with limited evidence-based information.
- Strength training may be a component of a young athlete's exercise regimen and may be included to increase performance and reduce obesity.
- Further research is needed to address the role of strength training in pediatric athletes.

### Definitions

- *Strength training* is a broad term that is defined as the use of resistance methods to increase one's ability to exert or resist force. Machines, free weights, and/or a person's body weight can be used.
- Olympic-style *weightlifting* and *powerlifting* are competitive sports that contest maximum lifting ability.
  - Olympic-style *weightlifting* involves the clean-and-jerk and the snatch.
  - *Powerlifting* involves the squat, the bench press, and the dead lift.
- *Bodybuilding* is an esthetic sport that involves weight training but not competitive lifting.

### How Much, How Soon?

- Following appropriate resistance techniques and safety can be safe and effective for preadolescents and adolescents.
- Children and adolescents should refrain from Olympic-style weightlifting, powerlifting, and bodybuilding until skeletally mature.
- It is important to note that several sports use intrinsic strength techniques to perform sports-specific exercises, such as the young gymnast who tumbles and vaults bearing his/her entire body weight on his/her hands and wrists.

### Anatomy and Physiology: Training Effects on Strength

- Strength gains are a result of neuromuscular adaptation in preadolescents. Muscle hypertrophy is not seen in preadolescents but is evident during puberty.
- Effects of strength training on body composition can be evident at all ages and exert favorable effects on improving lean body mass, particularly in obese children.
- Increases in neuronal activation and intrinsic muscular adaptations and improvements in motor coordination (learning) seem to play vital roles in strength development during childhood.
- No long-term studies have been conducted on the effects of preseason resistance training on improved sports performance in children.
- It appears that strength gains made during training can be lost during periods of rest or "detraining." These must always be evaluated in the context of naturally occurring strength gains that are associated with normal growth and development.
- Good nutrition and age-specific activity guidelines should be followed. Strength training has no known negative effects on growth.

### Risk Factors for Injury

- The greatest injury risk to children who perform strength training is lack of appropriate supervision.
- Other risks include inappropriate technique, inappropriately sized equipment, or inappropriate amounts of weight and repetitions.
  - Common acute injuries in weight training include fractures and muscle–tendon injuries.
  - Overuse injuries include sprains, strains, and growth plate injuries. Injuries to the spine and shoulders are most common.
  - No cases of growth plate fractures have been reported under appropriately supervised settings.

### Guidelines for Strength-Training Programs

- Program design considerations should include education on appropriate techniques, progression, function, and fun.
- Strength training should be incorporated into the child's exercise program with aerobic conditioning, flexibility, and sports participation.
- The following should be kept in mind when creating a strength-training program:
  - Body weight exercises should be done first.
  - Perform 2–3 sessions per week, including 2–3 sets of 10–15 repetitions of each exercise
  - Implement variations in a program and gradually add weight in 10% increments
  - Use child-sized equipment for preadolescents, such as smaller, 1-pound plates, small dumbbells, and/or resistance bands
  - Appropriate supervision at all times is essential
  - Emphasize correct form and technique
  - Use single- and multiple-joint activities and train antagonistic muscle groups equally
  - Design an individualized program, vary it regularly, and maintain workout logs to monitor progress

## CHANGING TRENDS IN EXERCISE PATTERNS

- As an increasing number of children are participating in organized athletics at younger ages, the incidence of overuse injuries is increasing. It is estimated that 30–45 million youngsters aged 6–18 years participate in athletics (two thirds in organized and one third in recreational sports). Many children participate in year-round same-sports activities or simultaneously on multiple sports teams.

## EFFECTS OF EARLY SPORTS SPECIALIZATION

- Early sports specialization has become more common.
- Participation in one sport and year-round participation can be both physically and mentally detrimental to children, leading to burnout and overuse injuries.
- It is important to assess the nature and level of sports participation in young athletes in context of their chronological age, developmental stage, and physical maturity.
- Recent research has revealed decreased rates of injury and less psychological stress by avoiding sports specialization before puberty.
- Early sports specialization is most often adult driven.
- There is an assumption that practicing only one sport will lead to better abilities and achievements in that sport.
- There is evidence that specialization in a sport before puberty is not necessary to become an "elite athlete" (e.g., earning a college scholarship, making it to professional status).
- Although some degree of sports specialization is necessary to improve skills within a field of sport, this should, in most cases, occur after the athlete reaches puberty. This can lead to a wider range of developed physical and mental skills within a sport that are gained from playing other sports before specialization. In addition, athlete burnout and the risk of quitting sports can potentially be decreased.
- Recent research has revealed that pediatric athletes who did not start intense training and specialized competition until after puberty were more likely to achieve an elite status within their sport, with the exception of women's gymnastics. However, gymnasts are at a significant risk of burnout and overuse injuries.
- Overtraining can lead to burnout and/or increased injuries, which can eventually cause a young athlete to stop participating in athletic activities, sometimes permanently. Parental influence is a critical confounding factor that affects a young athlete's participation in sports and must be addressed by physicians who care for pediatric athletes.

### Risks of Overuse Injuries

- There are both intrinsic and extrinsic risk factors for overuse injuries (Table 10.2).
- Recommendations for overuse injury prevention include the following:
  - Limit one sporting activity to 5 days per week
  - Provide 1 day of rest from organized activity
  - Take 2–3 months off per year from a single sport
  - Alter workout routines to maintain interest and fun
  - Educate athletes and parents about wellness, nutrition, sleep, hygiene, and stress management
  - Monitor for warning signs of injury
  - Monitor special events such as tournaments and showcases of fatigue, pain, and alterations in sports performance

**TABLE 10.2 FACTORS AFFECTING THE RISK OF OVERUSE INJURIES**

| Extrinsic Factors | Intrinsic Factors |
|---|---|
| Training errors | Growth |
| Schedules | Anatomic alignment |
| Workload | Muscle–tendon imbalance |
| Environment | Flexibility |
| Psychological factors | Prior injury and conditioning |
| Equipment | Menstrual dysfunction |

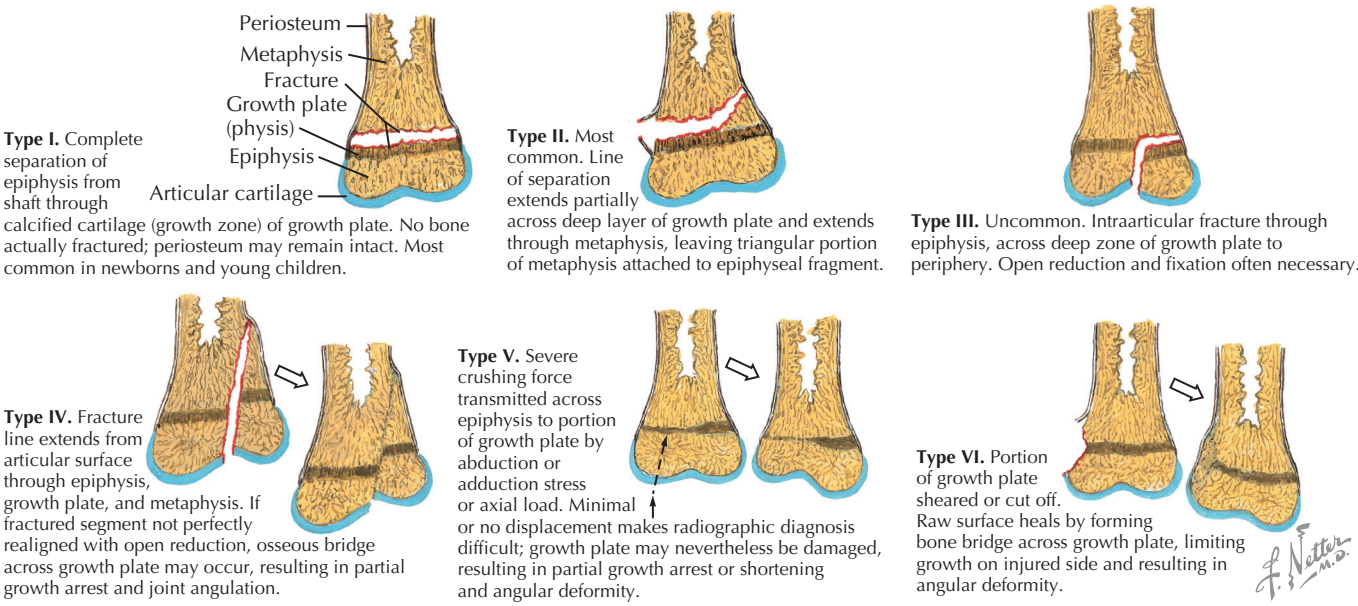



**Figure 10.1.** Injury to growth plate (Salter–Harris Classification, Rang Modification).

## ORGANIZED SPORTS FOR CHILDREN AND PREADOLESCENTS

- Changing trends include a movement away from spontaneous, unstructured activity that allows imagination, enjoyment, and motor skill development.
- Organized sports participation is greatly influenced by goals and expectations of parents and coaches.
- Reactions and feedback from parents and coaches have a strong influence on the attitudes and confidence of preadolescent athletes.
- If the focus is placed on skill development, cooperative play, and having fun, then athletes seem to have greater enjoyment of sports participation.
- Advantages of organization include establishment of rules for participation, equity in matching competitors at similar skill levels, definition of readiness-to-play criteria, and fairness in the establishment of teams to promote safe participation.

### Sports-Specific Issues: Endurance Event Competitions

- Endurance events such as marathons and triathlons are increasing in popularity among youth athletes.
- The American Academy of Pediatrics has stated that "triathlons for children and adolescents are reasonably safe as long as the events are modified to be age appropriate."
- Modifications include shorter duration of activities and following conservative guidelines for safety and environmental conditions (e.g., EAPs and exercising in heat and humidity).
- Training regimens are often altered from traditional plans, including lower weekly mileage, gradual increases in training, and extra attention to hydration and nutrition.

## COMMON PEDIATRIC SPORTS INJURIES RELATED TO GROWTH AND DEVELOPMENT

### Physeal Fractures

**Overview:** Injuries unique to young athletes are growth plate fractures. An injury at the end of a long bone in a skeletally immature athlete is a physeal (growth plate) fracture until proven otherwise. The physis, made of bone and cartilage, is relatively weaker than the surrounding ligaments, tendons, joint capsule, and other soft tissue structures; thus, application of excessive force to the musculoskeletal system will likely result in a physeal fracture.

**Presentation:** The highest rates of physeal fractures are seen during the growth spurt of adolescence and are usually associated with concentric, eccentric, or shear forces applied on the physis at the time of injury. The Salter–Harris (SH) classification is based on the pathoanatomy of fractures and the radiographic appearance of the bone itself (Fig. 10.1):

- Type I: transphyseal injury
- Type II: transphyseal injury with metaphyseal extension
- Type III: transphyseal injury with epiphyseal extension into the joint space
- Type IV: extension from the epiphysis through the physis and into the metaphysis
- Type V: rare and involve a crush injury to the physis itself

**Physical examination:** Most common finding on examination is tenderness at the ends of long bones over the physis. Swelling, ecchymosis, bony deformities, inability to bear weight, or decreased resistance on strength testing may be present.

**Diagnosis:** Requires a high index of suspicion and a basic knowledge of growth plate anatomy.

- Radiographs are required.
- A bone scan, magnetic resonance imaging (MRI), or computed tomography (CT) scan may be necessary if the diagnosis is in question or the patient is not responding to clinical treatment as expected.

**Treatment:** Treatment depends on the location and classification of the injury as well as the degree of displacement of the fractured fragment.

- Most cases require casting with or without a closed reduction for minimally displaced fractures.
- Other fractures with significant displacement and/or instability of fractured fragments will require an open reduction-internal fixation (ORIF).
- Certain SH-I fractures, such as SH-I distal radius and fibular fractures, can be treated with splints.

**Prognosis and return to play (RTP):** Prognosis of SH-I to SH-III fractures varies but in general is good. Partial or

complete growth arrest with malalignment is the greatest risk factor.

- Monitoring for 6–12 months after injury is typically required to assess continued long bone growth.
- RTP varies from approximately 3 to 12 weeks; immobilization is usually 3–6 weeks as physeal injuries heal quickly.
- General RTP criteria should apply, including an assessment of range of motion, strength, and ability to perform sports-specific exercises with or without the use of a brace or other assistive device.

## Apophyseal Injuries

**Overview:** Apophyseal injuries include both acute and overuse injuries and represent a stress injury to growth plates. Acute injuries are avulsion fractures at the apophyseal growth plate where a muscle–tendon unit attaches. Overuse injuries are far more common and typically present in a similar fashion as tendonitis with growth plate involvement.

**Presentation:** Concentric, eccentric, and shear forces create excessive biomechanical force at the apophysis that can initially result in a painful acute inflammatory condition (apophysitis). Excessive workload of repetitions of a sports-specific maneuver can lead to a chronic overuse tendinopathy presentation. Certain injuries are predictable in certain types of sports based on the stresses to the body in that sport. Common types of apophyseal injuries have been summarized in Table 10.3.

**Physical examination:** Tenderness on palpation at the tendon and the apophysis is common. Perform a general assessment of biomechanical and genetic predisposition (e.g., check for severe pes planovalgus feet, genu valgum, or benign hypermobility)

**Diagnosis:** Diagnosis is often clinical, based on a high index of suspicion. Radiographs are helpful to evaluate acute injuries, malalignment, and growth disturbances but are frequently normal. Additional imaging studies are useful adjuncts for more chronic, severe, and persistent cases.

**Treatment:** Acute injuries may require a brief period (2–4 weeks) of immobilization, but more severe injuries can require ORIF to correct significant displacement (e.g., avulsion of the tibial tuberosity in the knee). Overuse injuries are treated similarly as tendonitis/tendinosis with relative rest and guided rehabilitation. Rehabilitation focuses on stretching to minimize forces at the apophysis as well as to improve strength and proprioception. An assessment of sports-specific biomechanics may aid in the prevention of recurrences if errors in technique are corrected.

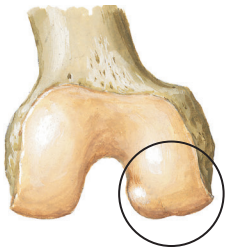
**RTP:** RTP is allowed when pain has resolved and the athlete can perform the necessary sports-specific maneuvers. A guided gradual resumption of normal sports activities is usually necessary to prevent frequent recurrence of symptoms.

## Osteochondritis Dissecans (OCD)

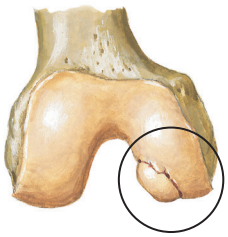
**Overview:** The term *osteochondritis dissecans* (OCD) is attributed to Konig in 1883 and is based on the theory that inflammation contributes to subchondral necrosis of bone and cartilage (Fig. 10.2). Trauma, ischemia, ossification defects, and genetic abnormalities are all likely to contribute to the pathophysiology. OCD has a juvenile and an adult form, distinguished by closure of the physes. The role of subchondral bone in providing

**TABLE 10.3 COMMON APOPHYSEAL INJURIES IN THE PEDIATRIC ATHLETE**

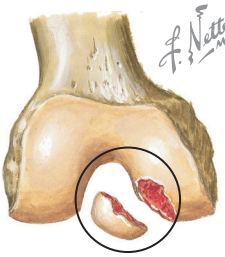
| Injury | Location |
|---|---|
| Little League Elbow | Medial Epicondyle at Flexor–Pronator Attachment |
| Little League Shoulder | Proximal Humerus Epiphysis |
| Gymnast's Wrist | Distal Radius Epiphysis |
| Sinding–Larsen–Johansson Syndrome | Inferior Pole of Patella |
| Osgood–Schlatter Disease | Tibial Tuberosity |
| Iselin's Disease | Base of Fifth Metatarsal |
| Sever's Disease | Calcaneus at Achilles Tendon attachment |
| Pelvis Apophysitis | Anterior Superior Iliac Spine, Anterior Inferior Iliac Spine, Iliac Crest |
| Hip Apophysitis | Ischial Tuberosity, Greater and Lesser Trochanter |



**Stage 1.** Bulge on medial femoral condyle due to partial separation of bone fragment. Articular cartilage intact, but defect evident on radiographs.



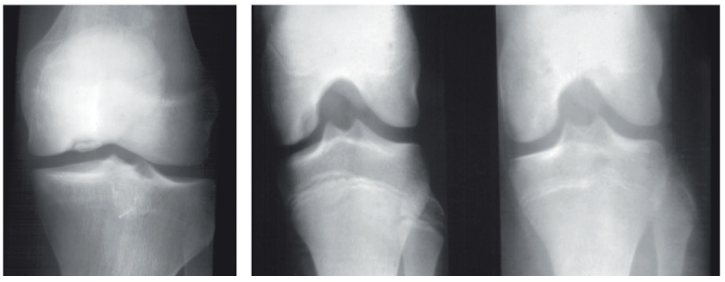
**Stage 2.** Fragment demarcated by separation of articular cartilage.




**Stage 3.** Fragment of cartilage and bone completely separated as loose body. This often migrates to medial or lateral.

Circles indicate arthroscopic view.

Stage 2 lesion



Tunnel view radiographs of small OCD lesion involving medial femoral condyle treated with activity modification. Complete healing occurred.

**Figure 10.2.** Osteochondritis dissecans.

cellular and humoral factors for healing contributes to the multilayered organization of articular cartilage and affects its ability to heal with conservative management. The three most common areas in pediatric athletes where OCDs occur are the knee (femur), elbow (capitellum), and ankle (talus).

**Presentation:** Juvenile OCDs (JOCDs) are more common in athletes, and 40%–60% of JOCDs have a preceding history of trauma. Vague symptoms such as recurrent pain, crepitus, decreased range of motion, joint tenderness, and/or joint effusions may be present. More severe or advanced cases may include mechanical symptoms such as catching or locking.

**Physical examination:** Tenderness may be reproduced at the site of the OCD lesion, so the medial femoral condyle, the capitellum, or the talar dome should be palpated as clinically indicated. The presence of a nontraumatic joint effusion in an athlete is highly suspicious for an OCD lesion as is the presence of mechanical symptoms. Gait abnormalities may be observed in knees of OCD patients and include walking with the tibia externally rotated to decrease pressure on the lesion.

**Diagnosis:** Often made based on plain radiographs

- **Knee:** AP, notch or skier's, lateral, and sunrise views should be obtained. Often, the lesion in the posterior aspect of the medial femoral condyle is evident only on the notch or skier's view.
- **Elbow:** AP, lateral, and oblique views should be obtained with consideration of comparison views.
- **Ankle:** AP, lateral, and mortise views are necessary. The anterolateral talar dome and the posteromedial talus are common areas of occurrence in the ankle.
- In general, up to 50% of OCD lesions can be missed on plain radiographs. MRIs substantially improve the ability to image the lesion and also allow for staging of joints with OCDs, with 92% sensitivity and 90% specificity. See Table 10.4 for MRI staging of OCD lesions.

**Treatment:** Varies, depending on the age of the patient, skeletal maturity, and location and size of the lesion. Conservative treatment with rest and decreased mechanical forces is often effective in 50%–91% of stage I–II lesions that are stable in skeletally immature patients. Indications for surgical referral to an orthopedic surgeon include persistent symptoms such as recurrent effusions, instability, chronic pain, ongoing mechanical symptoms, concomitant injury, loose bodies, and skeletally mature patients.

## Spondylolysis

**Overview:** Studies indicate that 10%–30% of adolescent athletes suffer from low back pain. In young athletes, back pain that persists for >3 weeks should be evaluated for the presence of spondylolysis (Fig. 10.3). Spondylolysis can be acute (pars stress fracture) or chronic. Spondylolisthesis refers to subluxation or anterior displacement of a vertebral body. Patients with congenital spondylolysis have radiologic evidence by the age of 5–6 years, but symptoms are often delayed until adolescence with athletes presenting at the age of 12–18 years. Both unilateral and bilateral pars defects are seen. In general, bilateral defects have a higher complication rate, slower healing times, and are associated with a risk of slippage resulting in a concurrent spondylolisthesis.

**Presentation:** Typically, an athlete presents with activity-related back pain that is worse with extension. Neurologic and nighttime symptoms are rare.

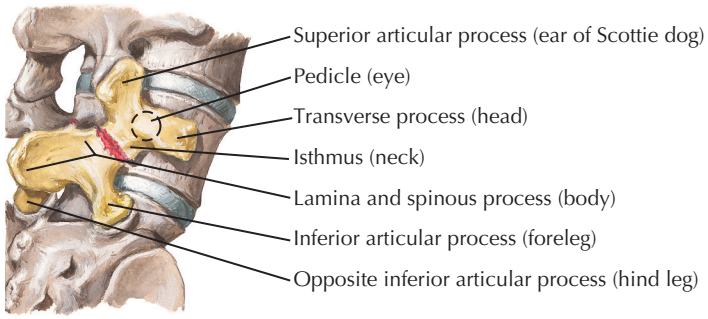
**Physical examination:** There may be tenderness at palpation at the level of the fractured pars. Paraspinal muscle tenderness may be present. A "stork" test is a single-leg balance, spine-extension test that produces back pain when positive.

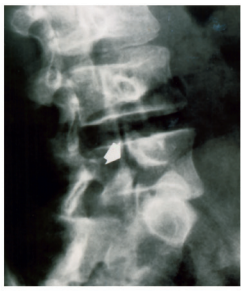
**Diagnosis:** Standard radiographs are AP, lateral, and right and left oblique views. A "Scottie dog" sign is a fracture of the pars seen best on oblique views. The lateral view will demonstrate the presence or absence of a "slip" or spondylolisthesis.

- Radiographs have a low sensitivity for detection of spondylolysis.
- SPECT bone scans are highly sensitive for pars injuries.
- A reverse-angle thin-cut CT scan is best at evaluating healing or sclerosis versus nonunion of pars defects. If symptoms are localized, CT may be useful in diagnosis.

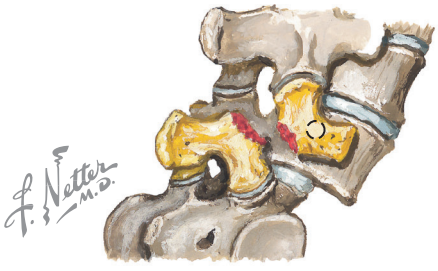
**TABLE 10.4 ANDERSON'S MRI STAGING OF OCD LESIONS**

| Stage | Findings on MRI |
|---|---|
| I | Thickening of articular cartilage and low signal changes |
| II | Articular cartilage is disrupted with low signal rim behind the fragment |
| III | Articular cartilage is widened with high signal changes behind the fragment |
| IV | Loose body: displaced intra-articular fragment |



**Spondylolysis without spondylolisthesis** Posterolateral view demonstrates formation of radiographic Scottie dog. On lateral radiograph, dog appears to be wearing a collar.



**Isthmic-type spondylolisthesis** Anterior luxation of L5 on sacrum due to fracture of isthmus. Note that gap is wider and dog appears decapitated.

**Figure 10.3.** Spondylolysis and spondylolisthesis.

- MRIs are used with an increasing frequency in the evaluation of both pars defects and disc pathology with relatively good sensitivity and specificity.
- A clinician should use his or her knowledge of the clinical situation, including physical examination, RTP concerns, and cost, along with other factors in the decision to utilize the different available imaging modalities.

**Treatment:** Activity modification with decreased sports participation until pain free and guided rehabilitation that emphasizes core strength, posture, and hamstring/hip flexor flexibility. Sports participation is usually restricted for a minimum of 6 weeks. Bracing, although controversial, can be an important part of the treatment for pars defects, particularly if athletes are not pain free with rest from activities. Bracing type is controversial as is the length of time required (12–23 hours/day for 6 weeks to 6 months is the most common):

- Boston brace is the traditional gold standard. It is a rigid lumbosacral orthosis (LSO).
- Semirigid orthoses (e.g., Warm-N-Form) are useful, cheaper, and are increasingly popular due to improved compliance and similar outcomes when compared to use of a Boston brace.
- Soft lumbar orthoses are occasionally useful for less severe unilateral cases and when compliance is poor with the Boston brace or Warm-N-Form.
- Braces are unlikely to significantly limit extension; in fact, efficacy may be achieved through a proprioceptive mechanism.

**Prognosis:** Unilateral pars defects have a better prognosis for healing without long-term complications than do bilateral defects. Nevertheless, there is a relatively low rate of bone healing (50%): 25% cases show fibrous union or partial bone healing and 25% result in nonunions, as observed on follow-up CT scans.

**RTP:** Most athletes return to their previous level of sports participation. Athletes should be pain free. Some can return to participation in a brace after the initial period of rest (usually 6 weeks). High-risk back-extension activities such as gymnastics and weightlifting should not be performed for a minimum of 6 weeks and then only if athletes are pain free during activity. Recurrent symptoms are common. Treatment should include an evaluation of sports biomechanics and workload. Adjustments in amount and frequency of training as well as changes in technique should be made as part of a comprehensive treatment plan.

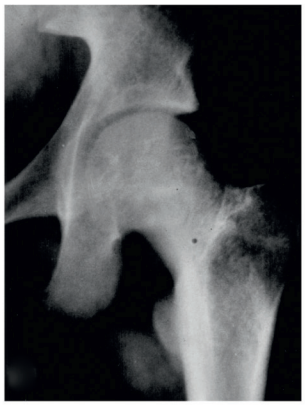
## Hip Disorders in Young Athletes

**Overview:** Hip injuries in young athletes can be among the most serious injuries to occur. Hip trauma may be epiphyseal, apophyseal, or diaphyseal. Moreover, numerous soft tissue injuries are seen, but growth plate injuries are still the most common. Skeletal and vascular growth affects the development of immature bone, appearance of ossification centers, and blood supply to the femoral head.

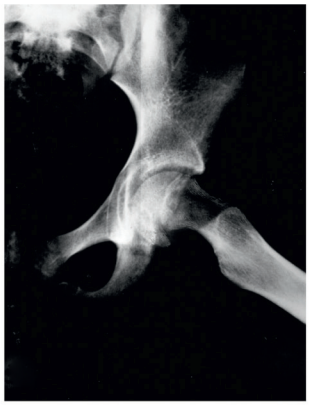
**Presentation:** Acute injuries to the hip are common; they are usually the result of an injury during sports participation and require urgent evaluation. Overuse injuries to the hip may have a more insidious presentation and require a high index of suspicion and a thorough evaluation for accurate diagnosis. Athletes with hip disorders may not always experience hip pain. They may have referred pain to the thigh or knee alone, with or without stiffness, muscle weakness, and/or a limp.

- **Slipped capital femoral epiphysis (SCFE)** usually presents in an overweight or obese, rapidly growing male, aged 10–14 years. It is bilateral in 50% of cases (Fig. 10.4).

Best diagnostic in physical examination. With patient supine, as thigh is flexed it rolls into external rotation and abduction.



Slipped capital femoral epiphysis not readily apparent on anteroposterior radiograph because slip is usually posterior.



Frog-leg radiograph, which demonstrates slipped epiphysis more clearly, always indicated when disorder is suspected.

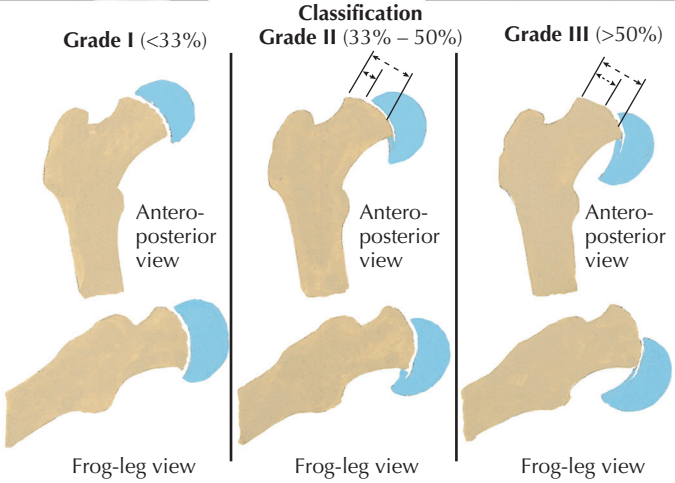
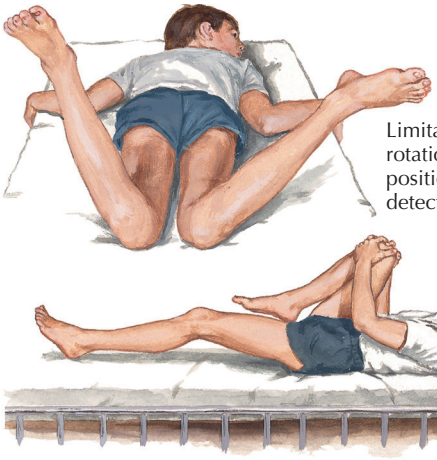



**Figure 10.4.** Slipped capital femoral epiphysis.



Limitation of internal rotation of left hip. Hip rotation best assessed with patient in prone position because any restriction can be detected and measured easily.

**Thomas' sign:** Hip flexion contracture determined with patient supine. Unaffected hip flexed only until lumbar spine is flat against examining table. Affected hip cannot be fully extended, and angle of flexion is recorded. 15° flexion contracture of hip is typical of Legg-Calvé-Perthes disease.

**Trendelenburg's test**
**Left:** Patient demonstrates negative Trendelenburg's test of normal right hip.
**Right:** Positive test of involved left hip. When weight is on affected side, normal hip drops, indicating weakness of left gluteus medius muscle. Trunk shifts left as patient attempts to decrease biomechanical stresses across involved hip and thereby maintain balance.

**Figure 10.5.** Physical examination in Legg–Calvé–Perthes disease.

- **Legg–Calvé–Perthes disease (LCP)** is a condition of unknown etiology that appears as avascular necrosis of the femoral head. It is commonly seen in children age 4 to 10 years, with a 4:1 male predilection and a 20% occurrence of bilateral cases (Fig. 10.5).

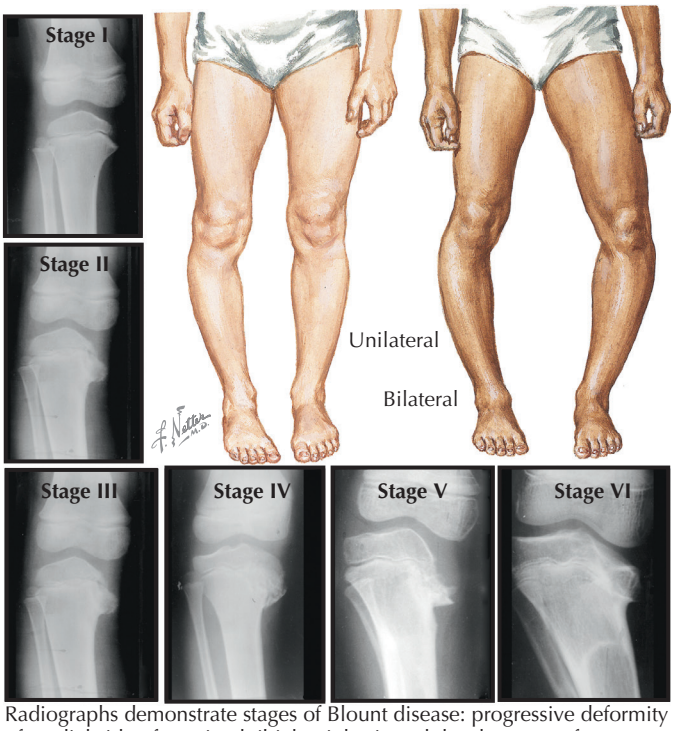
**Physical examination:** A patient with SCFE or LCP may present with hip pain or just knee/thigh pain. A limp is often noted. A Trendelenburg gait is characteristic (see Fig. 10.5). Chronic cases may present with a limp or stiffness in the absence of pain. There may or may not be tenderness on palpation over the anterior hip joint. More typically, particularly in an SCFE case, when the hip is flexed, the leg rides into external rotation with considerable limitation of internal rotation.

**Diagnosis:** Radiographs should always include a lateral and frog-leg anterior-posterior (AP) pelvis view. Comparison of both sides allows easier identification of pediatric hip pathology. In SCFE, subtle blurring or widening of the femoral physis is often noted in early cases. In more severe cases, there is an obvious deformity of the femoral neck. In Perthes cases, radiographs reveal a characteristic cessation in the growth of the bony epiphysis accompanied by sclerosis, fragmentation, collapse, and/or advanced avascular necrosis. LCP is staged based on radiographic appearance as well as the percent of femoral head involvement and alignment. Once the diagnosis is made, both SCFE and LCP patients should be immediately referred to a pediatric orthopedist for definitive care.

**Treatment:** Definitive treatment for SCFE is internal fixation or pinning of the epiphysis to stabilize it and prevent further slippage. Realignment is not routinely attempted because of the risk of disruption of vasculature to the femoral head. The **treatment of LCP** is complicated. Initial treatment involves rest with crutches and physical therapy to preserve range of motion and joint stability. For advanced cases, various surgical containment procedures may be performed by a pediatric orthopedist.

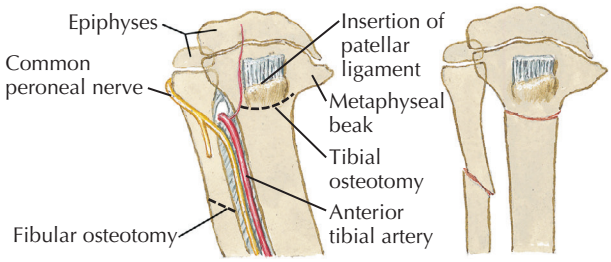
## Blount Disease (Tibia Vara)

**Overview:** Tibia vara is a condition that involves a growth disturbance of the medial tibial physis (Fig. 10.6). It is most commonly seen in obese boys aged 10–14 years but can been seen in children as young as 2–3 years old and is more common in African Americans. Blount disease should be included in the differential diagnosis for any child presenting with a limp or bowlegs (genu varum) at any age.




Radiographs demonstrate stages of Blount disease: progressive deformity of medial side of proximal tibial epiphysis and development of metaphyseal beak

**Schema of U-shaped osteotomy**




Neurovascular compromise, anterior compartment syndrome, undercorrection or overcorrection, and failure to correct internal tibial torsion are potential problems.

**Figure 10.6.** Blount disease.

**Presentation:** Typical presentation is of an obese child with a chronic progressive limp and either a unilateral or bilateral bowing of the tibia. Knee pain may be present and often localizes to the medial tibia.

**Physical examination:** Findings often include a leg length discrepancy, tenderness at the medial tibial physis, and tibial torsion associated with tibia vara. Instability to valgus stress testing may be present. Gait is usually antalgic and is notable with a varus thrust.

**Diagnosis:** Radiographs of the knee often reveal tibia vara and demonstrate growth arrest of the medial tibial physis with acute medial angulation of the proximal tibia. A standing bilateral lower extremity AP view showing pelvis to foot is the standard orthopedic view, but it is often not obtained until after diagnosis is made based on standard knee radiographs.

**Treatment:** Patients with Blount disease should be referred to a pediatric orthopedic surgeon for definitive care. Surgical treatment include an osteotomy to realign the tibia into slight valgus, thus eliminating the leg length discrepancy and bowing. Other options include a stapling procedure of the lateral tibial physis to stop growth throughout the proximal tibia; this can result in a considerable leg length discrepancy compared with the contralateral side and may lead to further complications. Moreover, weight management as part of the comprehensive treatment for obesity must be emphasized in such patients.

## Tarsal Coalition

**Overview:** Tarsal coalition involves a congenital bony or fibrocartilaginous fusion of tarsal bones (Fig. 10.7). It is seen in 1%–3% of the general population and may be autosomal dominant with variable penetrance. Bilateral occurrence is seen in 50%–60% of cases. Two types are commonly seen: calcaneonavicular and talocalcaneal.

**Presentation:** Symptoms usually develop in the second decade of life as increased bony ossification occurs. Progressive stiffness and limping may be seen. Pain is initially activity related.

**Physical examination:** A rigid flatfoot is seen with hindfoot valgus, along with decreased subtalar joint motion and limited inversion. Tenderness may be present along the lateral aspect of the subtalar joint and/or sinus tarsi.

**Diagnosis:** Bilateral weight-bearing AP, lateral, oblique, and Harris radiographs of the feet should be obtained, however, these are often nondiagnostic, particularly in the setting of a fibrocartilaginous coalition. Calcaneonavicular tarsal coalition is more likely to be seen on plain films. Secondary findings may include beaking of the talar neck and widening of the talonavicular joint. A bilateral noncontrast CT of the feet is the gold standard.

**Treatment:** A referral to a pediatric orthopedic surgeon is recommended. Initial treatment is conservative and includes observation, physical therapy, gait training, and orthotics. Immobilization may be performed for 4–6 weeks for a more painful condition. Definitive treatment may include surgical excision to decrease pain, improve mobility, and limit progressive degenerative changes. Surgical intervention is most effective if performed before the age of 14 years.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

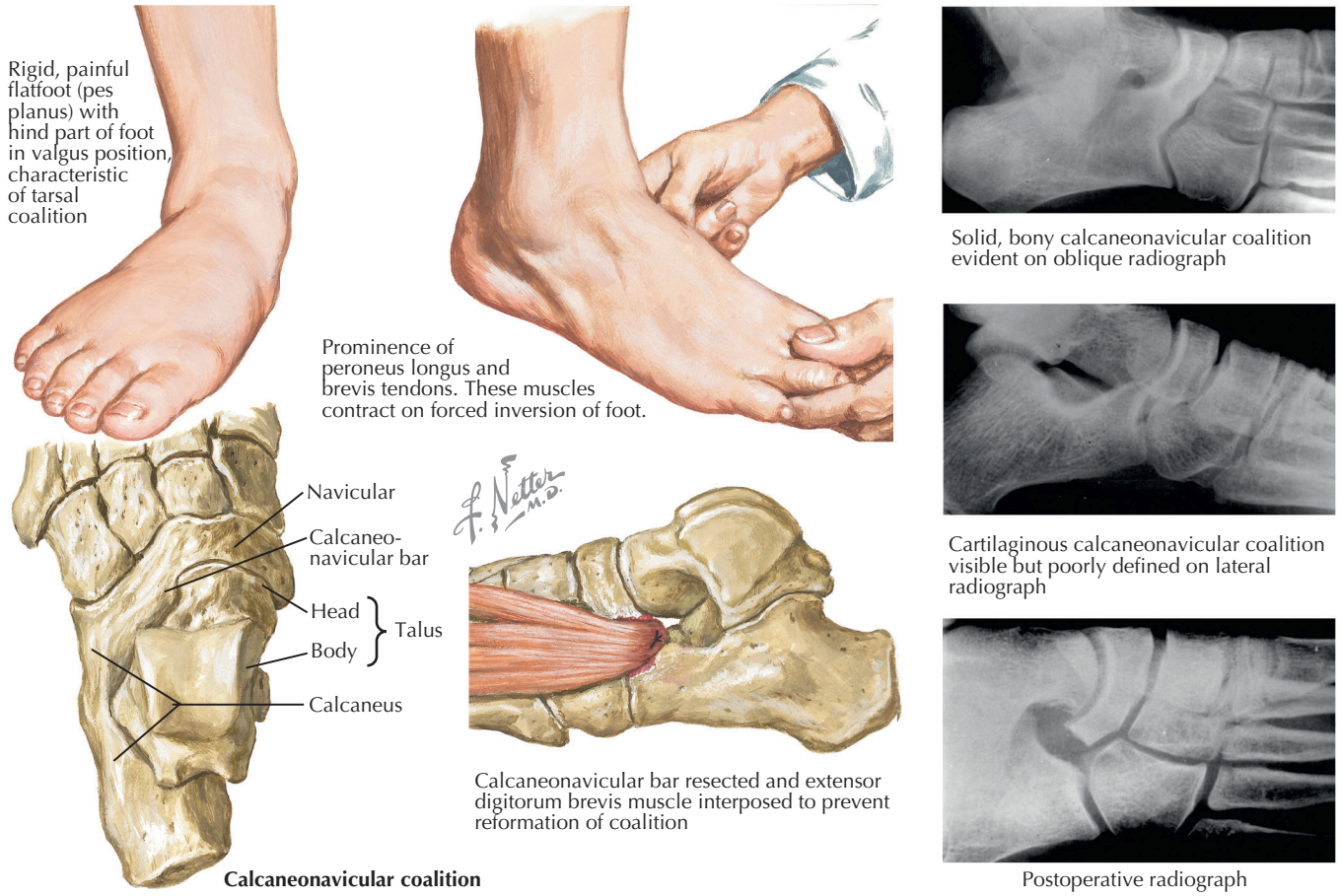



**Figure 10.7.** Tarsal coalition.

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# THE HIGH SCHOOL ATHLETE: SETTING UP A HIGH SCHOOL SPORTS MEDICINE PROGRAM

11

Shayne D. Fehr • Kevin D. Walter • Stephen G. Rice

## GENERAL PRINCIPLES

### Athletic Healthcare in High-Level Collegiate and Professional Sports

- At the very highest levels of sports, organizations are much like corporations; they function to increase success of their shareholders by providing a product (a winning team) directly linked to profitability.
- While there are ethical concerns with this model, it provides an environment of highly established care because the health of an athlete is directly linked to a team's success.
- Below are the characteristics of such models:
  - Remarkable financial resources
  - Well-defined roles and responsibilities
  - Risk management and loss control
  - Optimization of athletes' health for on-field performance
  - High organizational control and attention to detail
  - Professional personnel secured in adequate quantity
    - Certified athletic trainers (ATCs)
    - Qualified team physicians (sports medicine fellowship-trained, primary care physicians, and orthopedists)
    - Other allied healthcare professionals including certified strength and conditioning coaches, nutritionists, psychologists, optometrists, dentists, exercise physiologists, and physical therapists
  - Compliance with *Team Physician Consensus Statement* (see Recommended Readings)
  - Policies delineated and enforced routinely

### Differences in Athletic Healthcare for the High School Athlete

- The effect of high-profile professional and collegiate sports has trickled down into secondary schools, with many school administrators, coaches, and parents looking for the same type of on-demand medical care.
- However, most schools have the following concerns:
  - Lack of financial resources
  - Lack of leadership—just maintain status quo
    - Turnover of personnel (school board, superintendent, principal, athletic director, and coaches)
    - Not a priority—too many other issues
  - Lack of medical resources
    - No ATC or physician
    - Inadequate medical knowledge among coaches and athletic directors
    - Communication with medical community often poor
    - Results in incorrect or delayed care
  - Lack of policies/standards
    - No overall single system of care—each coach does his/her own thing
    - Assumed to be met by minimal standards and effort by external personnel or agencies, such as state-required preparticipation physical examinations, volunteer team physicians, or presence of ambulance at home varsity football games

### Solution: Goals and Requirements

- Goals: appropriate healthcare for athletes and minimal liability through a risk management (loss control) policy
- Requirements: knowledge, organization, and commitment toward detailed planning

### Approach to Optimal High School Athletic Healthcare

#### Key Elements

Four key elements of the approach: family, school, medical community, and ATCs

FAMILY INVOLVEMENT

- Most high school athletes are minors and are dependent on their parent(s) or guardian(s)
- School, ATC, and physician must focus on communication with parents/guardians of injured athletes
- Parents/guardians:
  - Know the athlete's medical history
  - Are usually more concerned with their children's health and academics than sport
  - Are important resources regarding psychosocial dynamics
- Financial concerns may prevent seeking appropriate care

SCHOOL COMMITMENT

- School should assume responsibility for operating safe programs.
  - Obligations toward students and their families and commitment to meeting them; solutions must be internal as well as external
  - Qualifications and backgrounds of athletic directors and coaches
  - Should work as a unit, operating a single interscholastic athletic program and single athletic healthcare program
    - Institution of policies, guidelines, and procedures for daily use
    - Record-keeping system
    - Emergency action planning and first-aid/CPR training
- Seek assistance from the medical community for all sports: preparticipation physical examinations as well as preseason fitness screening, weekly school visits, event coverage, and therapies and treatments
  - Know what is desired from physicians, physical therapists, and clinicians
  - Designate team physician(s)
- Hire National Athletic Trainers' Association (NATA)-certified athletic trainers (and licensed in state if available) as they are the most suitable professionals to coordinate and operate the athletic healthcare program
  - Cannot be present simultaneously at all athletic venues
    - Use of student athletic trainers and educated coaches for assistance
  - Requires a support system
    - Policies and procedures, including record-keeping, accountability, and quality assurance systems

- Requires wireless communication and golf cart to meet obligations of daily coverage and emergency response during fall and spring seasons
- Should insist on medical supervision and quality assurance system
- Should have adequate budget
- Reasonable schedule demands
  - Known high turnover rate for ATCs due to heavy workload
  - Consider second ATC in large high schools

MEDICAL INVOLVEMENT (TEAM PHYSICIANS)

- A written contract is best.
  - Delineates responsibilities and expectations
  - Helps ensure that the school has given careful thought to its obligations
  - Good communication leads to good working relationships
- Monetary compensation—yes or no?
  - If compensated, may nullify "Good Samaritan" immunity
  - Amount offered is frequently meager compared to earnings in office
    - True value of assistance provided?
- Responsibilities (enumerated in Team Physician Consensus Statement)
- Jurisdiction: Are your decisions final?
- Medicolegal (liability) concerns
  - Good Samaritan Law immunity may cover team physician in certain states
    - Team physician is not really a Good Samaritan under strict definition—"someone without obligation who steps forward to render emergency care"
    - Has clearly defined responsibilities toward athletes, school, and athletic program
    - Event coverage is evidence of that responsibility
    - May be covered by "good intent, no compensation" concern
    - Good Samaritan immunity extends only to "emergency care" rendered during event coverage; protection does not extend to preparticipation physical examinations, weekly injury clinics at school, and return-to-play clearance examinations
  - Potential responsibility and liability for ATC's actions
    - Need to clarify issue with school district
    - ATCs generally function "under direct medical supervision of a physician"
    - In states that have not specifically defined the "scope of practice" for ATCs through licensure, certification, or registration, the team physician needs to assess implications and responsibilities of "direct medical supervision." An analogous supervisory situation may be the physician–physician's assistant relationship.
    - Written standing orders for the ATC and emergency action plans (EAPs) are essential requisites for limiting any liability risks
  - Malpractice insurance and liability coverage
    - Incorporated into your personal or clinic policy (already existing or a new rider clause to be added)
    - Through school district insurance policy
- Medical privacy concerns (HIPAA and FERPA)
  - Health Insurance Portability and Accountability Act (HIPAA) of 1996 and Family Education Rights and Privacy Act (FERPA) in 1974 were developed to regulate "protected health information"
  - FERPA regulations prevail within the domain of public schools.
    - FERPA governs school nurses, school physicians, coaches, and ATCs.
  - HIPAA Privacy Rule allows release of medical information without authorization for "treatment, consulting with other providers, referring the patient to other providers, and notifying a patient's family"
    - Athletes who consult at medical facilities outside of school will most likely fall under purview of HIPAA.
  - Eligibility decisions regarding "cleared" or "not cleared" on preparticipation physical examinations can be provided to coaches and school administrators (without inclusion of other medical information) without signed consent.
  - For group preparticipation physical examinations conducted at the school, must ensure confidential storage of forms, with information pertaining to restrictions shared only with those who have "need to know"
    - Need to know—always includes ATC, school nurse, and school physician (team physician)
      - Variable for coach and administration depending on circumstances because the athlete's well-being may require one to have an understanding of his/her limitations or signs/symptoms
  - Coaches and administrators, as well as school nurses and ATCs, must be made aware of FERPA and HIPAA regulations and constraints regarding privacy of healthcare information.
- Degree of involvement
  - Set overall medical policy with athletic director and ATC
  - Strongly consider forming a medical advisory board with school district
  - Provide medical advice to the interscholastic athletic program
  - Provide medical coverage at games. Ideal goal—to see every team member at least once during the season—may require division of coverage among several physicians, possibly by sport or on a rotational basis
    - Football team: home varsity coverage (mandatory); away varsity and home junior varsity coverage (recommended)
    - Wrestling team: preseason weight class recommendations, midseason weight certification, assessment of skin for communicable diseases, and coverage of home matches (recommended)
    - Coverage of all tournaments at home school
    - Soccer team (boys and girls): coverage of events as schedule permits
    - Basketball team (boys and girls): coverage of events as schedule permits
    - Other sports as schedule permits
  - Develop an emergency contact plan and emergency action plan, including use of automated external defibrillator (AED) for sudden cardiac arrest
  - Develop a concussion action plan, including return to sports and academics
  - Conduct preparticipation physical examinations
  - Visit school/athletes regularly
  - Educate coaches and ATCs
  - Provide support for ATC's authority
    - Medicolegal supervisor of the ATC
    - Assess knowledge, skills, and experience of the ATC and mutually develop an appropriate set of standing orders with cumulative working relationship and legal scope of practice for ATCs in their jurisdiction
    - Role in creating a job description
    - Role in interviewing and hiring
    - Role in job evaluation
    - Role in quality assurance of care rendered by ATC
      - Frequent and regular communication
      - Chart review and case studies as needed
- Role of a team physician in school without an ATC
  - Understand history and culture of the school
  - Assess strengths and weaknesses of how athletic care is and was provided

- Greater challenge to meet responsibilities
- Possible institution of athletic healthcare system (AHCS; see the following section)
- Encourage school to recognize need for ATC
- Role of a new team physician in a school with an established ATC
  - The team physician should understand the methods and culture of the existing system.
  - ATC may welcome an active, involved, "hands-on" team physician or prefer a more distant consultant model if he or she is comfortable as the central focus of the AHCS and confident of his/her abilities and skills.
  - Team physician should develop appropriate relationship with ATC.

ATHLETIC TRAINER

- Hiring considerations and working conditions (team physician should help school in the hiring process)
- Scenario—getting a job is not easy (but it is getting easier!). ATC submits resume and NATA pamphlets as to why certified ATC is necessary.
- ATC may be perceived as a salesman.
  - Identifying "a problem I didn't know existed"
  - Offering solution to the problem
- Funding is a factor—ATC is usually a low-paying, entry-level job
- If ATC is hired, under what conditions?
- Full- or part-time ATC?
- Teacher and ATC? How many classes?
- How many working hours per week, including games?
- How many working days per year?
  - ATC needs more days than regular school calendar.
  - If general contract calls for same number of days as teachers, it must take into account preseason football days, weekends, and holiday tournament days.
    - Possible solution: part-time substitute ATC, who works 1 day per week throughout school year (40 days); this schedule decreases the risk of burnout for the full-time ATC (from not having time off) and allows ATC to work same number of days as teachers
    - Part-time ATC can service several schools each week, if more work is desired.
- Medical backup and supervision
  - Head team physician should be specifically recognized as the medical supervisor of the ATC.
  - Degree and frequency of communication should be clearly established.
  - Whose decision is final regarding return to play?
- Adequate budget for equipment and supplies, professional fees, books, and continuing education
- Quality of training room
- Written job description
- Job performance (accountability and quality assurance)
  - Evaluated by team physician and others (e.g., athletic director, school nurse, coaches, principal, and athletes)
- Potential for career advancement

## ATHLETIC HEALTHCARE SYSTEM

### Generic Model System

- The AHCS was initially developed by adapting college and professional sports medicine programs to high school level.
- This can be installed at any school, large or small sized, and at any school location, rural or urban.
- The AHCS should be tailored to each school and adhere to accepted standards of practice.
  - Effectiveness of the AHCS is greatly improved with an ATC and a sports medicine-trained team physician.

### Model System

- The AHCS was developed under strict guidelines of the US Department of Education from 1978 to 1982 and validated in 1982, 1987, and 1995.
- The AHCS was nationally disseminated in the 1980s and 1990s by grants through the National Diffusion Network.
  - This consortium was well known by schools with a track record for proven, cost-effective programs that worked.
- In 2002, NATA developed a consensus statement with recommendations and guidelines for secondary school sports.
  - In 2004, the consensus statement was augmented by an updated, science-based document.

### Key Elements of the Athletic Healthcare System

#### 1) Assessment

- Complete evaluation of existing programs
- Standards for care delineated
- Areas of assessment
  - Identify AHCS team members that have a diverse skill set
  - Athletic facilities—safe practice and competition venues
  - Athletic equipment—selection, fit, and maintenance
  - Emergency action plan—development and implementation
  - Treatment facilities/athletic training room
  - Provision of athletic healthcare services
  - Documentation strategies and injury surveillance
- Self-assessment initially followed by external evaluation
  - Concept and methodology similar to assessments by the Joint Commission on Accreditation of Health Organizations (JCAHO) and educational assessments
  - Formal written report issued
  - Develop action plan based on weaknesses and deficiencies
  - Assessment frequency—every 3–5 years

#### 2) Education

COACHES

- Know rules and expectations for coach education in your state or league
- Coaching education and standards provided by the National Federation of State High School Associations (NFHS), the National Association for Sport and Physical Education, and various other groups
  - Basic life support (BLS)
  - First aid, health, and safety
  - Concussion education
  - Nutrition and hydration
  - Mental health and sports psychology
  - Behavior and engagement of athletes and parents
  - Injury prevention and injury management
- Education on the culture of sport
  - Encourage an environment of honesty in reporting injuries without repercussions
  - Recognize injury, treat and completely rehabilitate to allow pain-free participation
    - No longer fall back on "no pain, no gain" philosophy
- Education on how to most effectively partner with ATCs and team physicians
  - Share responsibilities
  - Defer to knowledge of objective healthcare providers
  - Work with AHCS team to install injury reduction programs

HIGH SCHOOL STUDENT ATHLETIC TRAINERS

- Assist coaches and ATCs in daily tasks
- Courses in summer and/or during school year
- May be funded through vocational education monies
- Provides career opportunities in the healthcare field

ATHLETIC DIRECTORS AND OTHER ADMINISTRATIVE PERSONNEL
- Athletic administration courses through national associations like the NFHS and the National Interscholastic Athletic Administrators Association
- Regional and state conferences
  - Focus on organizational management and implementation of a safe athletic program
  - Safety and liability issues
  - Strategies to interact with coaches, sports medicine team, and state associations

### 3) Athletic Training Room/Treatment Facilities
- Recommendations
  - Should have privacy for evaluation of student-athletes
  - Should be accessible for both male and female athletes
  - Should not be a conditioning center or a weight room
  - Often used as a rehabilitation room
  - Ensure appropriate disposal of biohazard waste
  - Have a plan for equipment and supply inventory, stocking and storage
  - Location: ideally close to locker rooms and athletic fields
  - Adequate plumbing and drainage for ice machine and tubs
  - Ensure appropriate heating, ventilation, and lighting
  - Ensure layout of room allows smooth flow of traffic
  - Educational resources for student-athletes: books, posters, and manuals
  - Ensure safety and security is assessed

### 4) Standard Procedures
PREPARTICIPATION EVALUATION
- Coordinate with medical community, ATC, and coaches
  - See preparticipation evaluation chapter for additional details
  - Identify athletes with medical conditions (e.g., diabetes, asthma, seizures, or anaphylaxis) and develop emergency and preventative management plans

PRESEASON SCREENING
- Create off-season fitness plan for athletes of every sport
  - Develop injury and illness prevention plans
  - Provide sound nutritional counseling and education
- Physical assessment
  - Flexibility
  - Strength and endurance
  - Aerobic capacity
  - Body composition
- Education and culture
  - Create an environment for appropriate reporting of injuries
  - Develop "mental toughness"—psychological and emotional makeup
  - Educate on playing within rules
  - Emphasize sportsmanship
  - Educate on appropriate nutrition and hydration
    - Discussion on safe weight gain and weight loss
    - Educate on avoiding supplements and performance-enhancing substances
  - Review common adolescent high-risk behaviors (e.g., substance abuse, use of seat belts and bike helmets, internet and social media safety, or bullying)

TREATMENT PROTOCOLS AND GUIDELINES
- Should be shared with athletes, coaches, and parents
- Should be created in conjunction with the team physician
- Identify local experts for referral of injuries, psychosocial pathology (e.g., eating disorders or depression), and medical illness
- PRICES
  - **P**rotection—brace/splint use
  - **R**est
  - **I**ce
  - **C**ompression
  - **E**levation
  - **S**upport—crutch use
- Use of OTC medications—if allowed
- Educational handouts for athletes and families on injuries
- Steps of rehabilitation
  - Control pain and swelling
  - Restore range of motion
  - Restore strength
  - Restore stability
    - Taping, bracing, and rehabilitation exercises
  - Restore general functioning
    - Aerobic capacity
    - Core stability
    - Neuromuscular function
    - Balance
  - Restore sport-specific functioning

RETURN-TO-PLAY POLICY
- Create in conjunction with the AHCS team, particularly the team physician
  - To reduce the risk of inappropriate clearance by an external physician, establish which provider has the final say on medical clearance—should ideally be the team physician
- Should be shared with parents, coaches, and athletes during preseason meetings
  - Set expectations for when an injured athlete can resume play
- Concussion-specific
  - Should comply with state legislation
    - Written medical clearance
  - Follow a step-wise return-to-play progression
    - Concussion in Sport Group, Zurich Guidelines, 2014
  - Before beginning the program, an injured athlete should have fully resumed academics and must be asymptomatic and off the medications used for symptomatic treatment.
- Musculoskeletal injury
  - Little to no swelling
  - Little to no tenderness on palpation
  - Full, pain-free range of motion
  - Full, pain-free strength through range of motion
  - Pain-free joint stability—through rehabilitation, taping, bracing, or surgery
  - Full, pain-free general functional activities
  - Full, pain-free or minimal discomfort during sport-specific activities
- Medical disorders/issues
  - Should have written medical clearance delineating activities allowed and any follow-up care or considerations necessary

COMMUNICATION
- List of key people and contact information disseminated among the AHCS
  - Should include hospitals, clinics, and physicians in the region
- All AHCS personnel should possess individual cell phones in case of emergency.
  - If cell phones do not have reception, alternative modes of communication should be established (e.g., walkie talkies).
- Information cards for every athlete
  - Medical conditions
  - Emergency contacts

### 5) Emergency Preparedness
EMERGENCY ACTION PLAN
- Emergency information cards for every athlete
- Written emergency action plan should be designed for every practice and competition location
  - Annual practice at every location with staff and the team
  - Attention to location of AEDs
  - Checklist for first-aid kit

- Identify the procedure for alerting emergency medical services (EMS)/911
  - Map for route to athletic facility and fields
  - Sports medicine staff can call ahead to inform the hospital facility
- Identify procedures for transfer of care from the AHCS to the EMS team
- Ensure parents, guardians, and caregivers of the injured athlete are notified

**TABLE 11.1 INJURY RATES BY SPORT AND TYPE OF EXPOSURE: HIGH SCHOOL SPORTS-RELATED INJURY SURVEILLANCE STUDY, US, 2014-15 SCHOOL YEAR***

| | #Injuries | #Exposures | Injury Rate (per 1,000 AEs) |
|---|---|---|---|
| **Overall Total** | 9,273 | 5,165,857 | 1.80 |
| Competition | 4,891 | 1,285,926 | 3.80 |
| Practice | 4,353 | 3,822,140 | 1.14 |
| Performance | 29 | 57,791 | 0.50 |
| **Boys' Football Total** | 3,620 | 919,659 | 3.94 |
| Competition | 1,957 | 157,332 | 12.44 |
| Practice | 1,663 | 762,327 | 2.18 |
| **Boys' Soccer Total** | 575 | 330,072 | 1.74 |
| Competition | 376 | 101,172 | 3.72 |
| Practice | 199 | 228,900 | 0.87 |
| **Girls' Soccer Total** | 832 | 308,502 | 2.70 |
| Competition | 582 | 98,288 | 5.92 |
| Practice | 250 | 210,214 | 1.19 |
| **Girls' Volleyball Total** | 372 | 292,543 | 1.27 |
| Competition | 164 | 97,074 | 1.69 |
| Practice | 208 | 195,469 | 1.06 |
| **Boys' Basketball Total** | 551 | 412,372 | 1.34 |
| Competition | 297 | 128,053 | 2.32 |
| Practice | 254 | 284,319 | 0.89 |
| **Girls' Basketball Total** | 576 | 312,827 | 1.84 |
| Competition | 347 | 97,541 | 3.56 |
| Practice | 229 | 215,286 | 1.06 |
| **Boy's Wrestling Total** | 590 | 264,299 | 2.23 |
| Competition | 244 | 62,792 | 3.89 |
| Practice | 346 | 201,507 | 1.72 |
| **Boys' Baseball Total** | 304 | 286,729 | 1.06 |
| Competition | 188 | 101,791 | 1.85 |
| Practice | 116 | 184,938 | 0.63 |
| **Girls' Softball Total** | 240 | 208,904 | 1.15 |
| Competition | 134 | 73,033 | 1.83 |
| Practice | 106 | 135,871 | 0.78 |
| **Girls Field Hockey Total** | 162 | 82,933 | 1.95 |
| Competition | 78 | 26,609 | 2.93 |
| Practice | 84 | 56,324 | 1.49 |
| **Boys' Ice Hockey Total** | 87 | 52,034 | 1.67 |
| Competition | 69 | 18,269 | 3.78 |
| Practice | 18 | 33,765 | 0.53 |
| **Boys' Lacrosse Total** | 250 | 137,016 | 1.82 |
| Competition | 157 | 42,448 | 3.70 |
| Practice | 93 | 94,568 | 0.98 |
| **Girls' Lacrosse Total** | 176 | 101,508 | 1.73 |
| Competition | 85 | 31,344 | 2.71 |
| Practice | 91 | 70,164 | 1.30 |
| **Boys' Swimming Total** | 19 | 103,249 | 0.18 |
| Competition | 2 | 18,801 | 0.11 |
| Practice | 17 | 84,448 | 0.20 |
| **Girls' Swimming Total** | 28 | 115,160 | 0.24 |
| Competition | 6 | 19,880 | 0.30 |
| Practice | 22 | 95,280 | 0.23 |
| **Boy's Track Total** | 196 | 290,886 | 0.67 |
| Competition | 69 | 59,974 | 1.15 |
| Practice | 127 | 230,912 | 0.55 |
| **Girls' Track Total** | 219 | 243,824 | 0.90 |
| Competition | 60 | 49,621 | 1.21 |
| Practice | 159 | 194,203 | 0.82 |
| **Cheerleading Total** | 223 | 297,160 | 0.75 |
| Competition | 16 | 18,064 | 0.89 |
| Practice | 178 | 221,305 | 0.80 |
| Performance | 29 | 57,791 | 0.50 |
| **Boys' Cross Country Total** | 87 | 148,784 | 0.58 |
| Competition | 14 | 24,000 | 0.58 |
| Practice | 73 | 124,784 | 0.59 |
| **Girls' Cross Country Total** | 127 | 131,033 | 0.97 |
| Competition | 26 | 21,276 | 1.22 |
| Practice | 101 | 109,757 | 0.92 |
| **Boys' Tennis Total** | 12 | 58,997 | 0.20 |
| Competition | 8 | 18,774 | 0.43 |
| Practice | 4 | 40,223 | 0.10 |
| **Girls' Tennis Total** | 27 | 67,366 | 0.40 |
| Competition | 12 | 19,790 | 0.61 |
| Practice | 15 | 47,576 | 0.32 |

*Only includes injuries resulting in time loss of ≥1 days.
Data from National High School Sports-Related Injury Surveillance Study, available at: http://www.ucdenver.edu/academics/colleges/PublicHealth/research/ResearchProjects/piper/projects/RIO/Documents/Convenience%20Report_2014_15.pdf.

PRACTICE AND PLAYING FIELD/FACILITY SAFETY
- Safety inspections at the start of the year and the season
- Daily inspection of playing surface and surroundings

EVALUATION PROCEDURE FOR ACUTE "ON-THE-FIELD" INJURIES
- Triage severity of injury to first rule out "worst-case injuries"
  - Life-threatening injuries/unresponsive or unconscious athletes
    - Begin BLS with focus on airway, breathing, and circulation (ABC)
    - Activate EMS
  - Unstable athlete—if left unattended, would the athlete deteriorate?
    - Begin BLS and activate EMS
  - Examples of potentially life-threatening, limb-threatening, or unstable injuries
    - Airway obstruction
    - Acute bleeding (internal or external hemorrhage)
    - Anaphylaxis
    - Cardiovascular collapse
    - Heat illness
    - Neurologic impairment/head injury
    - Severe fractures
    - Dislocations
    - Eye trauma
- Have a plan to address common athletic injuries that may not require activation of EMS
  - Initial triage and determination if it is safe to move the athlete off the playing surface
  - Complete evaluation of injury on the sideline
  - Follow-up injury evaluation to ensure the athlete is not decompensating
  - Communication with coaches regarding the athlete's status
  - Communication with the athlete's parent, guardian, or care giver

### 6) Documentation and Injury Surveillance

- Always document every evaluation and care provided
- Reduces liability
- Allows review for quality improvement and accountability

RECORD KEEPING
- Paper or electronic
- Athlete emergency information card
- Daily report of attendance and injuries
  - Note the absent athletes to assess injuries or illnesses before return to play
- Keep athletic training room treatment log
  - Documentation on all treated athletes should include diagnosis, treatment(s), progress, and limitations.
  - May need to pass on treatment notes of the injured athletes to families or medical care providers
- Maintain a file for correspondence from medical care providers
- Maintain a master list of injured athletes for each team
  - Daily review with coaches to determine full, limited, or no participation

INJURY SURVEILLANCE
- Follow injury trends in a sport from year to year
- NFHS partners with High School Reporting Injuries Online (High School RIO) to compile comprehensive high school injury data (refer to Tables 11.1 and 11.2)
  - Random and convenient sampling at high schools by ATCs

**TABLE 11.2 METHODS OF INJURY EVALUATION AND ASSESSMENT: HIGH SCHOOL SPORTS-RELATED INJURY SURVEILLANCE STUDY, US, 2014-15 SCHOOL YEAR**

| | n | % |
|---|---|---|
| **% of Injuries Evaluated by:*** | | |
| Certified athletic trainer | 8,573 | 92.5% |
| General physician | 2,500 | 27.0% |
| Orthopedic physician | 2,076 | 22.4% |
| Neurologist | 87 | 0.9% |
| Physician's assistant | 86 | 0.9% |
| Chiropractor | 68 | 0.7% |
| Nurse practitioner | 49 | 0.5% |
| Dentist/oral surgeon | 20 | 0.2% |
| Other | 194 | 2.1% |
| **Total** | 9,273 | 100.0% |
| **% of Injuries Assessed by:*** | | |
| Evaluation | 9,005 | 97.1% |
| Plain radiograph | 3,172 | 34.2% |
| MRI | 987 | 10.6% |
| CT scan | 266 | 2.9% |
| Hematologic/laboratory examination | 93 | 1.0% |
| Other | 85 | 0.9% |
| **Total** | 9,273 | 100.0% |

*Multiple responses allowed per injury report.
Data from National High School Sports-Related Injury Surveillance Study, available at: http://www.ucdenver.edu/academics/colleges/PublicHealth/research/ResearchProjects/piper/projects/RIO/Documents/Convenience%20Report_2014_15.pdf.

- School-specific injury surveillance is helpful to determine any increase in injury rates that can provide AHCS and coaches information regarding potential risks
  - Example: communicable skin disease outbreaks in wrestling

## Adoption and Implementation Process

- Goal is quality and outcome/athletic safety improvements
- Identify local resources to maintain excellence and establish "ownership"
- Promote awareness among athletes, parents, coaches, boosters, school board members, school teachers and administrators, and local healthcare providers
- Networking can improve quality
  - Other schools or sports clubs
  - State athletic association
  - NFHS
  - Medical community

### Review of Components

- Identify members of the AHCS team
- Identify roles and responsibilities of each AHCS team member
- Create, review, and update policies and procedures
- Establish and maintain physical space and equipment
- Establish documentation standards and an injury surveillance system

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE FEMALE ATHLETE

# 12

Giselle A. Aerni • Jessica Knapp

## HISTORY OF WOMEN IN SPORTS

- Throughout history, women have participated in sports at much lower rates than men.
- The Modern Olympics was first held in 1896; in the inaugural year, there were no female participants. The year 1900 marked the first female participation, and women constitute 2.2% of athletes in just two of the 95 sporting events held.
- In the United States, in 1971, high school girls constitute just 7.4% of all high school athletes; concurrently, women accounted for 14.6% of Olympic participants.
- Title IX was passed in 1972, which stated that no person in the United States shall, on the basis of sex, be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any educational program or activity receiving federal funding.
- CDC data from 2013 showed that 48.5% of high school girls played one or more sports in the previous year compared with 59.6% of boys, indicating a statistically significant difference.
- A prospective study evaluating activity levels throughout childhood revealed that girls had less active minutes per day than boys across all ages and that overall active minutes decreased with age. By 17 years, only 6% of girls were achieving the recommended 30 minutes of daily activity as opposed to 100% of boys.
- From childhood to adolescence, a similar rate of decline is observed in sports participation levels. Although the rate of decline is similar between boys and girls, girls begin with overall lower levels of sports participation.
- Sociocultural determinants that improve female sports participation include parental support (may take form of verbal support, a rule to participate in sport, or role modeling), higher socioeconomic status, higher rates of sibling sports activity, and higher parental education level.
- Female participation in sports increased when the sport was perceived to be fun, involved friends, and had familial and school support through feedback and role modeling.
- Females with higher intrinsic motivation participate more in sports.

## PROS/CONS OF WOMEN IN SPORTS

- Adolescent participation in sports has short- and long-term risks and benefits over physical activity alone.
- Studies have revealed an overall reduction in all-cause mortality and a significant reduction in cancer rates into adulthood in adolescents who were former athletes.
- In addition, adolescent sports participation correlates with increased bone density in youngsters and in middle-aged women who previously participated in sports in comparison with those who did not.
- High school and collegiate girls who participate in sports every day have a decreased BMI and increased cardiorespiratory fitness compared with female nonathletes.
- Measures of mental health and well-being in terms of self-esteem, depression, and suicidality are all positively affected by sports participation in high school and college athletes. However, no apparent correlation has been observed between decreased suicidal ideation and depression levels and gender, race, or ethnicity.
- Cognitive benefits, academic performance, and education levels have an overwhelming positive response to participation in high school and collegiate athletes.
- The overall risk of victimization in the form of bullying, property violence, sexual abuse, rape, and violent contact is lower among female athletes than among nonathletes. These findings were consistent even with controls for race, mother's education, age, number of relationships, and alcohol consumption.
- Sexual behavior in female athletes is complex, but overall, most studies suggest that there are positive effects from sports participation with lower rates of sexual activity, pregnancy, and sexual risk-taking in high school and collegiate girls who participate in sports than in boys and nonathletes.
- Moreover, high school and collegiate female athletes exhibit lower rates of smoking, marijuana use, steroid use, and other illicit substance use than nonathletes.
- Alcohol use is much higher in the adolescent athlete population regardless of gender or race.

## PHYSIOLOGY

- Girls achieve physiologic maturity quicker than boys, thus achieving earlier peak height velocity (11.5 years [y] vs. 13.5 y) and skeletal maturity (13.5 y vs. 14.3 y). Along with hormonal changes at the time of puberty, there are many physiologic differences between male and female athletes.
- Tables 12.1 and 12.2 enlist the effects of estrogen and progesterone.

### Cardiology

- Female athletes typically have a slightly higher resting HR and a lower systolic BP than male athletes. With exercise, female athletes exhibit a lower absolute change in their systolic BP.
- While males typically have larger hearts at baseline, both males and female athletes demonstrate physiologic cardiac hypertrophy in response to training.
- The mechanism for hypertrophy may be different in females compared with testosterone-driven muscle hypertrophy in males. Females have a higher $Ca^{2+}$/calmodulin-dependent kinase (CaMK) and protein kinase B/glycogen synthase kinase-3-beta (AKT/GSK-3) pathway signaling as well as increased fatty acid metabolism, which may contribute more to female-specific physiologic cardiac hypertrophy.
- Pathologic cardiac hypertrophy and arrhythmias are less common in female athletes and considered secondary to estrogen's protective cardiac effects against harmful remodeling.
- Gender differences in cardiac functioning and contribution to maximal oxygen consumption ($VO_2$ max) may be more related to the larger size of male hearts that have greater stroke volume compared with smaller female hearts.

### Pulmonology

- Beginning at around 2 years of age, male lungs are larger than female lungs even when normalized to height/length.
- Females have smaller lung volumes and lower expiratory flow rates compared with males. In addition, with regard to total lung volumes, females have considerably smaller airways, which can contribute to expiratory flow limitations.

**TABLE 12.1 EFFECTS OF ESTROGEN**

| System | Effect |
|---|---|
| **Cardiovascular** | ↑ in thrombosis<br>↓ total cholesterol<br>↓ low-density lipoprotein (LDL) levels<br>↑ high-density lipoprotein (HDL) levels<br>Vasodilates vascular smooth muscle<br>↑ blood pressure |
| **Endocrine** | ↑ intramuscular and hepatic glycogen storage and update<br>Glycogen sparing (↑ lipid synthesis, ↑ lipolysis in muscle, and ↑ utilization of free fatty acids)<br>↑ insulin resistance and ↓ glucose tolerance<br>Deposition of fat in the breasts, buttocks, and thighs |
| **Bone metabolism** | Facilitates uptake of calcium into bone |
| **Neurologic** | Cognitive function and verbal memory in postmenopausal women |

**TABLE 12.2 EFFECTS OF PROGESTERONE**

| System | Effect |
|---|---|
| **Thermoregulation** | ↑ core body temperature of 0.3°C–0.5°C |
| **Respiratory** | ↑ minute ventilation<br>↑ ventilatory response to hypoxia and hypercapnia |
| **Metabolic** | Fluid retention<br>↑ dependence on fat as a substrate<br>Induce peripheral insulin resistance |

- On approaching maximal exercise, females increase their end expiratory lung volumes back toward resting, hyperinflating their lungs in response to these expiratory flow limitations. Simultaneously, higher end inspiratory lung volumes are required to maintain tidal volume. Ultimately, the intensity of breathing is greater in females, approximately twice as high at maximal exercise.
- The prevalence of arterial oxyhemoglobin desaturation is higher in females than in males owing to smaller lung volumes, smaller airways, lower resting diffusion capacity, and lower maximal expiratory flow rates.
- Moreover, females experience less exercise-induced diaphragmatic fatigue.

## Metabolism, Nutrition, and Hydration

- 17 β-estradiol promotes lipid oxidation during endurance exercise, and female athletes have been shown to have a greater dependence on lipid oxidation during endurance events. In addition, 17 β-estradiol promotes hepatic glycogen sparing.
- This enhanced capacity of lipid oxidation, as well as a greater ability to maintain plasma glucose, may account for the ability of female athletes to outperform male athletes at very high distances.
- Females have a lower leucine oxidation rate at rest and during exercise, which accounts for their lower protein requirements.
- There is some evidence that female athletes do not obtain the same benefits from traditional carbohydrate loading compared with male athletes, and in fact need to ingest a proportionally higher amount of carbohydrates in order to gain the same effects.
- Lower ferritin and total body iron tends to be associated with higher oxidative stress. Female athletes are frequently shown to have higher levels of reactive oxygen metabolites and lower iron stores than their male counterparts.
- Throughout the menstrual cycle, from early follicular phase to luteal phase, total body water in females can increase by as much as 2 L. Females have been shown to have lower urine osmolality than do males at rest, with daily activities and with exercise, most likely owing to differences in arginine vasopressin (AVP) concentrations.
- In a study of endurance cyclists matched for BMI and performance, no differences were observed in the ratings of thirst and thermal sensation or the rate of perceived exertion; however, marked differences were noted in terms of female cyclists having a greater total fluid intake, greater percentage of change in body mass, lower urine specific gravity of urine, and lighter color of urine.
- Female athletes do not sweat as much as male athletes do, but they sweat over a greater proportion of their body. In general, studies have shown that females tend to overconsume water in relation to their body size and metabolic rate.
- A study evaluating the relationship between blood lactate and cortical excitability during exercise revealed that females exhibited a significantly greater improvement in excitability of the primary motor cortex in response to increased lactate levels; this was attributed to the excitatory role of estrogens on the cerebral cortex.

## Muscles/Ligaments/Tendons

- Female athletes have been shown to have a similar composition of muscle fibers and enzymatic activity as their male counterparts do when involved with similar sports and activities; however, female muscle fibers are smaller.
- Female athletes are able to achieve significant strength gains with training, but they cannot achieve the same strength levels or hypertrophy as a similarly sized and trained male athlete owing to the difference in testosterone.
- Before puberty, both boys and girls have similar strength and joint laxity. At puberty, estrogen contributes to increased joint laxity in girls, while testosterone contributes to muscle hypertrophy in boys.
- Certain studies have shown lower collagen synthesis in tendons of combined oral contraceptive users, although not all studies have reported differences in tendon properties across the menstrual cycle.
- Some of the increased injury risk seen in female athletes after the age of puberty is attributed to lax joints and strength imbalance caused by these hormonal changes.

## Gastrointestinal Symptoms

- The incidence of gastrointestinal symptoms and complaints is higher in female endurance athletes.
- In addition, studies have revealed a higher incidence of gastrointestinal ischemia in female athletes than in male athletes; however, the underlying pathophysiology remains unknown.

# FEMALE ATHLETE TRIAD

- The female athlete triad is a spectrum of three components: low energy availability (EA), menstrual dysfunction, and low bone mineral density (BMD). Female athletes may have one or more of these components. Each component is on a spectrum of worsening intensity, ending with low EA with or without an

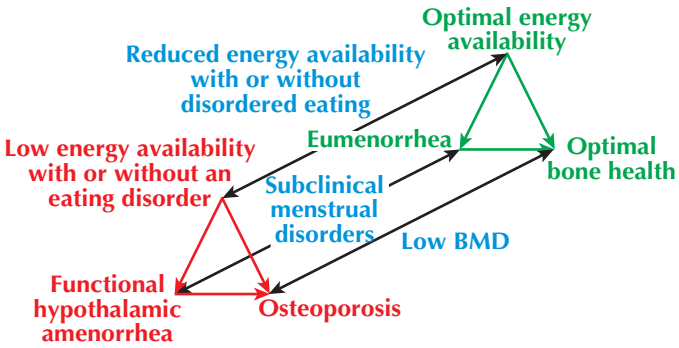



**Figure 12.1.** Female athlete triad continuum. (From Nattiv A, Loucks AB, Manore MM, et al. American College of Sports Medicine position stand. The female athlete triad. *Med Sci Sports Exerc*. 2007;39[10]: 1867-1882.)

### BOX 12.1 HEALTH CONSEQUENCES OF THE FEMALE ATHLETE TRIAD

- Fatigue
- Decreased recovery time
- Decreased training response
- Impaired performance
- Increased risk for heat illness
- Impaired endothelial-dependent arterial vasodilation
- GI changes
- Nutrient deficiencies
- Elevated LDL
- Vaginal dryness
- Impaired skeletal muscle oxidative metabolism
- Cardiovascular consequences
- Increased risk osteoporosis and fractures
- Reproductive dysfunction
- 2–4 times increase in stress fractures

eating disorder, functional hypothalamic amenorrhea, and osteoporosis (see Figure 12.1).

- Low EA can cause disturbances in menstrual hormone levels, which can negatively affect BMD. In addition, low EA can independently have negative effects on BMD.
- Females with female athlete triad are at a risk of short- and long-term health consequences (see Box 12.1).

## Energy Availability (EA)

- Low EA occurs when daily caloric intake does not meet the caloric expenditure needs of a female athlete. EA is the energy available for daily physiologic functioning after the body has used energy for exercise.
- Reduction in total EA results in adaptations over time to conserve energy. Chronic reduction in EA can lead to suppression of metabolic and menstrual hormones.
- In studies controlling for exercise levels, low EA alone is enough to cause amenorrhea, while restoration of the energy balance by meeting caloric needs is able to restore ovulation.
- Reduction in the total available energy in terms of caloric intake may be intentional or unintentional. Female athletes may not be aware that increased activity levels require increased caloric intake and may inadvertently find themselves with low EA.
- Low EA may also be the result of eating disorder concerns (for more information on eating disorders, see Chapter 27: Eating Disorders in Athletes).
- As caloric demands increase with activity, female athletes need to balance their EA by increasing caloric consumption.

## Menstrual Dysfunction

- Low EA leads to lower levels of menstrual hormones, which leads to menstrual dysfunction.
- Menstrual dysfunction begins with suppression of progesterone levels resulting in a luteal phase that is <11 days.
- Functional hypothalamic amenorrhea occurs when there is a disruption in gonadotropin-releasing hormone pulsatility; this disruption leads to decreased follicle stimulating hormone and luteinizing hormone levels and ultimately suppression of estrogen levels.
- Menstrual dysfunction due to low EA has been studied and can be reversed with restoration of appropriate energy balance.

## Low Bone Mineral Density (BMD)

- Suppression of menstrual hormones causes low BMD due to decrease in estrogen, androgens, insulin-like growth factor 1 (IGF-1), and growth hormone. Each of these hormones plays a role in increased bone deposition.
- Reduction of caloric intake resulting in low EA further reduces BMD by restricting available nutritional resources to build new bone.
- 90% of peak BMD is achieved by the age of 18 years. Adolescence is a critical age for achieving optimum BMD. Disruptions in bone mass accrual during this critical time period can have lasting consequences.
- American College of Sports Medicine (ACSM) defines low BMD as a Z score of <–1.0 in female athletes playing weight-bearing sports.
- Low BMD places the athlete at a higher risk of stress injury.
- Low BMD has been studied and shown to be reversible with restoration of EA.

## Screening

- The goal of screening is to identify athletes that have the triad, hopefully in the earliest stages, and intervene for treatment as well as to prevent future complications.
- As EA has a substantial effect on the other two components of the triad, one may choose to focus on screening for EA or eating disorders during a female athlete's preparticipation examination. Validated screening tools for EA and eating disorders include the Low Energy Availability in Females Questionnaire (LEAF-Q), Female Athlete Screening Tool (FAST), Brief Eating Disorders in Athletes Questionnaire (BEDA-Q), and the Athletic Milieu Direct Questionnaire (AMDQ).
- The Female Athlete Triad Coalition suggests screening for the triad during the preparticipation examination (see Box 12.2). Most questions are incorporated into the AAP- and AAFP-recommended preparticipation forms.
- If an athlete screens positive for low EA or eating disorder pathology or answers positively to any of the Triad Coalition questions, an interdisciplinary sports medicine team should further evaluate the athlete.

## Evaluation

- An EA calculator is available on the Female Athlete Triad Coalition website. Reviewing an athlete's daily diet and training logs can help identify issues that can be addressed. Sport nutrition may be helpful in evaluating the specific EA needs of an athlete.
- In athletes with menstrual dysfunction, it is important to first rule out pregnancy. Workup for endocrinopathy can include testing LH, FSH, prolactin, TSH, free T4, estradiol,

### BOX 12.2 PREPARTICIPATION SCREENING QUESTIONS FOR THE FEMALE ATHLETE TRIAD

- Have you ever had a menstrual period?
- How old were you when you had your first menstrual period?
- When was your most recent menstrual period?
- How many times have you had your periods in the past 12 months?
- Are you presently taking any female hormones (estrogen, progesterone, or birth control pills)?
- Do you worry about your weight?
- Are you trying to or has anyone recommended that you gain or lose weight?
- Are you on a special diet or do you avoid certain types of foods or food groups?
- Have you ever had an eating disorder?
- Have you ever had a stress fracture?
- Have you ever been told you have low bone density (osteopenia or osteoporosis)?

From De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad. *Clin J Sport Med.* 2014;24:96-119.

### BOX 12.3 INDICATIONS FOR DXA SCREENING

**1.** ≥1 "High-risk" Triad Risk Factors:
- History of DSM-V–diagnosed eating disorder
- BMI ≤17.5 kg/m$^2$, <85% estimated weight, OR recent weight loss of ≥10% in 1 month
- Menarche ≥16 years of age
- Current or history of <6 menses over past 12 months
- Two prior stress fractures, one high-risk stress fracture, or one low-energy nontraumatic fracture
- Prior Z score of <–2.0 (after at least 1 year from baseline DXA)

OR

**2.** ≥2 "Moderate-risk" Triad Risk Factors:
- Current or history of DE for ≥6 months
- BMI 17.5–18.5 kg/m$^2$, <90% estimated weight, OR recent weight loss of 5%–10% in 1 month
- Menarche between 15 and 16 years of age
- Current or history of 6–8 menses over past 12 months
- One prior stress reaction/fracture
- Prior Z score between –1.0 and –2.0 (after at least a 1-year interval from baseline DXA)

**3.** In addition, an athlete with a history of ≥1 nonperipheral or ≥2 peripheral long bone traumatic fractures (nonstress), should be considered for DXA testing if there are ≥1 moderate- or high-risk factors for the Triad. This will depend on the likelihood of fracture, given the magnitude of trauma (low/high impact) and age at which the fracture occurred. Moreover, athletes on medication for ≥6 months that may affect the bone should be considered for DXA testing.

From De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad. *Clin J Sport Med.* 2014;24:96-119.

testosterone, and DHEA/S ± 8 am 17(OH) progesterone, a progesterone challenge test, and possibly a pelvic ultrasound.
- DXA (or DEXA) screening is recommended to evaluate bone density in certain situations (see Box 12.3).
- The Female Athlete Triad Coalition developed a cumulative risk assessment that can be used to categorize risk magnitude and aid in clearance and return-to-play decisions.

### Treatment

- Treatment of the triad should be best undertaken by a multidisciplinary treatment team that includes a sport medicine physician, a mental health professional, a sport nutritionist, and possibly others as the situation warrants.
  - Inadvertent undereating and intentional weight loss without disordered eating may be treated with nutrition education and referral alone.
  - In case disordered eating is confirmed, a physician and a nutritionist should also be involved. Low EA involving clinical eating disorders should include psychological treatment by a licensed mental health practitioner.
- Treatment focuses on increasing EA through changes in diet and exercise. Restoration or normalization of body weight has shown success in resumption of menstruation and improvement of bone health.
- Consensus recommendations are to gradually increase caloric intake, beginning with a 20%–30% increase over baseline energy needs.
- If a female athlete is underweight and/or amenorrheic, the goal is to increase body weight by 5%–10%.
- Even with appropriate treatment of the triad and improvements in EA, recovery of normal menses can take months to over 1 year. Improvements in BMD may take years and may never reach optimal levels.
- Variable evidence is available regarding treatment of different components of the triad with medication. Therefore, the primary focus remains on correcting nutritional imbalance and supporting mental health and well-being.

### Return to Play

- Recovery takes time to increase energy levels, improve hormone levels, and increase BMD. The Female Athlete Triad Coalition consensus statement on monitoring and return-to-play guidelines is based on risk factors for each individual athlete.
- The return-to-play decision should be individualized and should include a comprehensive analysis of the individual athlete's situation and risk factors.
- Based on the severity of the athlete's presenting triad, prolonged monitoring may be required in order to ensure that optimal health is maintained.

## EATING DISORDERS AND BODY IMAGE IN SPORTS

- Female athletes are at a higher risk of eating disorders and subclinical eating disorders than the general population.
- Most studies on eating disorders in athletes use DSM-IV definitions wherein eating disorder not otherwise specified (EDNOS) includes subclinical eating disorders that have characteristics of eating disorders but do not meet the full criteria. The DSM-V updated EDNOS definition and created new categories of "other specified feeding or eating disorder" and "unspecified feeding or eating disorder" in attempts to be more specific with diagnosis. Unfortunately, much of the current body of research uses the older DSM-IV definitions and often the terms "disordered eating" or "subclinical eating disorders" are used, and hence, we have followed the same terminologies here.
- High school female athletes have a prevalence of eating disorders ranging 14%–32% compared with 0.5%–5% in high school female nonathletes.
- Collegiate female athletes have a prevalence of eating disorders ranging 6%–45% compared with 5%–9% reported in collegiate female nonathletes.
- Subclinical eating disorders have a prevalence of approximately 50% in high school female athletes compared with 25% in high school female nonathletes.

- The prevalence of subclinical eating disorder in collegiate female athletes is 20%–62% compared with 9% in adult female nonathletes.
- Most studies indicate that collegiate athletes have a higher prevalence of disordered eating and that this rate is sports dependent.
- In sports, there is often an ideal body image that suggests improved performance because the body could be considered as a tool for athletic performance by the athlete.
- Desires for a certain body image include a drive for thinness, muscularity, and/or leanness.
- Drive for thinness is often linked to disordered eating, while drive for muscularity is linked to disordered eating in male athletes but not in female athletes.

## CONCUSSION

- Female athletes have a greater reported incidence of concussions than do male athletes. This can be supported by comparing the incidence per athletic exposure, during both practice and game situations, between female athletes and their same-sport male counterparts.
- No definitive hormonal or anatomical etiologies have been identified to explain this increased incidence.
- In ice hockey, compared with male athletes, female athletes have been shown to have an increased incidence of concussion despite decreased number of impacts and decreased linear and angular acceleration forces of the impacts.
- Another study showed that females experienced a greater peak angular acceleration and displacement of the head–neck segment despite initiating muscle activity earlier and using a greater percentage of their maximal head–neck segment muscle area than males.
- A study evaluating soccer headgear showed that male and female soccer athletes had similar head accelerations without headgear, but female athletes actually increased their acceleration forces during heading activities when wearing headgear.
- In general, female brains have greater metabolic requirements than male brains, but this has not yet been clearly linked with the incidence of concussion.
- Baseline neurocognitive evaluations have shown some gender differences. In general, females perform better with verbal scores, while males perform better with visual tests. In addition, males have been shown to have faster speeds or reaction times, while females tend to be slower but more accurate. Differences in baselines could affect interpretation of postconcussion testing and the ability to create standardized norms.
- On initial assessment, evidence on a possible difference in the number of symptoms reported between males and females seems unclear.
  - A study showed that although males reported more symptoms, females exhibited larger neuropsychological deficits on testing.
  - Some differences have been observed in the types of symptoms reported at initial evaluation: males reported more amnesia, confusion, and disorientation, whereas females reported more drowsiness and sensitivity to noise.
- Most studies have not reported differences in the duration of symptoms and the time to return to play.
- Further research is needed to explore the explanations for the higher rate of reported concussions in female athletes.
- See Chapter 45: Head Injuries

## ASTHMA

- The overall prevalence, severity, morbidity, and mortality associated with asthma is worse in females; however, before puberty, it is actually males who have a higher incidence of asthma.
- This gender switch at puberty is possibly related to the difference in lung development between young males and females. Prepubertal males have an increased number of bronchioles, which are individually smaller in size and thus more susceptible to inflammation and constriction.
- Over time, incidence and severity of asthma improves with puberty in males, whereas it worsens in females. The incidence evens out in older age as postmenopausal females have a similar disease prevalence as do age-matched males.
- It is frequently noted that symptoms and severity of asthma can worsen with pregnancy.
- Because of the changing incidence with puberty and menopause, studies have investigated the role of estrogen and progesterone on the respiratory system. For example, progesterone has been shown to decrease the beat frequency of cilia, which may contribute to the worsening of asthma symptoms during the premenstrual phase.
- In addition, studies have revealed that women with mild-to-moderate asthma may benefit from taking OCPs, which limit premenstrual exacerbations of asthma.
- Females have been shown to report greater symptoms than males with similar diagnostic disease severity and lung function test results.
- It is important to be aware of the changing incidence and severity of asthma and airway hyperresponsiveness in female athletes as they go through puberty, pregnancy, and menopause as well as cyclical changes that may occur during normal menstruation.

## ANTERIOR CRUCIATE LIGAMENT (ACL)

- The incidence of ACL tear is 2–9 times higher in females than in males.
- Anatomic predisposing factors include greater Q angles, increased tibial and meniscal slopes, narrower femoral notches, and smaller ACL size.
- The relationship between hormonal fluctuations during menses and ACL injuries remains controversial. Both ACL and skeletal muscles have estrogen receptors, and studies have shown alterations in ligament laxity and neuromuscular control patterns in relationship to fluctuations in estrogen levels although no consistent relationship has been elucidated.
- Females have been shown to exhibit biomechanical and neuromuscular activation patterns that are associated with increased risk of ACL injury, including landing with greater knee valgus and a greater quadriceps-to-hamstring ratio activation and recruitment (see Figure 12.2).
- During growth and development, male and female children have been shown to have a similar risk of ACL injury before puberty. It appears that the anatomic, hormonal, and biomechanical patterns that place females at a higher risk of ACL injury do not develop until puberty.
- Meta-analysis has shown poorer subjective pain and activity scores after reconstruction despite similar objective measures between males and females.
- New research has identified differences in gene expression of extracellular matrix of the ACL between males and females with noncontact ACL injuries.

## PATELLOFEMORAL PAIN SYNDROME (PFPS)

- Patellofemoral pain is one of the most common presenting musculoskeletal complaints. Females are 2–3 times more likely than males to experience patellofemoral pain.
- There are no apparent differences in this incidence in terms of age or athletic participation.
- Anatomic factors that probably contribute to the higher incidence in females include wider pelvis, greater hip varus,

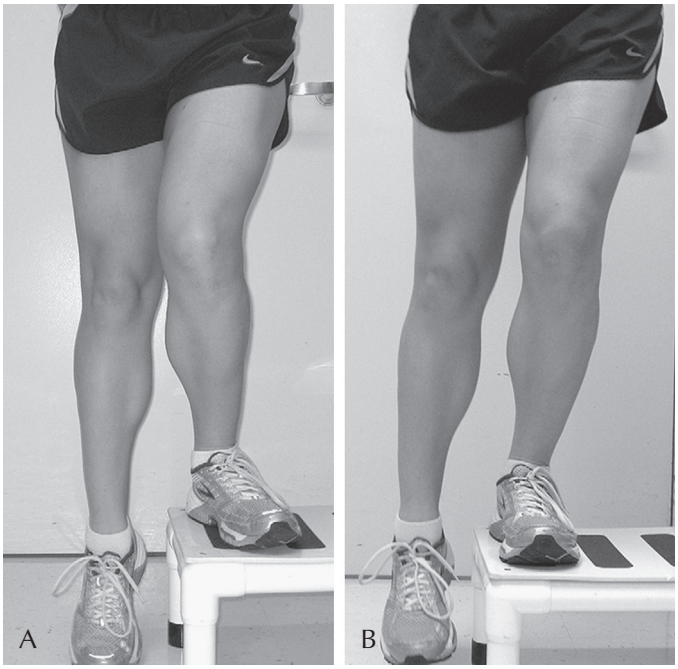
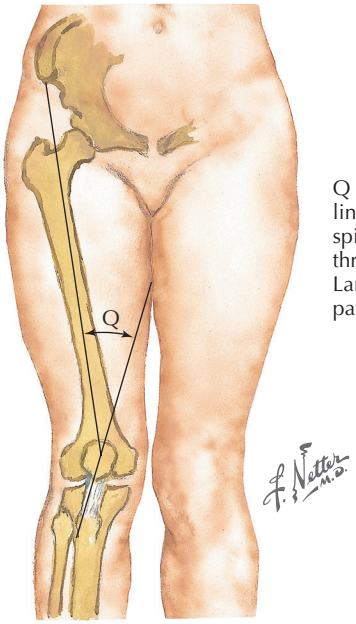



**Figure 12.2.** ACL injury biomechanics. **A** "Position of no return" that places the ACL at risk during landing or cutting. The left leg has adduction and internal rotation of the hip. The knee is in valgus with external rotation of the tibia and the foot pronated with weight on the ball of the foot. **B** A safer landing position has flexion at the hip with less or no adduction. The knee is flexed without tibial rotation, and the foot balanced. (From Boles CA and Ferguson C. The female athlete. *Radiol Clin N Am*. 2010;48:1249-1266.)




Q angle formed by intersection of lines from anterior superior iliac spine and from tibial tuberosity through midpoint of patella. Large Q angle predisposes to patellofemoral pain.

**Figure 12.3.** Quadriceps angle.

increased femoral anteversion, increased knee valgus, and increased external tibia rotation. Taken together, these factors contribute to a greater Q angle in female athletes (see Figure 12.3).

- Females have a higher incidence of patellar instability and dislocation. Anatomically, females have a higher incidence of patella alta, trochlear dysplasia, and an increased tibial tuberosity-to-trochlear groove distance, which, in general, may contribute toward both instability as well as patellofemoral pain.
  - Ultrasound studies have shown that at baseline, females may have slightly decreased vastus medialis oblique (VMO) fiber angles (the angle at which VMO fibers insert onto the medial side of the patella).
  - Moreover, ultrasound studies have shown that at baseline, the VMO insertion ratio (the extent to which VMO fibers are inserted across the entire medial border of the patella) is greater in females than in males; this would seem counterintuitive because the VMO is expected to help support patellar alignment and tracking. Thus, a greater insertional footprint would seem to decrease the risk of patellofemoral pain syndrome (PFPS). This may indicate a strength issue of the VMO or this greater footprint possibly compensates for the larger Q angle but does not compensate it completely.
- Dynamic or kinematic factors that may contribute to increased incidence of patellofemoral pain in females include excessive contralateral pelvic drop, more upright landing, less hip and knee flexion with landing, more weight on the forefoot, and ultimately, greater dynamic knee valgus.
  - Increased knee valgus can cause lateral patellar tracking, which leads to increased loading forces on the lateral patellofemoral joint.
  - A study in fencers performing an epee fencing lunge revealed that female athletes had a greater peak knee extensor moment, a shorter time to peak patellofemoral contact force, and a higher patellofemoral contact force loading rate.
- Nevertheless, these results were not consistent with those reported in studies evaluating hip and thigh strength. Certain studies have suggested that weak hip abductors may contribute to PFPS. However, studies evaluating gluteus medius have conflicted this finding, demonstrating that normalized gluteus medius activation is greater in females than in males at baseline, while other studies have shown no difference in gluteus medius activation in females with and without PFPS.
- The difference in terms of patellofemoral pain between males and females may be greater than simply incidence or prevalence. Certain studies have shown that males with patellofemoral pain may have a different biomechanical mechanism. In fact, male athletes with PFPS run and squat with less hip adduction, putting their knee into greater knee varum; such males were shown to have a more lateral center of pressure and an increased external knee adduction moment.
  - With an overly small Q angle, males may be overloading on their medial facet (as opposed to lateral facet for females with PFPS), which may have implications in diagnosis as well as treatment/management considerations for males versus females with PFPS.
- Gender has not been shown to be a predictive factor in short- or long-term outcomes for patients with PFPS.

## STRESS FRACTURES

- Female athletes have greater risk and rates of stress fracture.
- Bones in females are smaller and less dense than in males at peak bone mass.
- Estrogen is believed to play a significant role in bone metabolism: it is important for bone mineral deposition in females during puberty and peak bone mass accrual.
- Several studies have reported conflicting results regarding whether estrogen or mechanical loading is more important in female bone accrual. Certain studies have found that estrogen inhibits bone deposition in response to mechanical loading, while others have found that estrogen is necessary for females to exhibit any response to mechanical loading.

- A study that prospectively evaluated physical activity levels during childhood and obtained DXA scans at the age of 17 years found that the females who had been most active during their early childhood had significantly greater bone densities.
- In addition, women with menstrual irregularities, amenorrhea, or delayed menarche are shown to have lower bone density. If amenorrhea or delayed menarche occur during adolescence, which is the time period of peak bone mass accrual, these females may never achieve normal peak bone mass.
- Athletes who participate in weight-bearing sports and are eumenorrheic have higher BMD than do similar athletes who are amenorrheic/oligomenorrheic; therefore, athletes participating in weight-bearing sports should have higher than normal BMD compared with their nonathletic peers.
- Finally, females also experience a considerable decrease in bone mass at menopause when there is substantial decline in endogenous estrogen levels.
  - Bone loss at the microstructure level displays loss of connectivity between trabeculae in females, whereas in males, bone loss is a product of trabecular thinning and decreased bone formation. This may also contribute toward the increased risk of fracture in females.
- The role of estrogen and peak bone density are extremely vital for female athletes, particularly during adolescence and at or after menopause. Females with a prolonged history of amenorrhea, eating disorder, or female athlete triad may be at a markedly increased risk of stress injuries even if they resume normal menses and body weight.
- IGF-1 stimulates osteoblast proliferation, bone matrix protein synthesis, alkaline phosphatase activity, and differentiation. Elevated levels of IGF-1 are associated with increased BMD in women and vice versa. Studies on IGF-binding proteins have shown alterations in IGF-binding proteins 2 and 5, which are associated with increased risk of stress fractures.
- Bone-specific tartrate-resistant acid phosphatase 5b (TRAP-5b) is a bone resorption biomarker that has been shown to be significantly elevated in female athletes with stress fractures.
- Studies are currently investigating genetic influences of gender differences in bone health and risk of stress fractures.
- In addition, gender is not associated with differences in return to play.
- See Chapter 59: Stress Fractures, for additional information regarding the diagnosis and management of stress injuries.

## EXERCISE DURING AND AFTER PREGNANCY

- Physiologic changes during pregnancy include increased blood volume with decreased vascular resistance, compression of venous return, increased tidal volume, increased oxygen demand, shifted center of gravity, ligamentous laxity, and increased metabolism resulting in increased body temperature.
- There is a theoretically increased risk for certain injuries to mother and baby secondary to exercise, but there is no evidence to support this.
- Prenatal screening is no different for women who exercise during pregnancy.
- See Box 12.4 for contraindications to aerobic exercise during pregnancy.
- Exercise during pregnancy improves back pain, constipation, bloating, swelling, gestational diabetes, energy, mood, posture, sleep, and labor.
- Performing 30 minutes of moderate-intensity physical activity most days of the week is recommended throughout pregnancy, with adjusted vigorous activity on an individual basis. In general, exercise intensity recommendations are based on prepartum levels of exercise intensity.

### BOX 12.4 CONTRAINDICATIONS TO AEROBIC EXERCISE DURING PREGNANCY

Absolute Contraindications
- Hemodynamically significant heart disease
- Restrictive lung disease
- Incompetent cervix/cerclage
- Multiple gestation at risk for premature labor
- Persistent second- or third-trimester bleeding
- Placenta previa after 26 weeks of gestation
- Premature labor during the current pregnancy
- Ruptured membranes
- Preeclampsia/pregnancy-induced hypertension

Relative Contraindications
- Severe anemia
- Unevaluated maternal cardiac arrhythmia
- Chronic bronchitis
- Poorly controlled type 1 diabetes
- Extreme morbid obesity
- Extreme underweight (BMI <12 kg/m$^2$)
- History of an extremely sedentary lifestyle
- Intrauterine growth restriction in current pregnancy
- Poorly controlled hypertension
- Orthopedic limitations
- Poorly controlled seizure disorder
- Poorly controlled hyperthyroidism
- Heavy smoker

From American College of Obstetricians and Gynecologists Committee. Opinion no. 267: Exercise during pregnancy and the postpartum period. *Obstet Gynecol.* 2002;99:171-173.

- Pregnant female athletes should avoid scuba diving, supine activity, and higher altitudes if not acclimatized.
- Caution should be taken when participating in downhill skiing and contact sports, particularly after the first trimester when the gravid uterus exceeds the protection of the bony pelvis.
- Women should be mindful of caloric and hydration needs throughout pregnancy and the postpartum period and adjust intake to meet those needs.
- Postpartum exercise helps improve energy, sleep, stress levels, abdominal toning, and may help prevent postpartum depression.
- Consensus expert opinion recommends that after uncomplicated vaginal delivery, it is safe to start low-intensity exercise a few days after giving birth, with gradual increase over 4–6 weeks.
- Moreover, after cesarean section or other pregnancy complications, it is safe to start exercise after 4–6 weeks, with gradual increase over time.
- Team physicians taking care of high school, collegiate, or professional athletes should consider both the American College of Obstetricians and Gynecologists guidelines and policies of specific sports organization in balance with goals and circumstances of each individual athlete when making recommendations.

## PELVIC FLOOR DYSFUNCTION

- Pelvic floor dysfunction describes conditions related to weak pelvic floor muscles and includes stress urinary incontinence, fecal incontinence, pelvic organ prolapse, and sexual disorders.
- The incidence of pelvic floor dysfunction is higher in female athletes: 30%–80% of elite female athletes have stress urinary incontinence, although it is widely underreported.
- Stress urinary incontinence is the involuntary loss of urine and can be caused by exertion from exercise or increased abdominal pressure (e.g., during lifting weights).

- The incidence of stress urinary incontinence increases with higher-impact sports and in athletes with eating disorders. However, this increase has not been found to persist later in life, and the rates of incidence appear to return to baseline population rates.
- Female athletes with urinary incontinence often modify what sports they participate in (approximately 20% in one review) and some discontinue sports all together (10% in one review).
- There is mixed evidence to understand the effects of exercise on pelvic floor muscles. One theory is that exercise strengthens the pelvic floor. The second is that exercise stretches or overloads the pelvic floor and causes weakness, leading to pelvic floor dysfunction. One study supported another possible confounder in that exertion with exercise may fatigue the pelvic floor and cause dysfunction during that fatigue.
- None of these theories have been proven, but it is known that pregnancy with vaginal delivery can increase the rate of urinary incontinence, and pelvic floor exercises aid in reducing incontinence episodes.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# THE SENIOR ATHLETE

# 13

Jeffrey T. Abildgaard • John M. Tokish

## GENERAL CONSIDERATIONS

### Demographics

- It is widely accepted that the average life expectancy continues to increase; as it does, the proportion of older adults in the population also increases.
  - In Western industrialized countries, the average life expectancy increased from 47 years in 1900 to 78 years in 2007.
  - The number of individuals older than 85 years has increased by 232% from 1960 to 1990, along with a total population growth of 39% during the same period.
  - By 2030, 20% of the United States' (US) population will be older than 65 years.
- It is important to maintain a working knowledge of the anatomic and physiologic changes associated with normal aging and to be able to differentiate them from pathologic entities.

### Physical Activity and Health Promotion

- Numerous athletes are able to maintain a high level of participation and performance into their middle age and well beyond.
- Cumulative recommendations from the American College of Sports Medicine (ACSM), the American Academy of Orthopaedic Surgeons (AAOS), and the American Heart Association (AHA) encourage older adults to maintain a physically active lifestyle with an emphasis on moderate-intensity aerobic and muscle-strengthening activities as well as activities that promote increased flexibility and balance for older adults who are at a risk of falls.
- Evidence over recent years has demonstrated that senior athletes are not only able to participate in more endurance activities and competitive sports but also able to compete longer than ever and outperform historical comparisons.
- This growth is demonstrated by the increasing number of athletes participating in the National Senior Games, marathons, and triathlon competitions.
  - Participation in the National Senior Games, which is a biennial competition for men and women aged ≥50 years, has increased from 2500 competitors in 1987 to >12,000 athletes in 2007.
  - Almost half of all US participants in marathons are aged over 40 years. One-fourth of marathon runners in their 60s are able to outperform half of the runners aged 20–54 years.
  - The overall participation in triathlons has increased by almost 60% since 2008, and now, an estimated 850,000 adults aged over 40 years compete in triathlons in the US.
- Despite increased rates of participation, older adults remain the least active group in the US. Healthcare providers need to continue to counsel older adults regarding health benefits that coincide with an active lifestyle.
- Several changes associated with aging can be limited, prevented, or even reversed with sustained exercise.

## PHYSIOLOGIC CHANGES ASSOCIATED WITH AGING

### General Considerations

- Aging is an individual process that is influenced by a multitude of factors, including genetics, ethnicity, culture, diet, illness, environmental exposure, occupation, and physical activity.
- Age-related structural and functional changes occur at the molecular level and result in a gradual decline in the physiologic performance of virtually every organ system.
- At the cellular level, multiple changes are observed:
  - Decreased capacity for division and cellular repair
  - Impaired exchange of nutrition, cellular waste, and oxygen
  - Intracellular accumulation of lipids and pigments
  - Lipofuscin—"aging pigment" breakdown product of erythrocytes that collects in multiple issues, particularly smooth muscles and myocardium

### Effects of Aging on Specific Physiologic Systems

#### Cardiovascular

- An age-associated decline in cardiac output may be attributed to reductions in maximal heart rate, myocardial contractility, and stroke volume; these changes can result in an age-related drop in myocardial oxygen consumption/utilization (mVO2).
- Regular cardiovascular exercise and endurance training have been shown to increase mVO2 in individuals aged up to 70 years.
- Fatty and fibrous tissue deposits may occur in the myocardium that can interrupt normal conduction pathways contributing to occurrence of arrhythmias.
- Cardiac valves may thicken and lose compliance, contributing to the formation of valvular regurgitation, clinically manifesting as a murmur.
- Vascular physiology is affected by age-related changes in vascular compliance, microcirculation, and baroreceptor function; these changes, combined with atherosclerosis, can produce an increased peripheral vascular resistance, resulting in increased cardiac effort to maintain cardiac output.
  - Increased blood pressure and orthostatic hypotension are commonly observed as consequences of vascular changes.
  - Decreased capillary density and impaired vascular proliferation have also been implicated in reducing the exercise capacity of aging individuals.

#### Pulmonary

- Reduction in pulmonary microvasculature and alveoli number results in limitations in oxygen exchange and an increased sense of respiratory effort exerted during exercise.
- Decreased lung compliance results from weakness of respiratory and accessory muscles, decreased alveolar tissue elasticity, and stiffening of costovertebral and sternocostal cartilages.
- Overall, these changes contribute to a decrease in total lung capacity, vital lung capacity, inspiratory and expiratory air flows, and an increase in residual volume, respiratory frequency, and work of breathing.

#### Renal

- A progressive loss in the number of glomeruli along with increased vascular rigidity and atherosclerosis results in a decreased glomerular filtration rate with aging. Coexisting renal disease and nephrotoxic medications can exacerbate this age-related decline.
- In addition, the ability of the kidneys to concentrate urine decreases with age, resulting in a greater relative water output; this can negatively affect a senior athlete's ability to remain adequately hydrated.

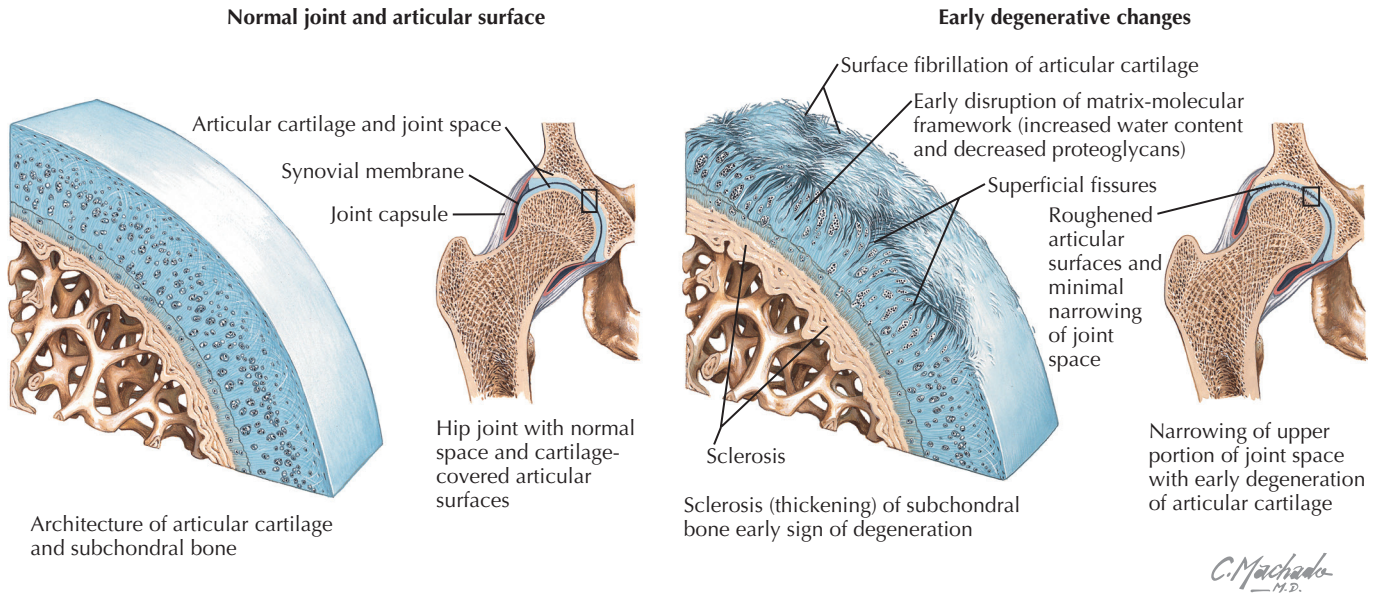


**Figure 13.1.** Early degenerative changes in articular cartilage with aging.

### Neurologic

- Progressive central nervous system deterioration has been shown to result in impaired hearing, short-term memory, balance, fine motor skills, and cognition. In addition, extrapyramidal dysfunction can result in impaired coordination and rapidity of motions and with a resultant increase in motor response time.
- Even in the absence of any known neurologic disease, peripheral nerve conduction velocities are known to decrease with age, affecting vibration and proprioception nerve fibers first.

### Vision

- Progressive vision impairment is common with age. Typical changes include decreased visual acuity, accommodation, contrast sensitivity, peripheral vision, and ability to adapt to low-light situations.

### Endocrinal

- As part of a normal consequence of aging, hormone levels gradually decline. Effects of this decline are noted in multiple organ systems:
  - With age, basal metabolic rate decreases along with increased rates of metabolic syndrome, diabetes mellitus type 2, and obesity.
  - Adrenal function is reduced, resulting in decreased aldosterone and reduced ability to regulate fluid/electrolyte balance as well as decreased stress response by cortisol.
  - Moreover, decreased rates of trophic hormones such as testosterone and insulin-like growth factor are associated with a decrease in skeletal muscle mass with age.

### Musculoskeletal

- Senile sarcopenia is the age-related decrease in skeletal muscle mass. Between the ages of 50 and 80 years, an average person will lose one-third of his muscle mass. At the cellular level, decreased cell numbers and proliferative capacity of satellite cells leads to decreased muscle regeneration and response to injury. Extracellularly, dysfunction of actin–myosin cross-linking causes muscle units to become stiffer and more susceptible to injury, particularly muscular strains.
- The structure of both ligament and tendon changes with aging. Aging ligaments exhibit fewer fibroblasts and mechanoreceptors, which may contribute to decreased ultimate failure load and mechanical stiffness. On the other hand, aging tendons exhibit fewer fibroblasts and increased degenerative changes; this along with a less robust blood supply (watershed areas) helps to explain the epidemic of tendon injuries in senior athletes.
- Articular cartilage is particularly susceptible to injury and degeneration with aging (Fig. 13.1). Few, if any, chondrocytes are produced after skeletal maturity. Production of extracellular matrix proteoglycans is more variable with time, rendering them less effective. Response to mechanical loading is impaired by decreasing cartilage water content and more rigid collagen fibrils, secondary to increased cross-linking, which increases the risk of fissuring or shear injury.
- With age, a progressive loss of bone mineral density results in compensatory widening of the diaphysis. Consequently, the susceptibility to fragility fractures and stress injuries increases.
- Men exhibit a 0.5%–0.75% annual loss of bone mass after the age of 40 years. In women, bone mass decreases much more rapidly, at a pace of 1.5%–2.0% before menopause and accelerating further after menopause to up to 3% per year.

## DECLINE IN ATHLETIC PERFORMANCE WITH AGING

### General Considerations

- Successful care of aging athletes depends on understanding the differences between normal and pathologic aging.
- It is well recognized that sedentary older adults exhibit more significant changes in functional capacity and body composition than adults who remain physically active as they age.

### Factors Affecting Performance

#### Decreased Muscle Strength

- Decrease in lean muscle mass (senile sarcopenia) is paralleled by an equal or greater decrease in strength and power. Senior athletes also experience an increase in muscle fatigability.
- Muscle power is lost at a greater rate than endurance capacity.
- Muscle cross-sectional area in men declines by 14.7% over a 12-year period, beginning at the age of 65 years.

### Loss of Endurance Capacity

- Decreased maximal aerobic capacity ($VO_2$ max) is a primary contributor to decreased endurance capacity.
- $VO_2$ max is dependent on heart rate, cardiac output, and tissue oxygen uptake.
- Peak blood lactate concentration is an indirect measure of anaerobic glycolytic activity. Peak lactate concentrations following maximal activity have been shown to be lower in men aged >60 years.

### Loss of Exercise Economy

- Decreased flexibility, joint motion, and coordination contribute to a decline in exercise economy.

## Effects of Age and Gender on Athletic Performance

- A slow progressive decline in athletic performance is seen in most athletes, starting after 35–40 years of age. Aging athletes remain capable of high levels of physical performance into their 70s.
  - Significant loss of function observed before the age of 70 years can likely be attributed to disuse, sedentary lifestyle behaviors, and genetic predisposition. Between 70 and 75 years of age, most athletes reach a tipping point, and athletic performance typically plunges.
- Analysis of running and sprinting records of master athletes in the Senior Olympics shows a <2% decline in performance per year among both men and women aged 50–75 years.
  - Age-associated decline in performance is typically greater in women. Examples in peer-reviewed literature have shown these differences to be greatest in endurance running and sprint-type swimming events (Fig. 13.2).

# BENEFITS OF EXERCISE IN OLDER ADULTS

## General Considerations

- A continually growing body of evidence establishes the benefits of exercise in aging athletes. Recommendations from the American College of Sports Medicine advocate for a combined physical activity regimen for all adults including resistance, endurance, flexibility, and balance training (Table 13.1).
- The prevalence of various diseases, including diabetes, cardiovascular disorders, mental disorders, and certain types of cancer, are lower in people who engage in consistent physical activity.
- The incidence of mortality and amount of functional disability is also lower in active aging populations.

## Musculoskeletal Benefits of Exercise

- Impact exercise can help to counteract age-related changes in bone mineral density.
  - Considerably increased bone mineral density has been observed in Senior Olympic runners aged >65 years compared with control subjects.
- Participation in resistance activities is the most effective method to offset sarcopenia.
  - Resistance training in elderly adults has been shown to increase muscle strength and cross-sectional area, increase joint range of motion, and also improve dynamic balance.
- While structural alterations in tendon and ligament tissues are observed with aging, mechanical properties can be improved with regular physical activity.
  - Physical activity has been shown to result in increased tensile strength, ultimate load, and mechanical stiffness of ligaments and preserve the size and mechanical properties of tendons.
- A decrease in age-related cartilage volume loss has been demonstrated with exercise.
  - Senior athletes participating in moderate- to high-intensity activities that load the knee and strengthen knee extensors exhibit decreased patellar and tibial cartilage volume loss.

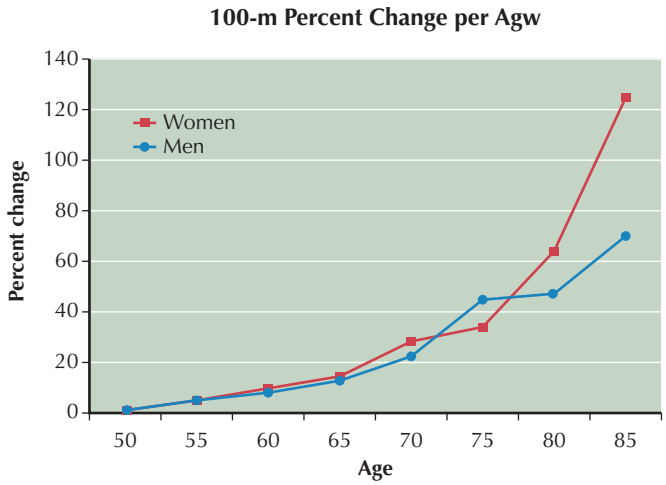


**Figure 13.2.** 2001 Senior Olympic 100-m track percentage of performance change with age. (From Wright VJ, Perricelli BC. Age-related rates of decline in performance among elite senior athletes. *Am J Sports Med.* 2008;36[3]:443-450.)

**TABLE 13.1 RECOMMENDED MINIMUM EXERCISE FOR SENIOR ATHLETES**

| Factor | Duration | Intensity (0–10)* | Comments |
|---|---|---|---|
| Endurance | 150–300 min/week | 5–8 | At least 10- to 30-min episodes |
| Resistance | 8–10 exercises of 8–12 repetitions | 5–8 | ≥2 days/week |
| Flexibility | ≥2 days/week | 5–6 | Sustained stretches, static/nonballistic movements |
| Balance | No specific recommendations | As tolerable | Progressively difficult postures, stressing postural muscle groups (particularly for frequent fallers) |

Adults who are unable to tolerate aforementioned recommendations should be encouraged to maintain the highest possible activity level and avoid a completely sedentary lifestyle.

*On a scale of 0–10 for level of physical exertion, 5–6 for moderate intensity, and 7–8 for vigorous intensity.

From Chodzko-Zajko WJ, Proctor DN, Fiatarone Singh MA, et al. American College of Sports Medicine: American College of Sports Medicine position stand: exercise and physical activity for older adults. *Med Sci Sports Exerc.* 2009;41:1510-1530.

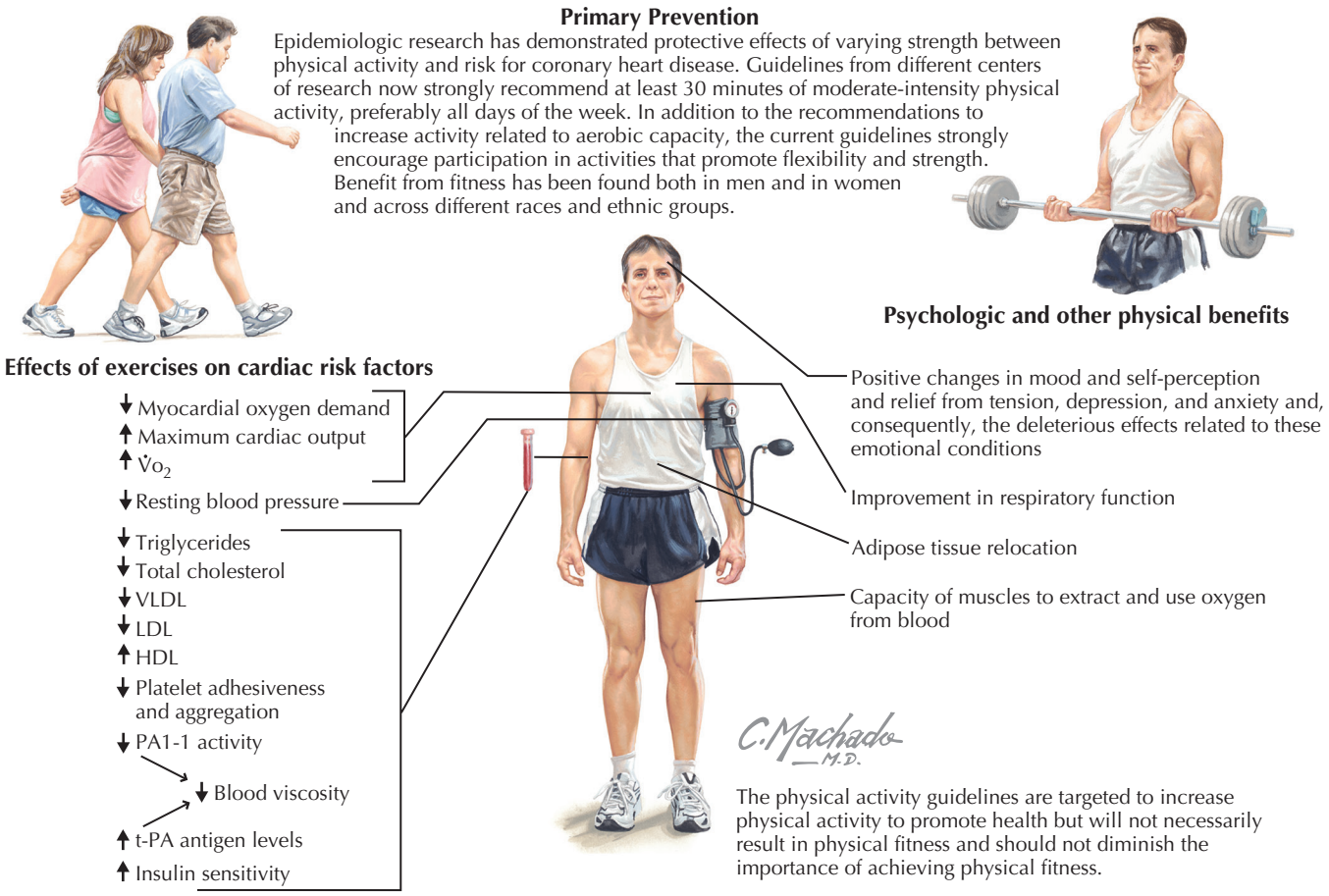


**Figure 13.3.** Effects of exercise on cardiovascular physiology.

## Systemic Benefits of Exercise

- Participation in sustained, moderately intense, aerobic activity has cardioprotective benefits (Fig. 13.3).
- Stroke volume, which is a major determinant of cardiac output, can decline with age. Sustained exercise can reduce this decline by 50%.
  - Additional cardiovascular benefits include increased vascular compliance, lower blood pressure, and decreased formation of atherosclerotic plaques.
- $VO_2$ max is frequently increased with moderately intense aerobic activity, which is beneficial for other organ systems: improved pulmonary gas exchange, renal function (resulting from better blood flow), and lactate threshold.
- Maintenance of flexibility with regular stretching activities has been demonstrated to decrease the risk of musculotendinous strains in senior athletes.
- Balance training can decrease the rate of falls in older adults at a higher risk of fall injuries.

## PREPARTICIPATION MEDICAL EVALUATION (PPE)

- In general, exercise does not provoke cardiovascular events in healthy individuals with a normal cardiovascular system. Individuals with atherosclerosis or decreased cardiac performance are at an increased risk of an acute myocardial event with moderate- to high-intensity physical activity; moreover, this risk is compounded in individuals with a previous sedentary lifestyle who abruptly begin intensive training regimens.
- The risk of sudden cardiac arrest or myocardial infarction is very low in healthy individuals who perform moderate-intensity activities. The ACSM cautions that vigorous exercise confers an acute increased risk of sudden cardiac death and/or myocardial infarction in individuals with either diagnosed or occult cardiovascular disease.
- Cardiac assessment is not required before initiating moderate-intensity exercise programs in a majority of senior athletes. Older adults with >2 risk factors are considered to be at a moderate risk of adverse responses to exercise and are advised to undergo medical examination and exercise testing before initiating high-intensity/vigorous exercise (Table 13.2).
- The American Heart Association Committee on Exercise endorses preparticipation exercise testing for all master athletes of any age with symptoms suggestive of underlying coronary disease and for those aged ≥65 years even in the absence of risk factors and symptoms.

## TREATMENT OF MUSCULOSKELETAL INJURIES IN OLDER ATHLETES

### General Considerations

- Senior athletes are often victims of two types of injuries: those that occurred in their youth and continue to be symptomatic and those that result from current athletic activity.
- Younger athletes have a higher incidence of acute traumatic injury to their ligaments and tendons than do older athletes, partly because of their participation in higher-velocity/higher-impact sports.

**TABLE 13.2 RISK FACTOR THRESHOLDS OF CORONARY ARTERY DISEASE FOR ACSM RISK STRATIFICATION**

| Positive Risk Factor | Defining Criteria |
|---|---|
| Age | Men ≥45 years; women ≥55 years |
| Family history | Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or any other first-degree male relative or before 65 years of age in mother or any other first-degree female relative |
| Cigarette smoking | Current smoker or smoker who quit within previous 6 months or exposure to environmental tobacco smoke |
| Hypertension | Systolic blood pressure (BP) ≥140 mmHg or diastolic BP ≥90 mmHg confirmed by measurements on at least two occasions or prescription for antihypertensive medication |
| Hypercholesterolemia | Low-density lipoprotein (LDL) cholesterol >130 mg/dL[1], high-density lipoprotein (HDL) cholesterol <40 mg/dL[1], or on lipid-lowering medications. If only total serum cholesterol is known, use >200 mg/dL[1] |
| Impaired fasting glucose | Fasting blood glucose >100 mg/dL[1] confirmed by measurements on at least two separate occasions |
| Obesity | Body mass index >30 kg/m$^2$ or waist circumference >102 cm for men and >88 cm for women |
| Sedentary lifestyle | Individuals not participating in a regular exercise program or accumulating ≥30 minutes of moderate-intensity physical activity most days of week for at least 3 months |
| Negative | |
| High serum HDL cholesterol | >60 mg/dL |

- A senior athlete is not immune to high-velocity injuries; however, injuries are far more likely to occur due to degenerative tissue problems that result from wear and tear due to chronic overuse or trauma experienced over years of athletic stress.
  - For example, postural malalignment of the knee, such as genu varus, can lead to overloading of the medial compartment with an increased risk of meniscal tears and degenerative chondral changes.
- Senior athletes are particularly prone to acute muscle strains and injuries at the myotendinous junction owing to age-related changes in biomechanical properties of the tendon and subsequent decrease in flexibility and tensile strength.
  - Age-related decrease in blood supply to the so-called watershed areas in tendons helps to contribute to increased pathology.
  - Ruptures of quadriceps and Achilles tendons occur more frequently in middle-aged and senior athletes.
- Tendinosis is common in older athletes and results from repetitive loading and cumulative microtrauma to tendons.
  - Common tendinopathies seen in older golfers include rotator cuff tendinopathy, medial and lateral epicondylitis, and inflammation of wrist tendons including extensor capri ulnaris tendinosis.
  - Lateral epicondylitis, or tennis elbow, occurs most commonly in middle-aged persons and is related to overuse of wrist extensors (Fig. 13.4).
  - Older joggers are particularly prone to development of Achilles tendinitis, along with posterior tibialis tendon insufficiency.
- The rotator cuff is particularly susceptible to age-related changes and pathology in aging athletes (Fig. 13.5). Rotator cuff dysfunction can range from tendinitis to massive rotator cuff tears. Partial-thickness tears are common in aging athletes and can represent a frequent source of disability.
  - Senior athletes who sustain a traumatic shoulder dislocation are more prone to rotator cuff injuries compared with younger athletes who are more likely to sustain labral injuries.

**Figure 13.4.** Lateral epicondylitis.

- The proximal biceps tendon can be a frequent source of pain in aging athletes and is particularly susceptible to degenerative tearing. Ruptures of this tendon can often relieve biceps-related pain; however, such ruptures frequently occur in association with rotator cuff pathology (Fig. 13.6). Distal biceps tendon ruptures are more frequently seen in aging male athletes; the risk of such ruptures is greater among athletes who participate in weight-lifting activities with eccentric loading of the biceps tendon.
- Repetitive, high-impact loading of joints seen in athletics can result in microtrauma to cartilage and subsequent degeneration.

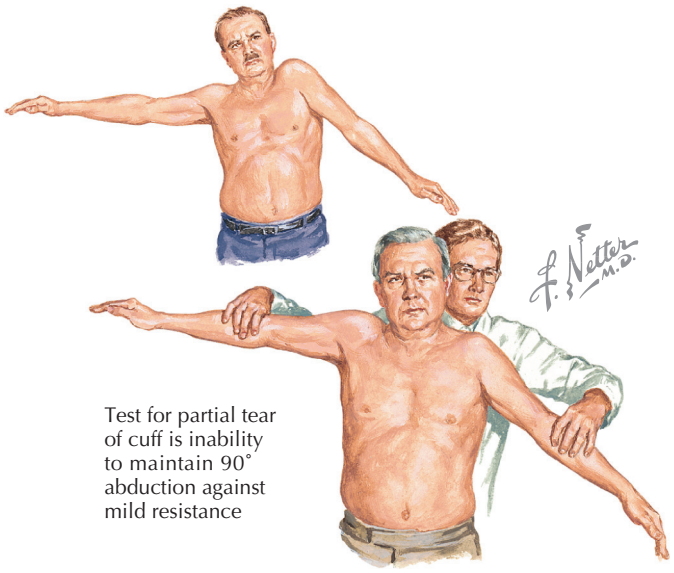


**Figure 13.5.** Aging athletes are prone to rotator cuff pathology. Patients with rotator cuff pathology can present with symptoms of pain and weakness. Patients with massive rotator cuff tears can demonstrate pseudoparalysis or the inability to actively elevate the shoulder along with anterior superior escape of the humeral head.

## Treatment Principles

- Some reports have indicated that senior athletes are more likely to wait longer to seek medical treatment for athletic injuries. In addition, several healthcare professionals may be more inclined to develop a negative attitude toward aging athletes and simply attribute injuries to expected effects of aging.
- Conflicting reports exist regarding healing rates in older athletes. Age alone does not appear to retard healing periods; however, degenerative changes in tissues at the time of the injury can adversely affect treatment outcomes.
- Initial management should include careful history and physical examination. Care should be taken to understand not only specific injury details and pathophysiology but also patients' desires and expectations regarding continued athletic activity.
- A vast majority of injuries in senior athletes are responsive to nonoperative treatment measures.
- Principles of relative rest and activity modification can be effective.
  - For example, conversion of tennis athletes from an overhead-stroke to a side-arm stroke can allow continued participation in the setting of activity but limit overhead discomfort.
- Physical therapy or home exercise regimens should be encouraged to minimize potential complications such as range-of-motion loss, cardiovascular deconditioning, or accelerated loss of bone mineral density. In addition, alternative methods of training should be discussed to avoid negative sequela from inactivity.

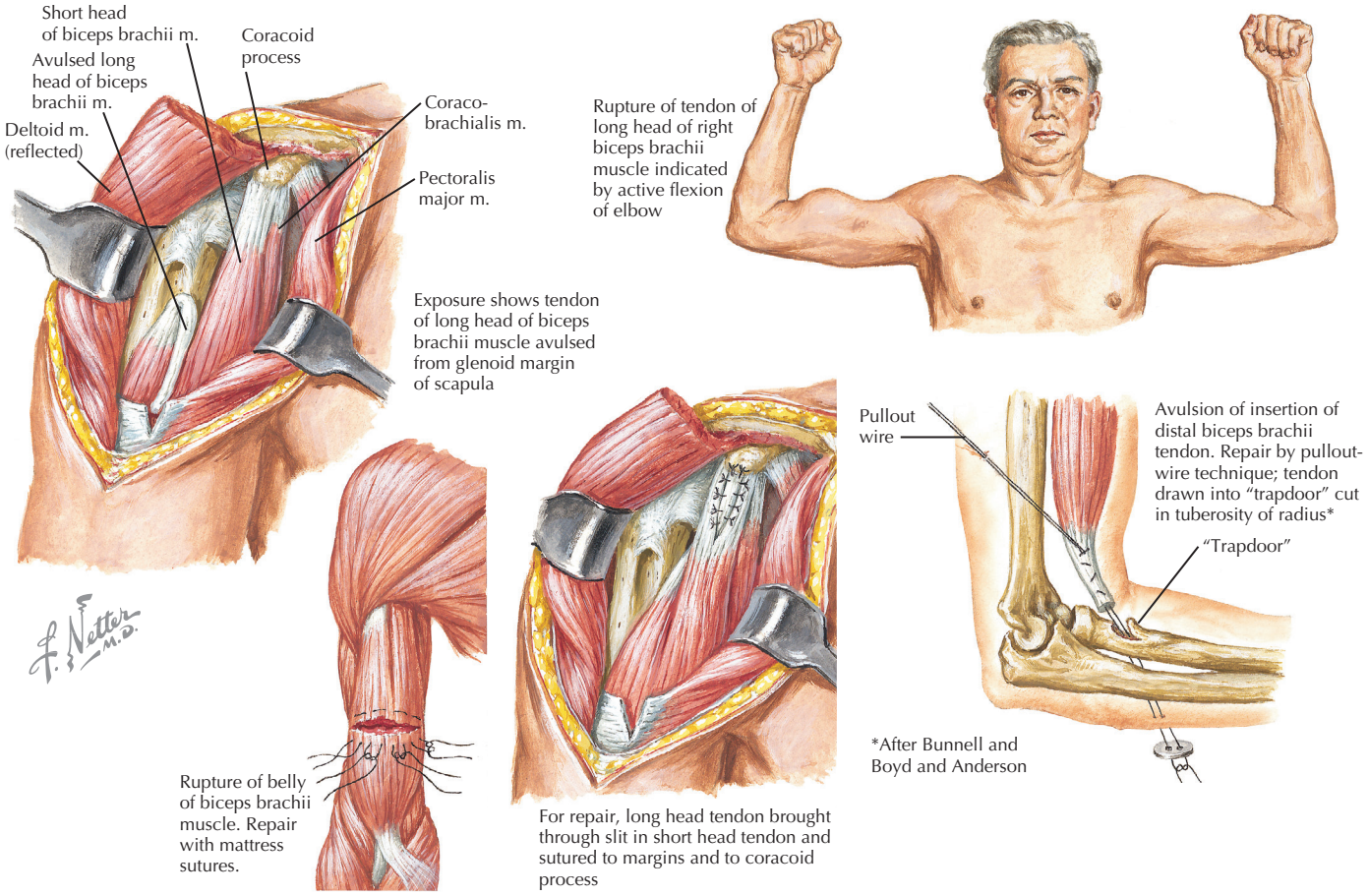


**Figure 13.6.** Rupture of the proximal and distal biceps brachii tendons.

## Pharmacotherapy

### NSAIDS

- Nonsteroidal anti-inflammatory drugs (NSAIDs) represent a common treatment modality for musculoskeletal injuries.
- Use of nonselective NSAIDs in patients aged >60 years has been associated with a four- to five-fold increased risk of gastrointestinal ulceration.
  - Individuals considered to be at a high risk (those with history of peptic ulcer disease and concurrent corticosteroid use and those who are anticipated to use NSAIDs for >3 months) should be placed on either misoprostol and/or a proton-pump inhibitor. In addition, a COX-2 selective inhibitor can be effective in patients with prior gastrointestinal irritation.
- Moreover, NSAIDs may negatively affect bone and soft tissue healing. Multiple reports have documented impaired fracture healing with concurrent use of COX-2 inhibitors during the postfracture or postoperative period. Healthcare providers may choose to discontinue use in settings of fracture or soft-tissue repair.

### Antiaging/Ergogenic Aids

- Testosterone: It is well understood that testosterone levels decrease with normal aging. Approximately 20% of 60-year-olds and 50% of 80-year-olds exhibit levels below the normal range observed in younger men. Testosterone replacement, designed to restore levels to those normal for younger men, have been shown to lead to increased lean body mass, muscle strength, bone density, and decreased body fat. Testosterone therapy is contraindicated in individuals with a history of prostate cancer and should be reserved for individuals with symptomatic hypogonadism rather than as an athletic performance aid.
- Human Growth Hormone: Currently, there is very little evidence to suggest that human growth hormone (hGH) supplementation offers any significant ergogenic advantage. Studies have shown that in a healthy elderly population, a minimal improvement in body composition leads to a considerable improvement in strength. At present, hGH supplementation is illegal for antiaging purposes in the US and cannot be recommended in the healthy elderly population.

## MUSCULOSKELETAL INJURY PREVENTION IN OLDER ATHLETES

- A primary goal of preparticipation physical examination is prevention of musculoskeletal injuries.
- Few studies exist on the prevention of injuries in older athletes; hence, evidence-based recommendations are difficult to provide. Typical assumption is that preventative guidelines for younger athletes can also be applicable to aging athletes.
- Appropriate warm-up before activity and adequate cool-down after activity are important.
- Abrupt changes in frequency, duration, or intensity of activity should be avoided.
- Days of intense physical activity should be alternated with less strenuous days to allow adequate recovery time.
- Soft playing surfaces can reduce ground reaction forces on lower limbs; however, uneven surfaces should be avoided, particularly in athletes with balance issues.
- Aging athletes should attempt to optimize environmental factors:
  - Athletes with deteriorating vision should optimize lighting conditions if possible.
  - Avoidance of extreme temperatures and humidity can aid hydration and thermoregulation.
- Activities that may exacerbate an underlying condition should be avoided; for example, athletes with osteoarthritis of the lower limb should restrict high-impact activities.
- Adequate nutrition should be maintained.

## EXERCISE AND OSTEOARTHRITIS

- It is estimated that 50% of the population aged >65 years has osteoarthritis in at least one joint. Osteoarthritis is the second leading cause of work disability in men aged over 50 years, behind only cardiovascular disease.
- Repetitive, high-impact loading of joints seen with a lifetime of athletic participation can result in microtrauma to cartilage and subsequent degeneration.
- Several risk factors have been identified for the development of osteoarthritis, including increased age, obesity, female gender, joint trauma, prolonged occupational or sports stress, joint hypermobility or instability, malalignment, and genetic predisposition.

### Management of Osteoarthritis

- Priorities in the management of osteoarthritis are to (1) reduce pain, (2) maintain mobility, (3) minimize disability, and (4) slow, halt, or reverse arthritis progression.
- Exercise programs with muscle strengthening for individuals with osteoarthritis have demonstrated improvements in strength, pain, function, and quality of life.
- Low-impact aerobic exercises can reduce pain and improve joint function.
- Participation in regular, low-impact exercise programs such as walking, aquatic exercise, and resistance training has not been shown to accelerate progression or exacerbate osteoarthritis in those who are already suffering from the disease.
- Over-the-counter supplements such as glucosamine and chondroitin sulfate have gained popularity in recent years. While these supplements contain properties that could aid in restoring cartilage, clinical trials have failed to demonstrate a clinically meaningful benefit despite an excellent safety profile.
- Corticosteroid injections can be rapidly effective, offering short-term pain relief and often functional improvement. Corticosteroid injections are ideal for acute flares of pain.
- Viscosupplementation with hyaluronic acid injections may be considered for osteoarthritis management. Early clinical results suggested a benefit of up to 6 months of pain relief, and several studies have demonstrated sustained relief beyond placebo injections with saline. However, a recent meta-analysis has demonstrated limited benefits, with results that did not affect the clinically minimal difference and noted a risk of adverse events that was not negligible. Currently, the American Academy of Orthopaedic Surgery clinical practice guidelines on management of knee osteoarthritis does not recommend the use of hyaluronic acid injections.
- Injections of biologics such as platelet-rich plasma (PRP) and stem cells are interventions that have spawned much interest in recent years.
  - Randomized clinical trials have demonstrated superior reduction in pain and functional improvement with PRP compared with viscosupplementation at 6 months in patients with early osteoarthritis aged <50 years. Additional studies have revealed that the benefit duration can vary widely.
- Mesenchymal stem cells from bone marrow and adipose tissues have demonstrated the ability to differentiate into chondrocytes. Use of these cells to treat osteoarthritis is under development and lacks randomized clinical trials at this time to demonstrate efficacy. However, early animal studies have shown the ability of stem cells to incorporate into areas of damaged articular

cartilage and improve clinical, radiographic, and histologic features of joints compared with controls.
- Surgical options for management of osteoarthritis in athletes can be divided into two strategies: joint preservation and joint replacement. For example, in the knee, joint preservation strategies can include arthroscopic debridement and meniscectomy, chondroplasty, cartilage restoration procedures such as microfracture, or high tibial osteotomy in patients with mechanical axis malalignment.

## ATHLETIC ACTIVITY AFTER ARTHROPLASTY

### General Considerations

- As the population of adults aged >65 years increases, the number of total joint replacements performed each year has steadily increased as well. Currently, over 500,000 hip and knee arthroplasties and approximately 50,000 shoulder arthroplasties are performed annually in the US.
- Patient expectations following arthroplasty are changing beyond traditional goals of pain relief and restoration of basic function with some patients choosing to pursue participation in athletic activity. Maintaining sports participation constitutes an important aspect of patients' quality of life.
- Patients should be encouraged to maintain a physically active lifestyle to not only promote joint health but also general health.
- Patient and sports-related factors must be considered when recommending return to athletic activity after arthroplasty.
  - Participant experience level: Prior experience and proficiency in a sporting activity increases the likelihood of safely resuming activity after arthroplasty.
  - Athletic activity and extent of participation: Higher activity levels have been implicated in early implant loosening, bearing surface wear, and decreased implant survivorship. Limiting activities to sports that demonstrate low joint loads (e.g., walking or swimming) can decrease excessive wear and may allow a more predictable long-term outcome.
- There is a lack of high evidence level studies to support recommendations for athletic activity after arthroplasty; therefore, it should be noted that a majority of recommendations are based on expert opinion.

### General Health Benefits

- Total joint arthroplasty has been shown to improve general and cardiovascular health.
- Marked improvements were observed in maximum oxygen consumption and Arthritis Impact Measurement Scales for mobility, walking, and range of motion following total knee arthroplasty when compared with a nonarthroplasty group.
- Increased ability to ambulate >60 minutes. Although patients are still at a risk of gaining weight following joint arthroplasty, they should be encouraged to take advantage of their potentially increased function.

### Total Hip Arthroplasty

- Osteoarthritis of the hip is commonly seen in senior athletes; total hip arthroplasty often provides excellent pain relief and restoration of joint function but is accompanied by concerns regarding implant durability and survivorship.
- Stem design can play an important role in the durability of hip arthroplasty. Proximally porous-coated stems offer the advantage of loading the proximal femur, which can lead to less thigh pain and decreased proximal stress shielding.
- While surgical approach is often determined by surgeon preference and experience, use of different approaches can be guided by patient-related factors. The anterior and anterolateral approaches offer decreased disruption of posterior tissues and have demonstrated dislocation rates of <1% without the use of hip precautions. The posterior approach may place individuals who participate in deep flexion activities involving the hip at an increased risk of dislocation.
  - Appropriate component positioning, appropriate soft tissue tensioning, and use of implants with larger head diameter can decrease the risk of dislocation.
  - Wear-related osteolysis is a major cause of failure in hip arthroplasty, making the choice of bearing surface material of critical importance in the athletic population.
- Conventional ultra-high molecular weight polyethylene (UHMWPE) demonstrates a wear rate of 0.1–0.2 mm/year. Wear rates >0.1 mm/year have been associated with osteolysis and eventual component loosening.
- Highly cross-linked UHMWPE is more resistant to wear and generates smaller wear particles. Wear rates with modern metal on highly cross-linked polyethylene are less than half of that observed with conventional polyethylene.
- Ceramic bearings demonstrate excellent wear properties but have been associated with component squeaking and fracture. While these issues have been largely resolved with newer-generation materials, ceramic components are limited to femoral heads of a smaller size, thus potentially increasing the risk of dislocation.
- Metal-on-metal bearing surfaces were originally deemed advantageous owing to large femoral head size; however, high serum metal ion levels and adverse tissue reactions have been well documented, leading to a drastic decline in their use.
- High rates of return to athletic activity after total hip arthroplasty have been well documented. Recommendations for athletic activity are sport specific and should be thoroughly discussed with the operating surgeon (Table 13.3).
- In general, rehabilitation after total hip arthroplasty lasts 4–5 months, and certain patients are able to return to sports within 3–6 months.

### Hip Resurfacing

- Hip resurfacing has several theoretical benefits over total hip arthroplasty including increased head size and range of motion, decreased femoral bone resection, and a decreased wear rate with metal-on-metal articulation.
- Findings of variable outcomes in the literature regarding implant survival and return to sports.
- Prospective studies with short-term follow-ups have suggested that return to sports can be >90%. Age at the time of surgery and preoperative activity level are common predictors of postoperative level of function.
- Implant-associated risk of femoral neck fractures, increased levels of metal ions in blood and urine, which carry an unknown risk, and risk of developing pseudotumors should be discussed with patients before surgery.

### Total Knee Arthroplasty

- Knee osteoarthritis is a common source of disability and is frequently encountered in the athletic population because of either a posttraumatic etiology or age-related degenerative changes.
- Total knee arthroplasty has demonstrated considerable success in patients with end-stage tricompartmental arthritis.
- Studies regarding levels of return to activity following total knee arthroplasty have reported an average return to sports of 63%–77% for athletes returning to low-impact activities.
  - Patients should avoid athletic activities until their quadriceps and hamstrings are adequately rehabilitated to decrease the risk of postoperative injuries.

**TABLE 13.3 ACTIVITY RECOMMENDATIONS AFTER TOTAL HIP ARTHROPLASTY**

| Recommended | Recommended With Experience | Not Recommended |
|---|---|---|
| Golf | Weight lifting | Singles tennis |
| Swimming | Cross-country skiing | Racquetball/squash |
| Double tennis | Downhill skiing | Jogging/running |
| Stair climber | Ice skating/roller blading | Snowboarding |
| Walking/speed walking | Pilates | Contact sports (football, hockey, or soccer) |
| Hiking | | High-impact aerobics |
| Stationary skiing | | Martial arts |
| Bowling | | Waterskiing |
| Cycling | | Handball |
| Elliptical | | |
| Low-impact aerobics | | |
| Dancing | | |
| Rowing | | |
| Weight machines | | |
| Treadmill (walking) | | |

From Vogel LA, Cartenuto G, Basti JJ, Levine WN. Physical activity after total joint arthroplasty. *Sports Health.* 2011;3:441-450.

**TABLE 13.4 ACTIVITY RECOMMENDATIONS AFTER TOTAL KNEE ARTHROPLASTY**

| Recommended | Recommended With Experience | Not Recommended |
|---|---|---|
| Low-impact aerobics | Cycling | Racquetball/squash |
| Bowling | Hiking | Contact sports (football, hockey, or soccer) |
| Golf | Rowing | Rock climbing |
| Dancing | Cross-country skiing | Jogging/running |
| Walking | Stationary skiing | Singles tennis |
| Swimming | Speed walking<br>Doubles tennis<br>Ice skating | Waterskiing<br>Baseball/softball<br>Handball<br>Martial arts |

From Vogel LA, Cartenuto G, Basti JJ, Levine WN. Physical activity after total joint arthroplasty. *Sports Health.* 2011;3:441-450.

- On an average, patients returning to golf demonstrate an increase in their handicap rate and decrease in the driving distance.
- Most patients can expect return to walking, cycling, and other low-impact activities in 3–6 months.

- Surgical implants that allow knee flexion beyond 125 degrees have demonstrated greater patient subjective outcomes and carry the potential for decreased bearing surface wear for individuals participating in deep knee flexion activities.
- General consensus by most orthopedic surgeons is to avoid high-impact activities (Table 13.4). Despite these recommendations, there are reports of patients participating in higher-impact activities with excellent mid-term results and no increased risk of revision surgery.

### Unicompartmental Knee Arthroplasty

- Unicompartmental knee arthroplasty has several advantages over total knee arthroplasty, including decreased recovery time, preserved bone stock, improved quadriceps function, increased knee flexion, and more normal gait kinematics.
- Unicompartmental knee arthroplasty is contraindicated in patients with concurrent ACL insufficiency, tricompartmental arthritis, fixed varus/valgus deformity >10 degrees, inflammatory arthritis, and <90 degrees of motion or a significant flexion contracture.
- Several studies have examined return to sports after unicompartmental knee arthroplasty with approximately 90% of patients returning to sport at any level and up to 60% returning to the same level of play.

### Total Shoulder Arthroplasty

- A recent study examining athletic activity after shoulder arthroplasty revealed that 64% of patients who underwent surgery listed their desire to continue to play sports as a factor in their decision to undergo surgery. In addition, 50% of patients were able to increase their level of participation, and 71% exhibited improvement in their ability to play.
  - Most patients were able to partially return to sports at 3.6 months and completely return to sports by 5.8 months after surgery.
- Historically, reverse shoulder arthroplasty has been performed in low-demand patients with rotator cuff arthropathy and massive irreparable rotator cuff tears. As the procedure has evolved, patients' expectations have risen as well. Literature regarding return to sports is limited; however, a 2015 study revealed that 60% of patients were able to return to sports after reverse shoulder arthroplasty; of these patients, 95% were able to play at the same level as they did before surgery.
- None of the previous studies have demonstrated an increased risk of implant loosening with activities such as golf; nevertheless, recommendations for athletic activities after shoulder arthroplasty are based on limited evidence and expert recommendation (Table 13.5).

## METABOLISM AND NUTRITION IN OLDER ATHLETES

### General Considerations

- Age-related decline in resting metabolic rate can contribute to a change in body composition.
  - Resting metabolic rate, which is estimated to constitute 75% of daily energy expenditure, decreases by 10% from childhood to adulthood and an additional 10% by the sixth decade of life.
  - Recent studies have suggested that age-related decline in resting metabolic rate can be attenuated with regular exercise.
- Typically, aging athletes attempting to lose weight combine physical activity with a decrease in caloric intake, which can lead to a negative energy balance. A deficiency of 1000 calories per day results in muscle catabolism and decreased metabolic

**TABLE 13.5 ACTIVITY RECOMMENDATIONS AFTER TOTAL SHOULDER ARTHROPLASTY**

| Recommended | Recommended With Experience | Not Recommended |
|---|---|---|
| Jogging/running | Racquetball/ squash | Contact sports (football, hockey, or soccer) |
| Aerobics | Baseball/softball | Martial arts |
| Tennis | Downhill skiing | Weight lifting |
| Basketball | Snowboarding | Volleyball |
| Stair climber | | Waterskiing |
| Hiking | | Handball |
| Cross-country skiing | | Gymnastics |
| Ice skating/roller blading | | Rock climbing |
| Bowling | | |
| Cycling | | |
| Rowing | | |
| Walking/speed walking | | |
| Dancing | | |
| Pilates | | |
| Golf | | |
| Swimming | | |
| Elliptical | | |
| Fencing | | |

From Vogel LA, Cartenuto G, Basti JJ, Levine WN. Physical activity after total joint arthroplasty. *Sports Health*. 2011;3:441-450.

efficiency. A chronic negative energy balance has been associated with decrease in bone mass and an eight-fold increase in the risk of stress fracture.

## Macronutrients

- In general, older adults need fewer calories to maintain body weight, which indicates an increased emphasis on the quality of calories consumed.
- Training volume is the key factor for predicting energy needs and nutrient requirements.
- Carbohydrate intake should be in the range of 45%–65% of total daily caloric intake. For older athletes, calculating carbohydrate needs based on grams per kilogram of body weight is more likely to be appropriate for energy needs of athletes. Typically, 5–7 g/kg/day is sufficient for general training needs; requirements of endurance athletes are often higher and may necessitate 7–10 g/kg/day.
- Protein recommendations for senior athletes have not been established; however, a diet with sufficient protein is important to help offset age-induced sarcopenia. Protein intake of 1.2–1.7 g/kg/day should provide adequate amino acids to help maintain a positive balance for muscle synthesis and repair.
- Recommended daily intake of fat should be 20%–30% of total daily calories. Low-fat diets have not been shown to increase athletic performance.

## Micronutrients

- To help offset age-related changes, adults over the age of 50 years have increased needs for vitamin D, vitamin B6, and calcium. Currently, there is lack of evidence to suggest exact requirements in older individuals who participate in sports.
- Recent reports state that 77% of the US population have vitamin D deficiency. Vitamin D has been considered essential for maintaining bone health. In addition, vitamin D receptors have been identified in almost every cell type in the human body and are thought to play important roles in skeletal muscle maintenance.
  - Multiple performance studies in older adults have related low vitamin D levels to decreased reaction time, poor balance, and an increased risk of fall injuries. Supplementation has demonstrated improvement with strength, walking distance, and decrease in general discomfort.
  - Previous studies have established optimal vitamin D level as >40 ng/mL. Supplementation in younger athletes to obtain optimal levels has demonstrated improvements with short-distance sprinting and jumping compared with individuals on placebo.
  - Current recommendations for adults include daily vitamin D intake of approximately 1000 IU. To optimize performance, athletes may consider supplementation to obtain levels >40 ng/mL.
  - In addition, vitamin D can be synthesized from exposure to the sun. Athletes living at latitudes above 35–37 degrees should not rely on sun exposure alone for vitamin D production.
- Calcium is equally important in appropriate maintenance of bone health. A minimum intake of 1200 mg of calcium per day is necessary.
- Vitamin B6 is necessary for breakdown of glycogen stores in muscle and helps convert lactic acid to glucose in the liver. Requirements for B6 increase with age. Men and women older than 50 years should consume at least 1.7 and 1.5 mg of vitamin B6 per day, respectively.
- Iron is the only micronutrient that is needed in smaller amounts for women with age. After menopause, the recommended dietary allowance (RDA) for iron decreases from 15 mg/day to 8 mg/day. For men, the value does not change with age. For athletes participating in endurance exercise, additional iron supplementation of approximately 30% is needed to offset losses observed with exercise.
- Antioxidant vitamins such as vitamins A, C, and E have been proposed to help reduce exercise-related tissue damage and even promote repair.
- Deficiency of most micronutrients is rare if athletes consume a balanced diet that includes fresh fruits and vegetables.

## Fluids

- Adequate hydration during athletic activity is vital to compensate for fluid loss. Older athletes are susceptible to dehydration due to a diminished thirst sensation, decreased renal function and urine concentration capacity, and greater insensible fluid losses compared with younger individuals.
- Adequate hydration during exercise can help to maintain performance level, to lower submaximal heart rate, and to help control core body temperature.
- Athletes should not rely on thirst to decide when to drink and should consume fluids on a regular schedule during activity. For optimal recovery, it is recommended that 150% of weight lost acutely during activity should be replaced with fluid.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE PHYSICALLY CHALLENGED ATHLETE

# 14

Katherine L. Dec • Daniel Contract

## GENERAL CONSIDERATIONS

### Definitions

**Physically challenged:** combines all groups of athletes competing in international competitions such as Paralympics; such athletes have an impairment that limits their ability to participate in athletic arenas within a manner considered "normal" for defined sport

**Impairment:** any loss or abnormality of psychological, physical, or anatomic structure or function

**Disability:** any restriction imposed from an impairment that limits an individual's ability to perform an activity within a manner considered "normal" for an able-bodied individual

**Handicap** (as defined by World Health Organization): a disadvantage, resulting from impairment or disability that interferes with a person's efforts to fulfill a role that is normal for that person; handicap is a *social concept*, representing social and environmental consequences of a person's impairments or disabilities

### Statistics

- Over 56.7 million disabled people in the United States (US); many nonambulatory
- "Disabled" classification broad; includes nonathletic population
- Over 200,000 people in the US with spinal cord injury (SCI)
  - Includes traumatic and nontraumatic
  - 11,000 new injuries per year; average age at injury is 32 years
  - 55% tetraplegia, 45% paraplegia
- Over 1,540,000 million people in the US with limb loss
  - Incidence of congenital limb deficiency is 60 per 100,000 live births.
  - People older than 65 years account for 19.4 per 1000 of those with limb loss.
  - Comorbidities: diabetes, vascular, and malignancy
  - Incidence:
    - Lower extremity amputation (LEA), diabetes, and younger than 30 years: 7.2%
    - LEA, diabetes, and older than 30 years: 9.9%
    - Dysvascular disease: 46.2 per 100,000 with limb loss
    - Trauma: over 5.86 per 100,000 with limb loss (war increases this rate)
    - Malignancy: 0.35 per 100,000 with limb loss
- Multiple sclerosis: 400,000 cases diagnosed in the US each year
- Muscular dystrophies: new cases estimated at 250,000 each year in the US; Duchenne muscular dystrophy is 1 of the 9 types of muscular dystrophy

### History

- First sports event, physically challenged: 1888, Sport Club for the Deaf; Berlin, Germany
- First international competition for the disabled: International Silent Games, 1924
- First international sports competition for people with various physical impairments: Stoke Mandeville Games for the Paralyzed, 1948
- Youth divisions, in addition to adult, for athletes with physical impairment: 1980s
  - Adaptive physical education, hippotherapy (a form of therapy using the characteristic movements of a horse to provide carefully graded motor and sensory inputs), and aquatic therapy: 1980s

### Competition

- Interscholastic, collegiate, professional sports: physical impairment cannot require changes in rules of a sport, lowering of standards for achievement, or modification of a defined sport to accommodate athletes
- Neither adaptive equipment nor physical impairment can impart danger or an advantage to athletes or others competing in that sport.
- Wrestling
  - Those with hearing loss have successfully competed with normal hearing athletes; if hearing loss is >55 decibels in the better ear, qualifies for physically challenged
  - Limb loss: must weigh in with prosthesis, if used
- Jim Abbott, professional major league baseball. Congenitally absent right hand
- Archery
- Below-knee amputations (BKAs)
  - Allowed in high school football after restrictions removed in 1978; check local competition rules
  - National Federation of State High School Associations' rules concerning contact sports:
    - Restricted to BKA; no upper extremity or above-knee prosthesis
    - Metal hinges restricted to lateral and medial; require covering
    - No metal in front of knee unless appropriately padded
    - Prosthesis wrapped with minimum of half-inch foam rubber or appropriate polyurethane
    - Approval of physician associated with amputee care recommended
- Paralympics
  - International competition following Olympics
  - Traditionally includes athletes with limb deficiency, cerebral palsy (CP), visual impairment, SCI, "les autres" (those not fitting into other groups), and intellectual disability
  - Currently, intellectual disability is not a participant group in the International Paralympic Committee (IPC)-sanctioned events. The IPC has asked the International Sports Federation for Persons with an Intellectual Disability (INAS-FID) to develop eligibility and verification processes that are commensurate with other IPC divisions to ensure fair competition.

### Classification Systems

- System used to equalize athletes in competition using objective methods:
  - **Medical diagnosis only:** e.g., CP, limb deficiency, and muscular dystrophy

- **Functional measurement only:** e.g., wheelchair mobility level, above-knee amputation (AKA), and BKA
- **Hybrid:** use functional measurements and medical diagnosis; multiple sclerosis with full trunk control and wheelchair mobility
- May be sports-specific for alpine skiing or cycling
- Classification systems may differ at international and local competitions.
- 1996 Paralympics: Of 3500 athletes, the most common impairment was limb deficiency.

## GENERAL CONSIDERATIONS FOR TREATMENT OF ATHLETE

- Cognitive age differences: coping with impairment
  - Adult: potential concurrent medical issues, social isolation
    - Management of comorbid diabetes, arthritis, or other diseases
- Youth: peer interaction, relationships
  - Missed social/peer opportunities
  - Constant change in size/fit of adaptive equipment
  - Health insurance: have benefit and Medicaid limits
- Counsel: assist athlete in redesigning athletic or career goals
- Financial needs: insurance coverage, private funds, or home equity loans
  - Paperwork, appeal process, or funding for equipment needs
- Physical office facilities: Americans with Disabilities Act (ADA) criteria for accessibility
  - Adjustable-height examination table
  - Appointment scheduling adjustment to allow time to address unique mobility, equipment, or comorbidity issues
- Establish virtual office with other healthcare professionals (e.g., neurosurgeon, physiatrist, therapist, vocational rehab, psychologist, primary care physician, prosthetics, or orthotist)

## ORGANIZATIONS

- Several US and international organizations address needs of physically challenged athletes (Box 14.1), e.g., Disabled Sports USA (DS/USA):
  - Founded 1967, by disabled Vietnam veterans
  - Provides opportunities for those with disabilities to gain confidence and dignity through sports, recreation, and educational programs
  - Nation's largest multisport, multidisability organization, serving >60,000 people

### BOX 14.1 RESOURCE ORGANIZATIONS

#### USA

- National Disability Sports Alliance (NDSA): www.ndsaonline.org
- United States Les Autres Sports Association (USLASA)
- Dwarf Athletic Association of America (DAAA): www.daaa.org
- Amputee Coalition of America (ACA): www.amputee-coalition.org
- National Center on Health, Physical Activity and Disability (NCHPAD): http://www.nchpad.org/
- Disabled Sports USA (DS/USA): www.dsusa.org
- Adaptive Sports Foundation: www.adaptivesportsfoundation.org
- BlazeSports: www.blazesports.com

#### International

- International Paralympic Committee (IPC): www.paralympic.org
- International Wheelchair and Amputee Sports Federation (IWAS): www.iwasf.com
- Member of the US Olympic Committee
- Sponsors the Wounded Warrior Project

## SPINAL CORD INJURY

### Physiologic Changes in Exercise

- Altered venous return, consequent decreased ability to respond to exercise stress
- Depending on level of SCI, possible blunting of heart rate response to exercise
- Vagal withdrawal, not sympathetic drive
  - Decreases reflexive regulation of blood flow
  - Decreased total peripheral resistance (increased vasodilation)
  - Increased peripheral pooling
    - Treatment to minimize: compression garments or abdominal binder
  - Decreased oxygenated blood to exercising muscle
    - Fatigue, limited aerobic endurance
- Cardiac repolarization abnormalities
- Decreased lactate threshold
- Limited pulmonary capacity, generally restrictive type (due to respiratory muscle weakness)
- Kinetic chain disruption
  - Loss of ground reactive force from lower extremity
  - Stabilizing muscles become prime movers.
- Greater muscular strength improves aerobic power and endurance.
- Paraplegics and people with high-level SCI can increase $VO_2$ max with exercise.
  - Dependent on intensity, frequency, and duration

### Medical Concerns in Athletes

#### History

- SCI level: complete or incomplete, type of injury (Figs. 14.1 and 14.2)

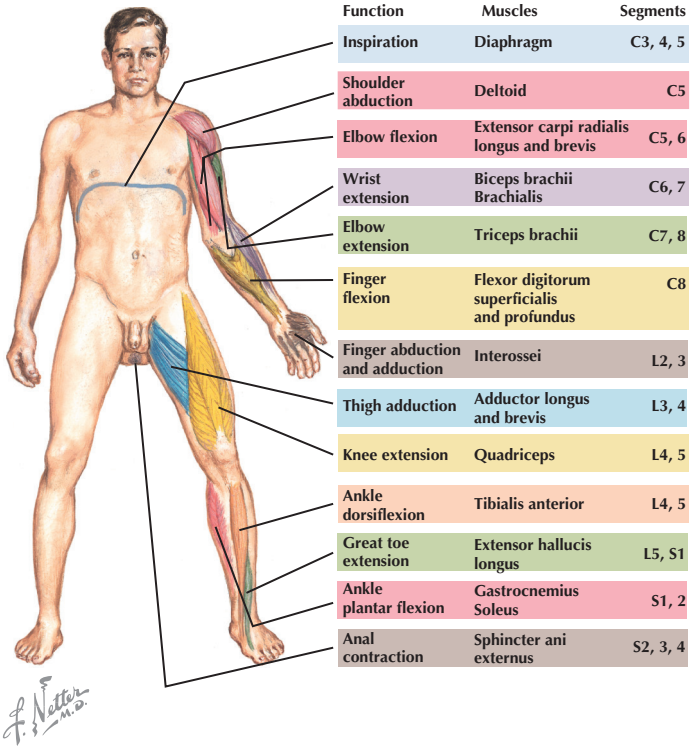


**Figure 14.1.** Motor impairment related to level of SCI.

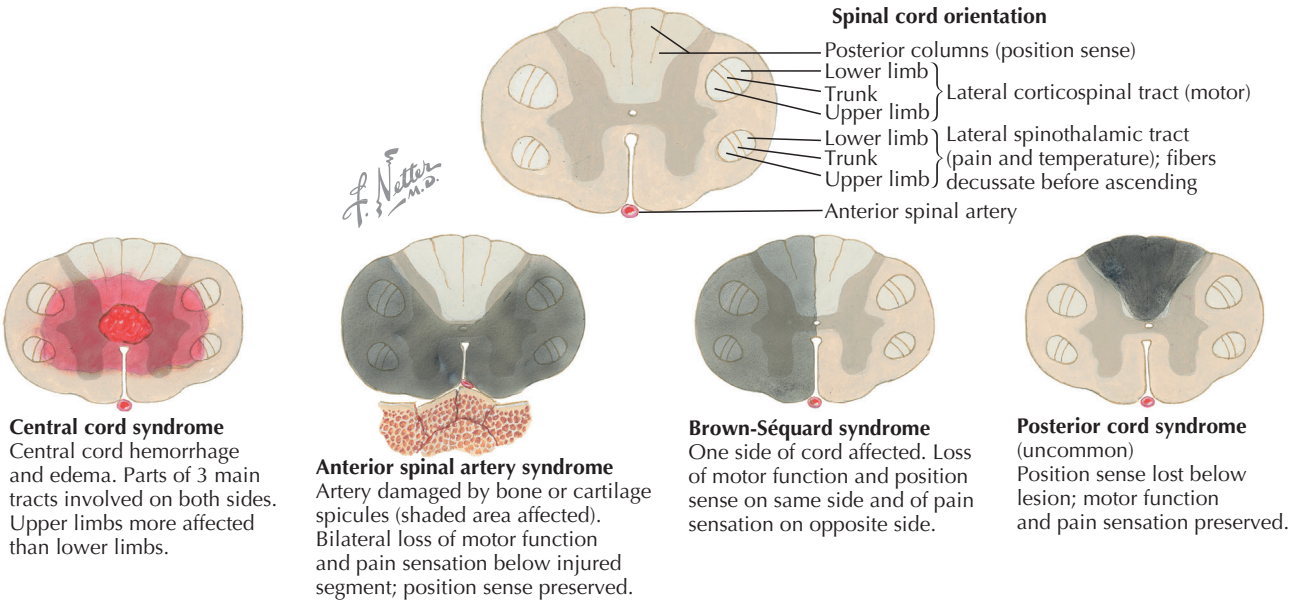


**Figure 14.2.** Incomplete spinal cord injuries.

- Surgeries related to injury: past spinal fusion, surgical muscle transfers for functional improvement, or surgically implanted medical devices
- Medications: antiepileptics, antispasmodics, tricyclic antidepressants, anticholinergics, baclofen pumps, pain medications, and others for comorbidities
- Comorbid medical issues; related to:
  - Impairment: e.g., pressure sores, type and success of bowel/bladder management program; recurring urinary tract infection (UTI)
  - Concurrent illness: e.g., traumatic brain injury (TBI), diabetes, visual impairment, amputation, cardiac disease, or seizures
- Level of functional independence: independent transfers with wheelchair, self-management of personal hygiene
- Adaptive equipment needs: for sports-specific or general mobility
- Prior training: environmental conditions, aerobic and anaerobic conditioning, or flexibility

## Physical Conditions to Consider

### Deep Venous Thrombosis (DVT)

- Risk greatest in the first 2 weeks after injury
- Other risk factors: obesity, trauma to pelvis and lower extremities, congestive heart failure, prior malignancy, tight garments below level of lesion, and previous thromboembolism
- Venous pooling in lower limbs
- Prevention: passive stretching of limbs, abdominal binder, and functional electrical stimulation (latter needs further research)

### Heterotopic Ossification

- Etiology unclear
- Symptoms: pain, increased warmth, swelling, and decreased joint motion or contracture
- Incidence: 16%–53%
- Locations: hip, followed by knee, shoulder, and elbow
- Prevention: initial treatment after onset of SCI with nonsteroidal anti-inflammatory drugs (NSAIDs) and passive range of motion (ROM)/mobilization
- Risk decreases 2–3 times with appropriate treatment
- Treatment:
  - Stretching and passive ROM exercises
  - Medication: NSAIDs (e.g., indomethacin) and bisphosphonates (e.g., etidronate)
    - Etidronate disodium: blocks aggregation, growth, and mineralization of calcium hydroxyapatite crystals; heterotopic ossification can occur in previously etidronate-treated patients
  - Surgical excision: high reoccurrence rate, lesser if delayed until skeletal maturity/low bone turnover rate
- Imaging: three-phase bone scan; radiographs often negative during initial phase of symptom presentation; may take 4–5 weeks for findings to appear
- Laboratory findings: significant elevation in fractionated alkaline phosphatase during bone ossification

### Autonomic Dysreflexia (AD)

- Medical emergency in T-6 and above SCI level
- "T-6" refers to motor and sensory nerve impairment of SCI at the sixth thoracic level
- No supraspinal neurologic inhibition; sympathetic nervous system is left unchecked
- Symptoms: elevated blood pressure, headache, piloerection, profuse sweating, nasal congestion, and/or bradyarrhythmia
- Elevated blood pressure: a systolic increase of 20–40 mmHg or diastolic increase of 10 mmHg in adults, or 15 mmHg systolic increase in adolescents; may be the *only* symptom
- Cause: noxious stimulus to spinal cord below level of SCI, such as pressure sores, UTI, fracture, tight clothes, distended bowel or bladder, or heterotopic ossification
- Treatment: remove offending stimulus (Box 14.2)
- Performance enhancement: boosting
  - Increases cardiovascular demand; a small-scale study demonstrated increased release of catecholamines during exercise; higher peak performances, heart rate, $O_2$ consumption, and blood pressure

### Pulmonary Complications

- Atelectasis, pneumonia, and mucous plugging are most common causes of morbidity in high-thoracic SCI and tetraplegia.

**BOX 14.2 TREATMENT STEPS FOR AUTONOMIC DYSREFLEXIA (AD)**

- Sit person up, lower legs
- Loosen clothing or constrictive devices
- Check blood pressure every few minutes until patient stabilizes
- Remove offending stimulus
- Bladder distension is most common
- Bladder catheterization: 2% topical lidocaine jelly helpful
- 1–2 inches of topical nitropaste or immediate-release nifedipine 10 mg, bite and swallow
- Repeat medicine in 15 minutes if no improvement
- Continue to monitor blood pressure and symptoms for 2 hours after episode
- If offending stimulus still present, recurrence of AD possible

### Urinary Tract Infection

- Potential comorbid issue because of bladder management choice
- Long-term use of indwelling catheters: higher risk of UTI
- Symptoms can be masked as insensate below SCI level: patient does not experience typical urinary urgency, dysuria, or flank pain
- Initial symptoms: increased spasticity, malaise, sweating, or AD
- Treatment: rigorous personal hygiene for prevention
- High frequency of resistant organisms: obtain urinalysis with Gram stain, culture, and sensitivity when possible *before* initiation of antibiotics
- Abnormal bacterial count but no symptoms: usually managed without antibiotics

### Thermoregulation

- Sweating often impaired below level of SCI
- Less surface area (e.g., arms and upper trunk) for evaporative cooling
- Check skin under arms of a distressed athlete during competition; if hot, athlete is likely not adequately dissipating heat
- Treatment: lighter clothing, more fluids, and dousing the skin with water

### Hypothermia

- Contributing factors: decreased muscle mass below level of lesion, loss of vasomotor and sudomotor neural control, and possible decreased input to hypothalamic thermoregulatory centers
- Impaired or absent sensation intensifies the risk: unaware of clothing dampness, which augments heat loss

### Pressure Sores

- Inappropriately fitting prosthetics or poor seat position/posture in wheelchair athletes
- Risks: racing/track wheelchairs put knees higher than hips and increase pressure over sacrum and ischium
- Treatment:
  - Appropriate wheelchair positioning; accurate fit of prosthetics/orthotics
  - Performing regular pressure reliefs and weight shifts
  - Lifting self off a seat for 10–20 seconds per day
  - Adequate cushioning, seat system
  - Reducing skin moisture (wearing absorbent fabric)
  - Minimizing skin shear
  - Early wound management
    - Topical skin products before dermis is breached; debridement

### Neurogenic Bladder

- Disrupted regulatory control of bladder contraction and voiding
  - Areflexic (decreased tone/increased capacity, with urinary retention)
  - Reflexic (increased tone/decreased capacity, with involuntary voiding)
- Treatment:
  - Medication and/or program to empty bladder
  - Intermittent urinary catheterization: insert catheter into urethra and bladder to drain urine into a disposable bag; performed on a strict schedule
  - Continuous (indwelling) urinary catheter: long-standing indwelling catheters contribute to recurrent UTIs; condom catheters are an option in men
  - Suprapubic catheter: least common in athletes
  - Other option in complete SCI (minimal research in athletes): sacral anterior root stimulation
  - Medications:
    - Anticholinergics (e.g., oxybutynin, tolterodine, imipramine, and propantheline) for detrusor hyperreflexia
    - Alpha-adrenergic antagonists (e.g., terazosin, prazosin, and phenoxybenzamine) reduce internal sphincter tone
    - Antispasticity drugs (e.g., baclofen, diazepam, tizanidine, and dantrolene) of value in those with severe spasticity of perineal muscles
    - Medications for both types of neurogenic bladder can have side effects; impaired sweating, drowsiness, hypotension, and gastrointestinal problems

### Neurogenic Bowel

- Regulation of normal bowel elimination impaired
- Medications: include stool softeners (e.g., docusate, polyethylene glycol, and methylcellulose), prokinetics (e.g., senna), rectal suppositories (e.g., bisacodyl), and enemas
- Nutritional plan (including adequate soluble and insoluble fiber intake and fluid intake) and regular bowel program essential

### Spasticity

- See Spasticity subsection in the Cerebral Palsy section
- Medications can interfere with sports or function; may have negative effects on alertness and muscle strength; medications are more successful in SCI than in brain-mediated injuries

### Osteoporosis

- Up to 50% decline in total bone content 10 years after SCI
- Causes: immobilization, marked increase in osteoclastic bone activity combined with only slight increase in osteoblastic activity, parathyroid hormone suppression, reduced absorption of calcium from gastrointestinal tract, possible vitamin D deficiency, and loss of active muscle traction effect
- Treatment: limb ROM, weight-bearing exercises including standing frame, lower extremity orthoses, treadmill walking with partial bodyweight support equipment, functional electrical stimulation, and bicycle ergometry

## LIMB DEFICIENCY

### History

- Surgeries related to type of amputation or limb difference (Figs. 14.3 to 14.5), surgical muscle transfers for functional improvement, and surgically implanted medical devices
- Medications: antispasmodics, tricyclic antidepressants, analgesics (may affect cognition), and other medications for comorbidities
- Comorbid medical issues:
  - Related to impairment: e.g., skin breakdown from prosthesis

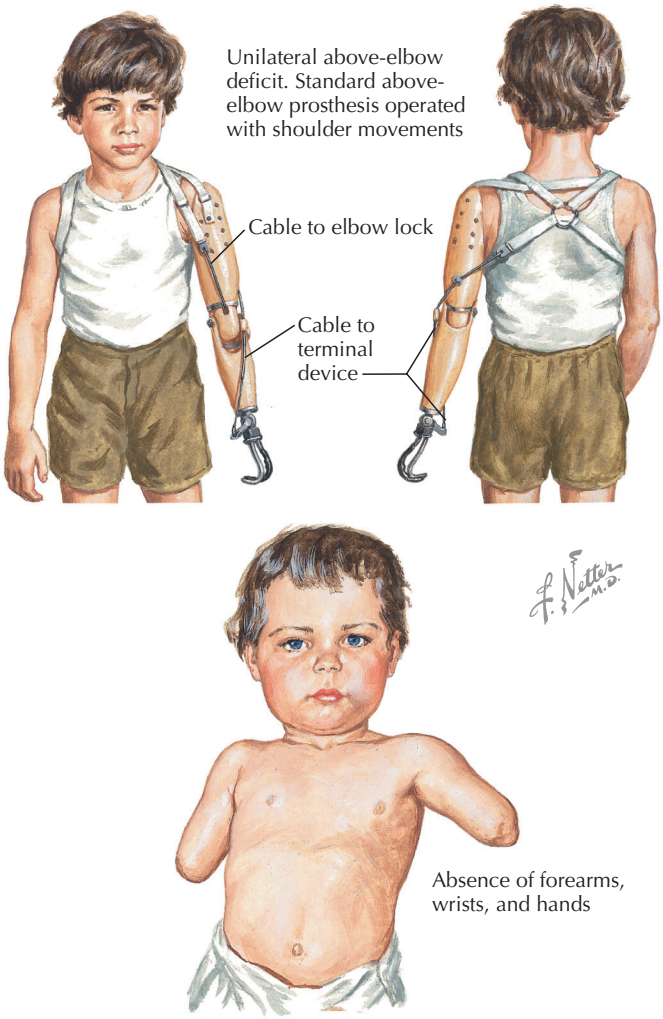


**Figure 14.3.** Congenital limb deficiency: hemimelia.

- Related to concurrent illness: e.g., traumatic brain injury, diabetes, visual impairment, low back pain, and cardiac disease
- Presence of phantom limb pain or sensation
- Level of functional independence: if multiple limb loss or difference, adaptive equipment needs for mobility
- Independence donning adaptive equipment; prosthesis (see Fig. 14.3)
- Adaptive equipment for sports-specific needs: prosthesis, orthosis, or sports equipment
- Prior training: environmental conditions, prior exercise program
- Level of aerobic and anaerobic conditioning, flexibility

## Physical Conditions in Athlete

- Skin breakdown: can occur with inappropriately fitting prosthesis, impact type of the sport at prosthesis–skin interface
- Treatment: achieving accurate fit (see Pressure Sores)

### Phantom Pain

- Phantom limb sensations or pain: occur within first few weeks after amputation in approximately 70% of individuals (Fig. 14.6)
- Theory: sudden lack of afferent input and cortical reorganization after amputation
- Medications: antidepressant and anticonvulsant drug classes; opioids such as tramadol; tricyclic antidepressants continue to be an initial choice; acupuncture and biofeedback (e.g., provide a reflection of the intact limb via a mirror box to allow patient to "see" and "move" the phantom limb) can be helpful

### Energy Expenditure

- Energy expenditure with LEA is greater than that in the able-bodied:
  - BKA: additional 16%–25% energy expended in ambulation
  - AKA: additional 56%–65% energy expended in ambulation
- Require good strength and balance on limb to better adapt to prosthetic component mismatch
- Quadriceps and hip abductors and adductors important for success with prosthesis

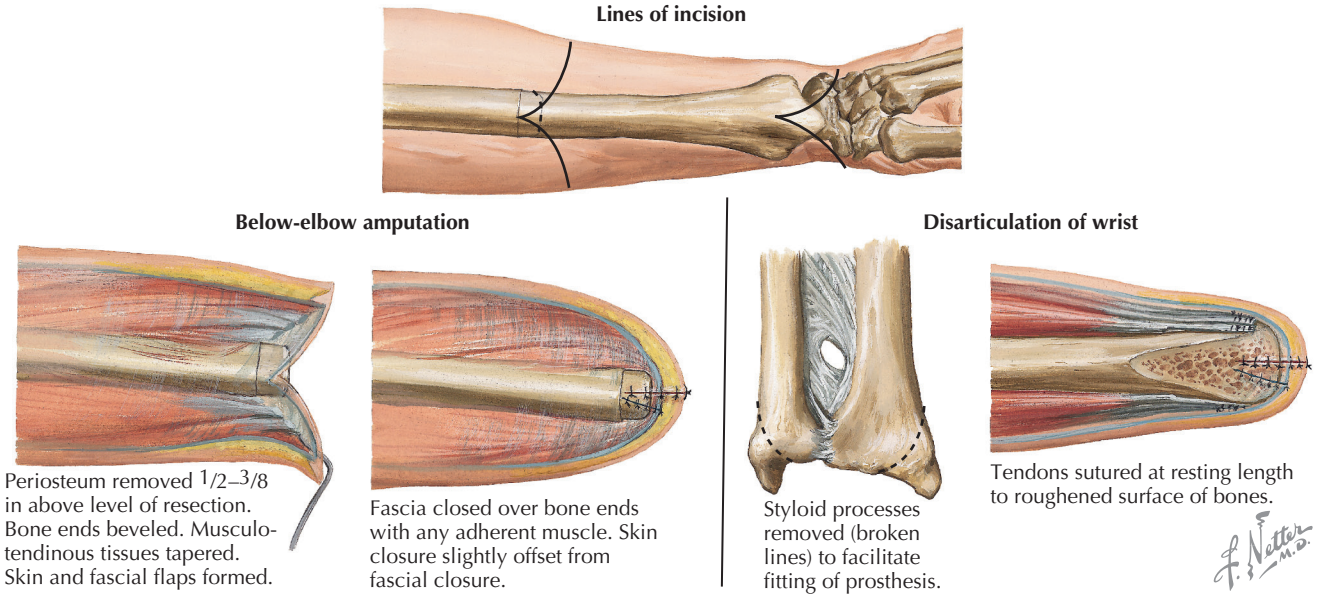


**Figure 14.4.** Amputation of the forearm and hand.

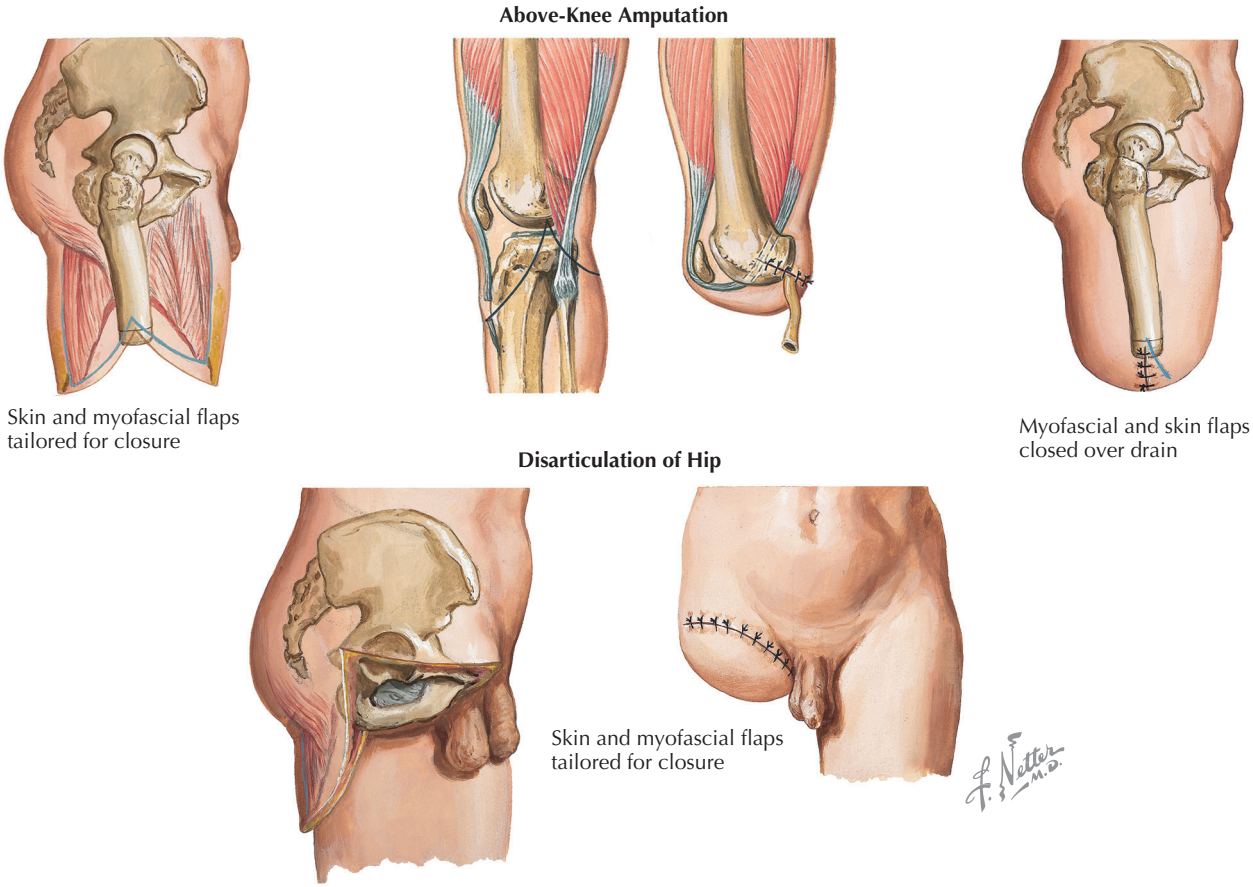


**Figure 14.5.** Above-knee amputation.

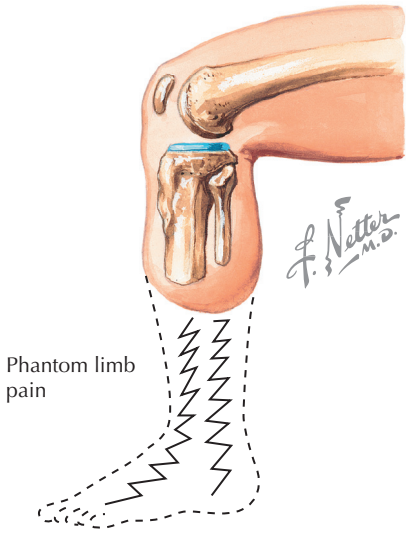


**Figure 14.6.** Complications of amputation.

### LEA Secondary Issues

- Low back pain: found in >50% of individuals with LEA
- Knee degenerative arthritis
- Knee pain: three times greater risk on the side contralateral to the side with AKA, twice greater risk with intact limb in BKA, five times *reduced* risk in prosthetic limb of the side with BKA

## CEREBRAL PALSY (CP)

Neurologic injuries may include persistence of primitive reflexes and posture-mediated movement patterns.

### History

- Surgeries related to injury: surgical muscle transfers for functional improvement, surgically implanted medical devices, and tendon-lengthening procedures
- Medications: antispasmodics, antiepileptics, tricyclic antidepressants, analgesics, medications for comorbid conditions such as cardiac disease
- Comorbidities related to:
  - Impairment: spasticity, nutritional support (e.g., gastrostomy tube), and seizures
  - Concurrent illness: e.g., diabetes, visual impairment, and cardiac disease
- Cognitive impairment: e.g., memory, calculation/organizational aspects
  - Intellectual disability *atypical for athletes* at international-level competitions
  - Impulsivity or risk-taking behavior may be present.
- Adaptive equipment: necessary for mobility or sports-specific equipment
- Level of functional independence: donning adaptive equipment, personal hygiene
- Level of independence in mobility: wheelchair, orthoses
- Prior training: environmental conditions, prior exercise program
- Level of aerobic and anaerobic conditioning, flexibility

## Glossary in Cerebral Palsy

**Spastic CP:** most common form; affects 70%–80%; increased muscle tone and stiffness; spasticity increases with excessive fatigue or anxiety
**Athetoid CP:** four limbs, trunk, and occasionally face; athetonia is slow, writhing involuntary muscle movement; muscle tone can be mixed: increased or decreased
**Ataxic CP:** four limbs and trunk, primarily decreased coordination of movement, also hypotonia; intention tremor present
**Diplegia:** lower limbs involved > upper limbs
**Hemiplegia:** upper and lower limbs on one side more involved
**Triplegia:** three limbs, usually both lower limbs and one upper limb involved
**Quadriplegia:** both upper and lower limbs involved

## Spasticity

- Pathophysiology unclear; theory is velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of stretch reflex; increased with nociceptive stimulus, such as UTI, distended viscera, and bowel obstruction
- Decision to treat spasticity depends on help (ability to ambulate) or hindrance (impedes independence with mobility and self-care; pain; pressure ulcers)

## Treatment

- Daily stretching to prevent contractures
- Appropriate wheelchair positioning or adaptive equipment helps decrease muscle tone.
- Oral medicine:
  - Baclofen: works centrally; high doses of baclofen require weaning-off period to avoid the risk of withdrawal seizures and hallucinations
  - Dantrolene: works peripherally on muscles; risks: rarely, liver toxicity; liver function laboratory tests obtained at treatment initiation and every 3–6 months during treatment
  - Tizanidine: works centrally; side effects with treatment initiation may decrease patient compliance: dizziness, dry mouth, and sedation
  - Gabapentin: works centrally; may require higher doses
- Baclofen infusion via an implanted pump in the abdomen
- Injections: phenol or botulinum toxin A into key spastic muscles (e.g., thigh adductors)
- Risks: chronic dysesthesia, pain, skin sloughing, peripheral edema, wound infection
- Surgery: tendon lengthening, muscle-release procedures for joint position issues
- Intention tremor (subset): medication options include primidone, propranolol, buspirone, and benzodiazepines and localized botulinum toxin A injections; cooling extremities with circulating cold wrap also reduces tremors 30 minutes after cooling
- Seizure control with medications
- Heterotopic bone formation: see Physical Conditions to Consider in SCI and Heterotopic Ossification

# LOCOMOTOR DISORDERS

## History

- Surgeries and medications utilized are similar to those used in CP (see Cerebral Palsy)
- Comorbidities related to:
  - Impairment: spasticity, type and success of bowel/bladder management program, restrictive lung disease (muscular dystrophy or neuromuscular condition), nutritional support (e.g., gastrostomy tube), seizures, and skeletal deformity (Fig. 14.7)
  - Concurrent illness: e.g., traumatic brain injury, diabetes, visual impairment, low back pain, cardiac disease, and respiratory illness
- Adaptive equipment, level of functional and ambulatory independence, and training (see Cerebral Palsy)

Typical rigid deformities of all four limbs seen in infant with arthrogryposis

Deformities of upper limbs in older child

**Figure 14.7.** Arthrogryposis multiplex congenita.

## Physical Considerations in Athlete

Spasticity, neurogenic bowel or bladder, seizure, joint contracture, pulmonary issues caused by muscle weakness

## Short Stature Syndrome

### Disproportionate

- Average-size torsos, unusually short limbs
- Causes: skeletal dysplasia or chondrodystrophy, caused by inherited or spontaneous gene mutations
- Spondyloepiphyseal dysplasia (SED) and diastrophic dysplasia: typically progressive kyphosis and/or scoliosis (Fig. 14.8); eye complications can be present in SED
- Can involve joint defects, limited ROM, and high incidence of joint dislocation

### Proportionate

- Overall unusually small size for age
- Causes: probable endocrine or growth hormone deficiency

## Muscular Dystrophy and Genetic Myopathies

### Overview

- Collection of progressive genetic muscle diseases
- Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD) most common types; DMD more severe in involvement

- Others include myotonic muscular dystrophy and facioscapulohumeral muscular dystrophy
  - Variation in muscle involvement and concurrent medical issues

### Diagnosis

- Muscle biopsy (for histologic features)
- Electromyography (EMG)
- Genetic/DNA testing (for dystrophinopathy) and basic laboratory tests (elevated creatine kinase)

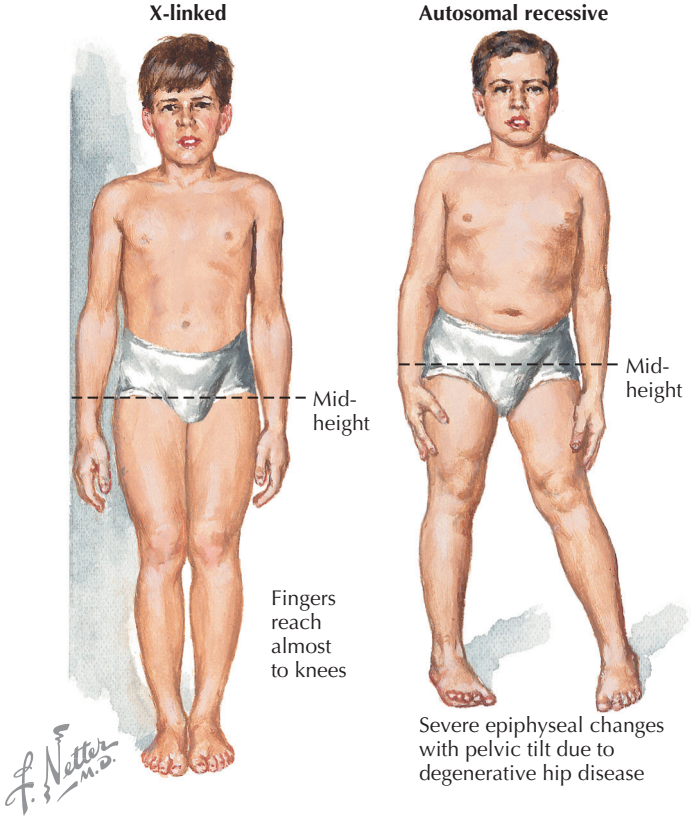


**Figure 14.8.** Spondyloepiphyseal dysplasia tarda.

### Management

- Multidisciplinary therapies (PT or OT) address specific functional deficits
  - Mild, noncontact physical activity such as swimming
  - Resistance training should be performed only in muscles with at least antigravity strength
  - Monitoring of physical and physiologic status: nutritional status (albumin), cardiac function (routine ECG, echocardiogram), and ventilatory function (pulmonary function tests—obtain before exercise prescription)
- Corticosteroids may help improve muscle strength and overall energy in DMD
- Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) may slow the progression of cardiomyopathy in DMD/BMD.
- Noninvasive (positive airway pressure) ventilator support and cough assist devices to help manage respiratory secretions—improve life expectancy and quality of life in DMD
- Novel therapies entering clinical trials: tailored to genetics of the disease (e.g., ataluren)

## Friedreich's Ataxia

Issues in balance, coordination, and vision can affect function in a competition.

**TABLE 14.1 VISUAL IMPAIRMENT RATINGS ACCORDING TO THE INTERNATIONAL BLIND SPORTS ASSOCIATION (IBSA)**

| | |
|---|---|
| B1 | Visual acuity < Logarithm of minimum angle of resolution (LogMAR) 2.6 |
| B2 | Visual acuity ranging LogMAR 1.5–2.6 and/or visual field constricted to diameter <10 degrees |
| B3 | Visual acuity ranging from LogMAR 1.4–1.0 and/or visual field constricted to diameter <40 degrees |
| B4 | National only—Visual acuity above 6/60 up to and including visual acuity of 6/24 (up to 25%); no visual field considered |

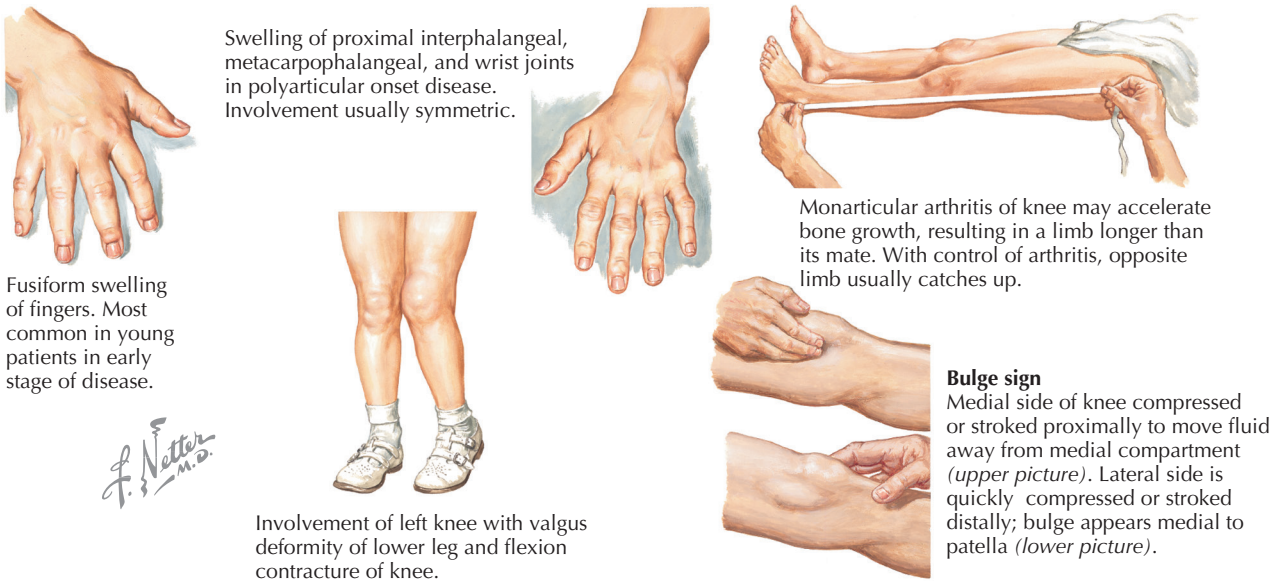


**Figure 14.9.** Joint issues in juvenile arthritis.

**TABLE 14.2 VISUAL IMPAIRMENT RATINGS ACCORDING TO THE IPC**

| | |
|---|---|
| T11 | Equivalent to B1; from no light perception in either eye to light perception, but inability to recognize the shape of a hand at any distance or direction. Athletes in this class are allowed to use guides in competition. |
| T12 | Equivalent to B2; ability to recognize objects up to a distance of 2 meters (i.e., below 2/60) and/or a visual field of <5 degrees |
| T13 | Equivalent to B3; ability to recognize contours 2–6 meters away (i.e., 2/60–6/60) and visual field of 5–20 degrees |

### Juvenile Rheumatoid Arthritis (JRA)

Joint impairment: *if* significant, athlete with juvenile rheumatoid arthritis (JRA) may qualify for competitions in the "les autres" category (Fig. 14.9)

## VISUAL IMPAIRMENT

- Classification assists fair competition by grouping athletes and determining eligibility
- Visual class: based on eye with better visual acuity and/or fields while wearing best optical correction
- The main alternative classification system to IBSA (B1–B3) (Table 14.1) is the T classification system, created by the IPC (Table 14.2).
- See http://www.ibsasport.org/classification/

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 15 CARE OF ATHLETES AT DIFFERENT LEVELS: FROM PEE-WEE TO PROFESSIONAL

Ashraf M. Elbanna • Kurt P. Spindler • Mark Schickendantz

## GENERAL CONSIDERATIONS (Box 15.1)

### Availability

- Being a team physician requires a tremendous time commitment.
- Must have a sincere desire to care for athletes and allow their expedient return to play

### Ability

- Possess the necessary skills to accurately diagnose and appropriately treat the athlete
- In addition to team physicians' *actual* ability, their *perceived* ability is important. One must portray a certain level of confidence when examining, treating, and discussing treatment options with an athlete.
- A certain level of comfort and familiarity comes with time; the more experience team physicians gain by covering sporting events and communicating with athletes, the more confident and competent they will become.

### Affability

- Possess the necessary social skills required to interact with not only athletes but also parents, coaches, and other staff members
- Nurturing a professional relationship with athletes will not only increase their confidence in their own ability but also increase their adherence to treatment guidelines, particularly if it conflicts with their personal desire to return to play at any cost.
- A team physician should not try to become best friends with an athlete and should maintain a professional relationship.

### Advocacy

- View the individual as a patient first and then as an athlete
- Always uphold your Hippocratic oath to do no harm to the patient

### Affiliation

- Surround yourself with those who have more experience, and learn from their prior experience

## DIFFERENCES FOUND AT EACH LEVEL OF PLAY

### Athletic Trainers

- **Pro** teams will have multiple certified athletic trainers (ATCs) on staff; most also employ a Medical Director, who oversees the Athletic Training staff. The team physician must understand the organizational hierarchy and establish appropriate communication so that decision making is performed by necessary individuals.
- **College** teams will typically have multiple ATCs, depending on the size of the school and available resources.
- **High School**/Grade school teams may not have an ATC at all, depending on available resources.
  - Responsibilities typically relegated to an ATC may fall upon the team physician's shoulders.
  - Must arrange appropriate follow-up for athletes who need to be supervised during the week if no ATC is available to do so

### Team Physicians

- **Professional** teams typically have a medical team structure that includes a Head Team Physician and several assistant Team Physicians (Table 15.1).
  - Most will also have a list of Consultants who provide specialty care, such as Ophthalmology and Cardiology.
  - With such a large staff, communication is critical. Ideally, the Head Team Physician is kept in the loop regarding the complete medical care of athletes, even if he/she is not the primary caregiver.
- **College** teams will also typically have multiple physicians responsible for the care of athletes; as mentioned earlier, collaboration and communication are paramount.
- At the **High School**/Grade school level, having only one physician is the most likely scenario.
  - Impossible to cover every single practice/training room/game; try to coordinate coverage with opposing team physician, if available

### Compensation

- At the **Pro** or **College** level, there may be some financial remuneration for your services. This typically is not substantial.
  - In several cases, your practice or the institution you are affiliated with pays for a marketing contract for the title of "official medical provider."
  - You may experience increased case volume based on either your presence on the sidelines as a team physician or the advertising associated with being the official medical provider.
- **High School**/Grade school level teams most often do not include any formal salary.
  - You may see an increase in surgical/clinical volume based on athletes operated on as well as friend and family referrals through word-of-mouth referrals from those affiliated with the team.
  - Do not expect this increased patient referral or surgical volume to be proportionate to the time and effort invested on your part to care for the team.
  - Even if you do not receive compensation, the team physician typically is not covered by a state's Good Samaritan laws. You must continue to uphold your ethical duty and provide the highest level of care possible irrespective of compensation.

### Public Scrutiny

- **Pro:** Team physicians may often face public pressure to try the latest fads in the medical field. They may also face this same pressure from the organization, coach, or the player. The physician has the autonomy to refuse to provide care that he or she

**BOX 15.1 THE FIVE AS OF BEING AN EXCELLENT TEAM PHYSICIAN**

- Availability
- Ability
- Affability
- Advocacy
- Affiliation

**TABLE 15.1 NUMBER OF PHYSICIANS PER TEAM PER COMPETITION LEVEL**

| Physician Type | High School | Collegiate | Professional |
|---|---|---|---|
| Orthopedic | 1.1 | 2.3 | 2.2 |
| Nonorthopedic | 0.2 | 1.1 | 1.9 |

From Makhni E, Buza J, Byram I, MD, Ahmad C. Academic characteristics of orthopedic team physicians affiliated with high school, collegiate, and professional teams. *Am J Orthop.* 2015;44(11):510-514.

does not deem medically appropriate. However, physicians should counsel the athlete regarding potential risks and benefits and encourage them to make an informed decision.
  - The outcome of the athlete, whether positive or negative, may have a huge impact on public perception of the physician's abilities. Although it is normal for physicians to be cognizant of the fact that their decisions will face public scrutiny, they should not be so overly concerned with this fact that it alters their decision-making process.
- **College:** May be similar to the pros depending on the prestige/ranking of the school. Division I schools in large power conferences will be more like the professional level, whereas Division II and III schools are more similar to high school.
- **High School/Grade school:** Typically, physicians will not experience public scrutiny that is commonplace at the college/professional level.

## Medical Resources

- **Pro:** Very low threshold for obtaining advanced imaging (typically MRI); no need to deal with insurance before authorizations typically needed for MRI or surgery; no shortage of supplies in the training room; adequate space available for examinations/treatment
- **College:** Depending on resources/funding of the program, this may vary considerably. Athletes will need to go through their insurance provider to obtain imaging, or to schedule surgery; advanced imaging is ordered with more discretion than at the pro level. Some schools may have insurance plans available for athletes who do not have prior coverage or are coming from overseas.
- **High School/Grade school:** Again, may considerably vary depending on school funding/resources or public versus private; not atypical for several inner city schools to have a shortage of supplies. Athletes often purchase their own tape and other medical supplies for use.

## Patient Autonomy/Decision Makers

- **Pro:** At the professional level, the team physician is responsible for providing team management with accurate medical information about the athlete. With regard to the athlete, it is imperative that the team physician clearly outline the risks, benefits, and alternatives to treatment so that the athlete can make an informed decision. Often, an athlete will ask for specific recommendations, wanting the physician to simply "do what you think is best." It is the responsibility of the team physician to always do what is in the best interest of the player and not the team.
- **College level:** Physicians have the authority to clear for participation/bench a player. Athletes themselves have the legal right and authority to make their own treatment decisions. They should be given all the information necessary to make an informed decision and should be counseled regarding potential conflicts between short- and long-term gains (i.e., if diagnosed with a potentially repairable meniscal tear, they should be told about short- and long-term outcomes, risks, and length of convalescence with both meniscectomy and repair).
- **High School**/Grade school team physicians have the ability to clear/bench a player. If under the age of 18 years, an athlete's parents have the final say regarding medical decisions/treatment options.

## Immediate Return to Play

- **Pro:**
  - At the professional level, judicious use of local anesthetic to allow immediate and safe return to play is indicated for selected conditions, such as low-grade acromioclavicular sprain. It is important that the team physician understand the league rules regarding use of other injectable agents such as Toradol.
- **College:**
  - May consider local anesthetic injections (not in knee, ankle, hip or shoulder [i.e., glenohumeral]), or possibly even ketorolac, to allow short-term participation with relatively minor injuries (e.g., AC separation or back spasms)
  - If the athlete has a suspected concussion and passes sideline testing, they may potentially be able to return to play once concussion is ruled out.
- **High School/Grade school:**
  - Rarely appropriate to administer injections of local anesthetic agents
  - Even the suspicion of a concussion warrants immediate removal, with no potential for return to play

## Drugs in Sports Appropriate at All Levels

- Physicians must discourage the use of illegal substances at all levels of play. Moreover, they should be familiar with the list of banned substances at each level of play: Pro, College, or High School/Grade school
- The physician should obtain a thorough history of all substances being used by an athlete, including natural supplements, and should encourage athletes to be honest and forthcoming regarding their use of all substances.
- The physician must encourage compliance with governing bodies at each respective level, in terms of participation and requirements for drug testing, and enforcement of all policies.

## Confidentiality

- Team physicians should be familiar with Health Insurance Portability and Accountability Act (HIPAA), and Federal Educational Rights and Privacy Act (FERPA); the latter pertains to the privacy of educational records/medical information that is part of a student athlete's health record.
- **Pro:** Professional sports teams have HIPAA forms that clearly outline who the athletes' job-related medical information will be shared with. All athletes are required to sign this document.
  - There is a distinction made between personal and team-related medical information at the professional level. The

physician has a duty to communicate any relevant team-related medical information with the organization that may have an impact on an athlete's participation.

- **College:** If the physician is employed by the school, medical information is not considered Protected Health Information (PHI); however, if the school directly receives any federal funding, the medical information is governed by FERPA. Technically, medical information that is not electronically stored is not governed by HIPAA; this would then allow physicians to communicate with coaches and trainers on the sideline regarding a player's assessment. Several schools now require athletes to sign a waiver before season play, which permits sharing of their medical information with coaches or ATCs.
- **High School/Grade school:** Parental consent must be obtained before sharing medical information of athletes aged <18 years. However, a physician also has an obligation to prevent harm to other players on the team, which may arise with certain infectious diseases.

## Second Opinions

- **Pro:** Rules related to second opinions vary from league to league. Level of play may also be a factor. For instance, Minor League professional baseball players are considered employed at will and as such have less autonomy than their Major League counterparts regarding medical care outside of the organization.
  - One should not take offense if an athlete chooses to pursue surgery or seek other forms of treatment from a different provider. Particularly at the professional level, several athletes may have pre-existing relationships with orthopedic surgeons, and they may choose to continue their care with them. If the athlete so desires, the team physician should facilitate second opinions and also share medical information with other healthcare professionals.
- **College** or **High School/Grade school:** Typically, an athlete will only seek external care in the form of surgery or imaging if their insurance provider does not allow care at their own institution.

## Team Travel/Cross Coverage

- **Pro:** Depending on the sport, physicians may or may not travel with the team. Note that for sports in which physicians do not travel, you may also be responsible for providing coverage to the opposing team. In such situations, do not directly approach players (except in cases of medical emergency). Always interact with the ATC of the opposing team, who will make the decision regarding whether or not they would like you to evaluate a player.
- **College:** May or may not travel; may share responsibilities with other physicians; important to develop a schedule before the season starts so that any lapses in coverage can be promptly remedied; be cognizant of laws when crossing state lines. A study conducted in 2012 revealed that 18 of the 54 medical boards (that responded) allowed physicians travelling with their team to practice medicine with their home-state license. The remaining 36 states (67%) do not provide a legal pathway for the practice of medicine without their state license. This may come into consideration if an unfortunate circumstance of malpractice claim arises (Fig. 15.1).
- Controlled substances should not be distributed outside of the physician's local jurisdiction, where their DEA permit is valid. Arrangements can be made with the host's team physician to provide controlled substances, if needed.
- When travelling to an outside venue, discuss emergency planning, including the location of AEDs or nearest ER, with the host team.

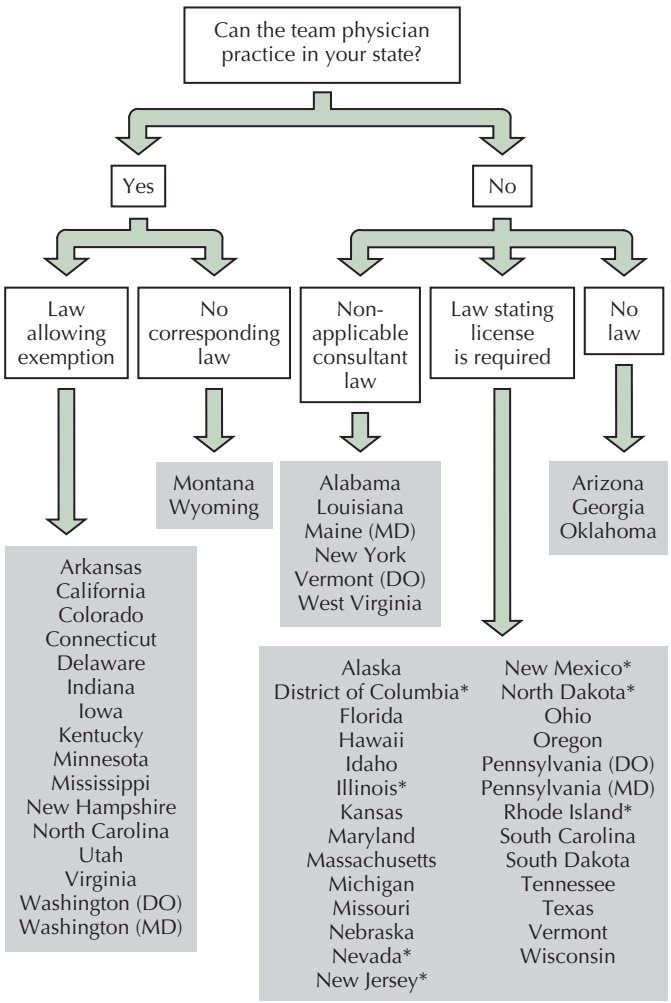


**Figure 15.1.** State licensure requirements, responses by the medical board. Asterisks (*) indicate information obtained from website. (From Viola T, Carlson C, Trojian TH, Anderson J. A survey of state medical licensing boards: can the travelling team physician practice in your state? *Br J Sports Med.* 2013;47[1]:60-62.)

- **High School/Grade school:** Typically, travel is limited to within state borders; similar understanding of local on-the-field and medical care in the community should be assessed.

## Factors to Consider Regarding Timing of Surgery, Return to Play

- **Pro:** What position does the athlete play? In season or off season? At what point in the season are you, nearing playoffs, etc. What is the team record? At what point in their career is the athlete?
- **College:** Does the athlete play more than one sport? More than one position? Do they have aspirations to turn pro? Is it possible/wise for them to red shirt this season due to an injury/surgery?
- **High School/Grade school:** Does the athlete play multiple sports, multiple positions? Are they in club teams year round? What year in school are they? Do they plan on playing in college? Is a scholarship at stake? Will they be cleared to play at the next game that may have college scouts present?

## SUMMARY

- Team physicians will often be faced with ethical dilemmas regardless of the level of participation.
- These conflicts frequently revolve around return to play, confidentiality, patient (athlete) autonomy, and informed consent.
- The team physician must, at all times, act upon their ethical duty to provide the highest level of care possible to the athlete.
- In situations where the physician's obligation lies to a team/organization, they must at no time pursue a course of action that they reasonably believes could lead to any harm to the athlete, either in the near or distant future.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 16 THE WILDERNESS ATHLETE AND ADVENTURER

Aaron D. Campbell

## INTRODUCTION

- Wilderness sports differ from traditional sports in several ways.
- Participation levels of athletes may vary: professional, sponsored, and paid athletes; athletic individuals and adventurers; and intermittent recreational participants.
- The setting of wilderness sports is often austere, remote, and not regulated by organized bodies as traditional sports are.
- Participants range in age from children to older adults and seniors.
- Typically, there is no distinction in participation between male and female participants in wilderness sports.
- Competition does occur but is often an individual endeavor to achieve personal best.
- Other important distinctions are that wilderness sports are typically not team sports, except for certain exceptions such as team-based overland adventure races and the team concept that applies to accomplishing common goals such as in climbing sports/events.
- Participants may be those with disabilities, pregnant females, or individuals who may have chronic health conditions, particularly older adults, who may be affected by conditions or environments where they occur (e.g., wilderness sports/adventures occurring at high altitudes).
- Although it is difficult to quantify participation in wilderness sports participation, primarily because of lack of tracking data, overall participation has increased in recent years.
- Approximately half of all Americans participated in at least one outdoor activity in 2014, which equates to over 141 million individuals collectively participating in approximately 12 billion outdoor outings.
- Increase in ski and snowboard sales, including backcountry equipment, as well increase in the sales and development of companies in the climbing and mountain biking industries support the increasing participation in wilderness and adventure activities.
- Accordingly, providers must understand how wilderness sports differ from traditional sports, particularly in order to evaluate and advise participants in the setting of a preparticipation evaluation (PPE) and to understand and manage injuries and illnesses occurring as a result of the activities.
- International travel or remote domestic travel is common in wilderness sports/adventures and calls for unique considerations:
  - Travel insurance is available for international travel.
  - Insurance is also available, as are GPS-locating devices, in the event where search and rescue services are warranted.
  - Travel-specific disease prevention utilizing preventive practices, vaccines, and occasionally medications (Traveler's Health at cdc.gov) is helpful.
- While it is difficult to comprehensively enlist all wilderness and adventure activities, examples of general categories of wilderness sports are illustrated in Table 16.1.

## TERMINOLOGY

**Wilderness**: A wild and uncultivated region such as a forest or desert, which is generally uninhabited or inhabited only by wild animals, and in which travel by land or water by humans can be considered a sport or adventure

**Backcountry**: Refers to areas outside of controlled borders in wilderness regions, such as out of bounds at ski resorts for skiing/snowboarding or, perhaps, areas that could be out of cell phone or Internet range; can be applied to any wilderness sport

## PREPARTICIPATION EVALUATION FOR THE WILDERNESS ATHLETE AND ADVENTURER

### Overview

- Preparticipation evaluation (PPE) is an evaluation of athletes or sports participants before participation in a sport, an event, or an adventure.
- PPE is common in traditional and organized sports, specifically high school or collegiate sports, where it is often required despite limited supporting evidence.
- PPE for wilderness sports is performed less frequently compared with that for traditional sports, but recent literature offers guidance and methodology for clinicians who perform PPEs for wilderness sports.
- Common reasons an athlete or adventurer may request a PPE may include a requirement from an agency or a guide service, a training course in outdoor education or wilderness skills, or individually requested based on personal goals and/or medical conditions.
- Examples of important considerations for PPE for the wilderness athlete/adventurer are outlined in Table 16.2.
- Laboratory data may be desired to ensure stability and optimization of chronic health conditions.
- Moreover, specialty consultation may be helpful in assessment of cardiovascular health (e.g., exercise treadmill stress test), pulmonary function and capacity, and gastrointestinal or musculoskeletal health.

### Setting, Timing, and Structure of a Wilderness Sports/Adventure PPE

- Can be office-based or conducted in a private setting; sometimes Internet or phone consultation
- Ample duration should be allowed before the event for completion of immunizations and additional evaluation, which may include further diagnostic testing. A minimum of 6 weeks is mostly sufficient.
- Providers should be prepared to perform a wilderness PPE, borrowing principles from traditional sports PPE and modifying with variables such as unique preparticipation planning, prolonged physical and mental demands, and other extrinsic challenges such as environment, regional location, and remoteness.
- As in most health evaluations, medical history is the most important factor during PPE for wilderness sports.
- Physical examinations may be more important in the presence of chronic health conditions and previous illness or injury associated with wilderness adventures.

### Ethical, Legal, and Administrative Considerations of a Wilderness Sports/Adventure PPE

- Should follow typical Health Insurance Portability and Accounting Act (HIPAA) procedures

**TABLE 16.1 EXAMPLES OF GENERAL CATEGORIES OF WILDERNESS SPORTS***

| Wilderness Event Category | Sport/Adventure |
|---|---|
| Climbing sports | Rock<br>Ice<br>Mixed rock and ice<br>Mountaineering/alpinism |
| Snow sports | Backcountry skiing<br>Backcountry snowboarding<br>Glacier travel |
| Water sports | River running (kayaking or rafting)<br>Surfing<br>Open water swimming |
| Cycling sports | Mountain biking |
| Endurance/trekking events | Hiking<br>Backpacking<br>Trekking<br>Trail running (ultra-marathons)<br>Overland adventure racing |

*Note: This is not an inclusive list.

- Utilized as an evaluation for a planned event but should also incorporate typical features of a wellness visit, including immunizations, status of chronic conditions, and assessment of medications
- Literature is available to guide inexperienced providers, but referral to a trained provider may be valuable in more challenging medical and environmental scenarios.
- Specialty consultation may be pertinent to further evaluate chronic health conditions such as heart disease (may need an ECG or stress test), pulmonary disease (may need pulmonary function tests, including provocative exams), and gastrointestinal disease (may need updated colonoscopy or unique dietary plans).
- See eForm 16.1 for an example of a health history form that could be used by a provider performing a PPE for wilderness sports or adventures.

## CHRONIC HEALTH CONDITIONS AND WILDERNESS SPORTS AND ADVENTURES

### Overview

- In general, key principles involve optimization and/or stabilization of chronic conditions before wilderness travel, sports, or adventures.
- Ensure access to medications used regularly or for disease exacerbations and include adequate supply for the same number

**TABLE 16.2 SIMPLIFIED LIST OF IMPORTANT CONSIDERATIONS FOR PPE OF A WILDERNESS ATHLETE OR ADVENTURER**

| Consideration | Rationale for Consideration |
|---|---|
| Travel | • Domestic vs. international will yield different health concerns<br>• Insurance policies for health and emergency issues abroad are available |
| Immunizations | • Important for international travel in endemic areas as well as in certain domestic areas |
| Chronic medications | • Additional supplies or pharmacy refills prior to travel depending on location and event demands |
| Prophylactic medications | • Helpful in both endemic international areas as well as certain domestic remote backcountry areas<br>  • Antibiotics<br>  • Pain management<br>  • Wound care<br>  • General illness (e.g., diarrhea, nausea/vomiting, and rashes) |
| General physical conditioning | • Fitness and conditioning base vs. need for specific activities in a healthy athlete/adventurer |
| Nutrition | • Adequate caloric replacement for the desired activity, considering lightweight, easily tolerated foods that include elements of healthy fats, carbohydrates, and proteins<br>• Other nutritional supplementation (e.g., vitamins) may be considered given the nature of prepared or dehydrated foods and inability to carry perishables or heavy meat products |
| Altitude | • Acclimatization, history of altitude illness, weather, and elemental exposure |
| Hot environments | • Heat exposure issues<br>  • History of heat illness<br>  • Cooling abilities<br>  • Sun protection<br>  • Hydration |
| Cold environments | • Cold exposure issues<br>  • History of cold illness<br>  • Frostbite<br>  • Layered clothing<br>  • Prevention of heat loss and sweating |
| Presence of chronic disease | • Stabilization/optimization of health conditions<br>• May need medical specialty consultation<br>• Diagnostic data during PPEs may be helpful in certain cases such as evaluation of iron levels before travel to altitude and assessment chronic disease stability (e.g., diabetes, CAD, valvular heart disease, COPD, and thyroid, hepatic, or renal disease) |

*CAD*, Coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *PPE*, preparticipation evaluation.

**TABLE 16.3 SIMPLIFIED LIST OF CHRONIC CONDITIONS AND RATIONALE FOR ISSUES IN WILDERNESS SPORTS/ADVENTURES**

| Organ System | Rationale for Health Issue |
|---|---|
| Pulmonary | • Airway is the primary organ system to sustain life.<br>• Multiple risk factors and health conditions affect airways.<br>• Allergens and air quality can lead to exacerbation of chronic conditions or trigger new acute symptoms.<br>• Asthma, COPD, and/or emphysema should be stable with available medications, along with instructions for management of acute exacerbations. |
| Cardiovascular | • Stress on the heart: conditions such as altitude, temperature extremes, and physical exertion can trigger chronic cardiac conditions such as ischemic heart disease, occult vessel lesions, arrhythmias, and pump deficits |
| Hematologic | • Patients with injuries sustained in wilderness areas may be at an increased risk if participants are using blood-thinning agents such as warfarin.<br>• Genetic bleeding disorders can pose issues for hemostasis in the event of lacerations, abrasions, or cuts.<br>• Blood clotting disorders may go unrecognized and possible delay in obtaining imaging results or in initiation of anticoagulation could be life threatening. |
| Gastrointestinal | • Infectious or food-borne issues leading to fluid loss through diarrhea or vomiting or the inability to eat can lead to dehydration or nutritional deficiencies.<br>• Counseling on food and toilet hygiene should be emphasized to prevent acute issues.<br>• Chronic conditions should be stable and medications must be available for exacerbations. |
| Musculoskeletal | • Use of the musculoskeletal system is inherent in physical movement. Injuries can lead to difficulties with ambulation or in performing essential tasks for completion of the sport or event or self-extrication.<br>• Joint laxities such as recurrent shoulder dislocations should be evaluated and managed with physical therapy and possible surgical invention.<br>• Conditions such as arthritis (any kind) could pose issues if pain or systemic exacerbations occur. |
| Central nervous | • Head injuries or spinal compromise can be life threatening or require prompt recognition for early and appropriate management to allow either resolution or immobilization for transport or extrication.<br>• Concussion management should be discussed in terms of recognition and management to avoid long-term complications.<br>• Disorders such as multiple sclerosis or Parkinson's disease could be exacerbated by heat or altitude; thus, thermoregulation is an important consideration. |
| Ophthalmologic | • Visual disturbances can compromise abilities to navigate and care for oneself during wilderness sports/adventures.<br>• History of refractive surgeries (LASIK, radial keratotomy) or glaucoma: vision may be affected by altitude or other barometric pressure changes<br>• Corrective eyewear should be available in multiple forms to avoid loss of visual acuity. |
| Psychiatric | • Psychotic episodes can be triggered in unfamiliar terrain or when threats are perceived from the environment and can compromise personal or expedition member safety.<br>• Coping skills mechanisms, response to stress, and stress management are essential.<br>• Concerns regarding substance abuse or medication withdrawal can be life threatening or lead to issues with other participants. |

*COPD*, Chronic obstructive pulmonary disease; *LASIK*, laser-assisted in situ keratomileusis.

of days of sojourn and additional 2–3 days of supply (or more) depending on potential travel itinerary changes, possibility of event prolongation, or loss of medication.

- Certain conditions may be affected more by specific environments, such as cold or hot temperatures or high altitudes, and others are more generalized.
- Some individuals with chronic health conditions or unexplained signs and symptoms may require more advanced diagnostic evaluation depending on the activity, duration of event, or required intensity (see the PPE section above).
- Table 16.3 illustrates a simplified list of chronic conditions and rationale for related issues experienced in wilderness sports/adventures.

## ALTITUDE-RELATED ISSUES

### High-Altitude Illness (HAI)

- Spectrum of illnesses that are specifically caused by rapid ascent to high altitude
- Includes acute mountain sickness (AMS), high-altitude cerebral edema (HACE), and high-altitude pulmonary edema (HAPE)
- The Wilderness Medical Society (WMS) organized an expert panel to produce evidence-based, comprehensive consensus guidelines for prevention and treatment of acute altitude illness. Most information presented in this section is adopted from these guidelines, which are regularly updated.
- High-altitude illness (HAI) can be ameliorated by low-risk ascent profiles or a well-planned and appropriately controlled ascent.
- Ascent profiles describe the number of days planned to achieve a particular altitude, where the sleeping altitude is most important, and several days may be required to achieve a maximum altitude with scheduled camps for sleeping along the way in order to avoid complications of HAI.
- Common signs and symptoms of HAI are enlisted in Table 16.4.
- Risk categories established for HAI are outlined in Table 16.5.
- Medications available for prevention of HAI are outlined in Table 16.6.

**TABLE 16.4 SIGNS AND SYMPTOMS OF HIGH-ALTITUDE ILLNESSES**

| High-Altitude Illness | Symptoms | Signs |
|---|---|---|
| Acute mountain sickness (AMS) | Headache plus one or more of the following signs varying from mild or moderate to severe:<br>• Nausea or vomiting<br>• Fatigue<br>• Lassitude<br>• Dizziness<br>• Sleep difficulty | Nothing specific |
| High-altitude cerebral edema (HACE) | Worsening symptoms of AMS, considered upper end of severe AMS<br>• Severe lassitude | • Ataxia<br>• Altered mental status<br>• Encephalopathy |
| High-altitude pulmonary edema (HAPE) | • Extreme fatigue<br>• Breathlessness at rest<br>• Chest tightness, fullness, or congestion<br>• Drowsiness | • Fast shallow breathing<br>• Cough with possible pink frothy sputum<br>• Gurgling and rattling breath sounds<br>• Blue or grey lips or fingertips |

Modified from Luks AM, McIntosh SE, Grissom CK, et al. Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness. *Wilderness Environ Med.* 2010;21(2):146-155.

**TABLE 16.5 RISK CATEGORIES FOR HIGH-ALTITUDE ILLNESSES**

| Risk Level | Ascent Vignette |
|---|---|
| Low risk | • No previous history of altitude illness and plans to ascend no more than 2800 m (9186 ft) as the maximum altitude for sleeping<br>• Plans to ascend to 2500–2800 m (8202–9186 ft) in no less than 2 days with increases in sleeping altitude afterward of no more than 500 m/day (1640 ft) |
| Moderate risk | • Past history of AMS and plans to ascend to altitudes of 2500–2800 m (8202–9186 ft) in a single day<br>• No past history of AMS but plans to ascend to >2800 m (9186 ft) in a single day<br>• Any individual with plans to increase their sleeping altitude by >500 m (1640 ft) in a single day at altitudes >3000 m (9842 ft) |
| High risk | • Previous history of AMS and plans to ascend to >2800 m (9186 ft) in a single day<br>• An individual who has experienced HAPE or HACE previously<br>• An individual planning to ascend to >3500 m (11,482 ft) in a single day for the first sleeping elevation<br>• An individual who increases their sleeping altitude by >500 m (1640 ft) while over 3500 m (11,482 ft)<br>• Shorter-duration ascents of high-altitude peaks where typical ascent plans involve rapid gains in altitude, possibly due to mild levels of technical terrain |

Modified from Luks AM, McIntosh SE, Grissom CK, et al. Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness. *Wilderness Environ Med.* 2010;21(2):146-155.

- For treatment of all HAIs, considerations include rest, medications (Table 16.6), descent, and oxygen.

#### Acute Mountain Sickness (AMS) (see Tables 16.4, 16.5, and 16.6)

- Symptoms can be mild, moderate, or severe.
- All symptoms include headache plus one or more additional symptoms ranging in severity.
- Prevention is based on a low-risk ascent profile, possibly a moderate-risk ascent profile if no history of AMS.
- In case of history of AMS, an individual with altitude experience and known elevations of symptom presentation can slow his/her ascent to altitude and/or increase the number of sleeping nights during ascent.
- Consider medications for prophylaxis in moderate- to high-risk situations

#### High-Altitude Cerebral Edema (HACE) (see Tables 16.4, 16.5, and 16.6)

- Considered to be a severe form of AMS
- Signs of CNS disturbance accompanied with severe symptoms of AMS
- Consider medications for prophylaxis in moderate- to high-risk situations

#### High-Altitude Pulmonary Edema (HAPE) (see Tables 16.4, 16.5, and 16.6)

- Hypoxia of altitude can lead to constriction of certain pulmonary blood vessels, shunting blood to fewer other blood vessels, which leads to increased pulmonary pressure and subsequent leakage of fluid to lung parenchyma.
- Contributing factors beyond the hypoxic environment at high altitudes are increased exertion and cold temperatures.
- Consider medications for prophylaxis for moderate- to high-risk situations

## Health Conditions Affected by Altitude

### Lung Disease and Altitude

- See Chapter 23: High-Altitude Training and Competition for discussion of the physiology of high altitude and definitions of elevation levels.
- Chronic lung disease may be worsened at high altitudes.

**TABLE 16.6 MEDICATIONS COMMONLY USED FOR THE PREVENTION AND TREATMENT OF HIGH-ALTITUDE ILLNESSES IN ADDITION TO A GRADUAL ASCENT RATE**

| High-Altitude Illness | *Prevention in Moderate- to High-Risk Situations | ****Treatment |
|---|---|---|
| Acute mountain sickness (AMS) | • **Acetazolamide 125 mg oral twice daily<br>Pediatrics: 2.5 mg/kg oral every 12 hours<br>• ***Dexamethasone 2 mg oral every 6 hours or 4 mg oral every 12 hours<br>Pediatrics: not recommended | • Acetazolamide 250 mg oral twice daily<br>Pediatrics: 2.5 mg/kg oral every 12 hours<br>• Dexamethasone 4 mg (oral, IV, or IM) every 6 hours<br>Pediatrics: 0.15 mg/kg/dose every 6 hours until symptoms subside |
| High-altitude cerebral edema (HACE) | • Acetazolamide 125 mg oral twice daily<br>Pediatrics: 2.5 mg /kg every 12 hours<br>• Dexamethasone 2 mg (oral, IV, or IM) every 6 hours, or 4 mg every 12 hours<br>Pediatrics: not recommended | • Dexamethasone 8 mg (oral, IV, or IM) loading dose, then 4 mg every 6 hours<br>Pediatrics: 0.15 mg/kg/dose every 6 hours |
| High-altitude pulmonary edema (HAPE) | • Nifedipine 30 mg SR oral every 12 hours or 20 mg SR oral every 8 hours<br>• Tadalafil 10 mg oral twice daily<br>• Sildenafil 50 mg every 8 hours<br>• Salmeterol 125 mcg inhaled twice daily | • Nifedipine 30 mg SR oral every 12 hours or 20 mg SR oral every 8 hours |

*For the general population, prophylactic medications should not be used in place of a gradual ascent rate and such medications are not typically needed in low-risk situations (refer to Table 16.6). In addition, prophylactic medications can be discontinued upon initiation of descent.
**Acetazolamide is a sulfonamide derivative; however, cross reactivity to sulfonamides is rare, and individuals reporting an allergy to sulfonamide medications should consider a trial of acetazolamide before the expedition. Anaphylaxis from sulfonamide medications is an absolute contraindication to the use of acetazolamide.
***Dexamethasone should be limited to no more than 10 days to prevent glucocorticoid toxicity and adrenal suppression.
****Continue until definitive care is reached at lower altitudes.
Modified from Luks AM, McIntosh SE, Grissom CK, et al. Wilderness Medical Society consensus guidelines for the prevention and treatment of acute altitude illness. *Wilderness Environ Med.* 2010;21(2):146-155.

- Etiology may include increased alveolar hypoxia, hypoxemia, increased pulmonary vascular resistance, and sleep-disordered breathing issues.
- Work of breathing increases due to increase in ventilation at high altitudes.
- If individuals are using supplemental oxygen at low altitudes, it is recommended they discuss how to use supplemental oxygen at high altitudes with their healthcare provider during a PPE because it may be different from their baseline use.

### ASTHMA

- The same management principles at low altitudes apply at high altitudes as well.
  - Asthmatics often do better at high altitudes due to a decrease in allergens and air pollution.
  - Exercise-induced bronchospasm may worsen at elevations over 2500 m (approximately 8200 ft) owing to large-volume ventilation of dry cold air and subsequent heat loss.
  - Participants should be advised to use inhalers and medications consistent with their routine asthma treatment regimen, although long-acting beta-agonists may be considered for EIB and prolonged event duration at high altitudes.

### COPD

- COPD should be optimized and stabilized before altitude sojourns, with instructions to continue scheduled inhaled medications and monitor oxygen saturation levels by using a pulse oximeter.
- Exercise tolerance and capacity should be determined for individuals with COPD participating in adventure and wilderness events requiring exertion that exceeds baseline activities.
- Patients with severe (FEV1 30%–50% predicted) or very severe disease (FEV1 <30% predicted) or those with carbon dioxide retention or right heart failure should be advised against exceeding baseline activity tolerance and ascending to an altitude higher than that of their current residence.
- Access to corticosteroids in addition to inhalers and counseling on how to manage COPD exacerbations is essential.
- Descent will always be an intricate part of the initial management in any COPD exacerbation.

### SLEEP-DISORDERED BREATHING

- Sleep-disordered breathing at altitude refers to both obstructive sleep apnea (OSA) and central sleep apnea (CSA).
- In general, OSA may improve at altitude because of increased muscle tone in upper airways.
- Individuals with OSA can develop CSA, or CSA can be worsened by altitude.
- Acetazolamide, a diuretic, is a favorable medication for preventing exacerbations of both OSA and CSA, whether or not continuous positive airway pressure (CPAP) is used.
- Participants with moderate to severe OSA should plan to bring and use their CPAP machines while at altitude, and battery/power will have to be considered.

## Cardiovascular Conditions

### HYPERTENSION

- A mild increase in blood pressure normally occurs with ascent to higher altitudes.
- Individuals with hypertension can observe considerable variability in systolic blood pressure increases at altitude (usually >3000 m or 9840 ft), with increases as high as 30–40 mmHg.
- Altitude-induced changes are transient and will resolve soon after descent to lower elevations.
- Owing to the transient nature of these changes, frequent monitoring of blood pressure is neither advised nor does it need to be treated in asymptomatic individuals.
- If symptoms develop while at altitude, no ideal treatment plan has been established except for continuation of routine dosing of regular medications and descent or perhaps extrication (depending on severity).

- Certain clinicians may recommend medications for pro re nata (PRN) use for significant blood pressure increases, but this should be individualized, and an action plan should be discussed with the healthcare provider during a PPE.
- For individuals with labile HTN, monitoring with a reliable, portable wrist cuff may be helpful, and use of nocturnal supplemental oxygen can improve the variation in blood pressures.

### ATHEROSCLEROTIC HEART DISEASE

- Atherosclerotic heart disease (ASHD) is a common cause of sudden cardiac death in older athletes.
- These individuals will most likely self-select their participation in altitude activities based on exercise capacity and the presence of symptoms.
- Important considerations for risk assessment include identifying cardiovascular risk factors and known disease status and stability, baseline exercise capacity, and anticipated exercise and event intensity.
- Exercise stress testing should be considered if there is a large discrepancy between the actual fitness level of the patient and the expected exercise/event demands (may be predicted using METs).
- Defining exercise capacity and disease stability will guide the development of a graduated training program to help accommodate the anticipated demands.
- Additional risk stratification should include symptoms noted at present, recent ischemic events, other comorbidities, and past results of EKG, exercise stress tests, and echocardiograms.
- All individuals with heart disease should be encouraged to achieve and maintain good cardiovascular fitness whether at low elevations or when they intend to travel to higher altitudes.

### CARDIAC ARRHYTHMIAS

- Ascent to altitude causes a release of catecholamines, which may trigger cardiac arrhythmias.
- Premature atrial and ventricular contractions are common at high altitudes and are usually benign.
- An individual experiencing arrhythmia symptoms that have not been appropriately evaluated or are unstable should not participate in wilderness and adventure activities.
- Individuals with poorly controlled, dangerous arrhythmias should be advised not to travel to high altitudes.
- Stable chronic atrial fibrillation may be managed with appropriate rate control medication and symptomatic paroxysmal atrial fibrillation with PRN rescue medications (e.g., beta or calcium-channel blockers).
- Patients taking anticoagulant and antiplatelet medications should be counseled about the risk of bleeding if there is a high risk of fall injuries.

### CONGESTIVE HEART FAILURE

- Congestive heart failure (CHF) must be well controlled before any ascent to altitude, and participants should be made aware of the associated risks during PPE.
- It is important to determine ACC/AHA classification (A–D) of heart failure.
- Class A and B patients with normal exercise capacity established by formal exercise testing usually tolerate wilderness activity well if exercise capacity does not exceed beyond wilderness activity demands.
- Class C patients are at a considerable risk of decompensation if activity demands exceed exercise capacity; thus, it is safest to advise against participation in wilderness events that are likely to stress the cardiovascular system to that degree.
- Ascent to altitude is associated with the risk of HAPE even in healthy individuals.
- Class D patients are typically incapable of participating in wilderness events.

## EBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eForm 16.1: Example of a PPE Health History Form for a Wilderness Sports/Adventure PPE

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## EBOOK SUPPLEMENTS

- eForm 16.1 Example of a PPE Health History Form for a Wilderness Sports/Adventure PPE

**Example of a PPE Health History Form for a Wilderness Sports/Adventure PPE**

| **Part I: Participant and Expedition Information** | | |
|---|---|---|
| Name: | *DOB/Age: | Gender: |
| Planned event (location, country, sport, expedition): | | |
| Date of planned departure: | Total trip/event days allowed: | |
| Plans for travel insurance or emergency management including self rescue: | | |

| **Part II: Medications and Immunizations** | **Yes** | **No** | **Provider Notes (Administrative use only)** |
|---|---|---|---|
| Do you need medication refills for chronic conditions? | | | |
| Do you need prophylactic medications such as antibiotics or pain management for travel? | | | |
| Are you up to date with standard immunizations (e.g., tetanus, influenza)? | | | |
| Are you aware of destination-specific immunizations that you may need for your travel, sport, or adventure? | | | |

| **Part III: Past Medical History of Chronic Health Conditions** | **Yes** | **No** | **Provider Notes (Status, last evaluation)** |
|---|---|---|---|
| **Cardiovascular conditions** | | | |
| High blood pressure or taking medication for blood pressure | | | |
| Previous heart attack or heart disease (history of angioplasty, coronary artery stenting, *CABG, *CAD, other) | | | |
| Heart arrhythmia | | | |
| Congestive heart failure | | | |
| Other cardiovascular conditions | | | |
| **Lung conditions** | | | |
| Asthma | | | |
| *COPD/emphysema | | | |
| Sleep apnea (obstructive or central) | | | |
| Use of a *CPAP machine | | | |
| Other pulmonary conditions | | | |
| **Central nervous system** | | | |
| Seizure disorder | | | |
| Headache disorder (migraine, other) | | | |
| Multiple sclerosis | | | |
| Movement disorders (Parkinson's, other) | | | |
| Other neurologic or central nervous system conditions | | | |
| **Gastrointestinal conditions** | | | |
| Acute gastrointestinal disorder | | | |
| Chronic gastrointestinal disorder (irritable bowel, inflammatory bowel, celiac disease) | | | |
| Other absorption conditions | | | |
| **Musculoskeletal conditions** | | | |
| Arthritis (osteoarthritis, rheumatoid arthritis, other) | | | |
| Joint instability or pain issues | | | |
| History of joint replacement | | | |
| Osteoporosis | | | |
| Other musculoskeletal conditions and injuries | | | |
| **Hematologic conditions** | | | |
| Bleeding disorder | | | |
| Blood clotting disorder | | | |
| Taking blood thinners | | | |
| Sickle cell disease or trait | | | |
| Iron deficiency | | | |
| Other hematologic conditions | | | |

| Part III: Past Medical History of Chronic Health Conditions (cont.) | Yes | No | Provider Notes (Status, last evaluation) |
|---|---|---|---|
| **Ophthalmic conditions** | | | |
| Blindness (any degree) | | | |
| Use of corrective eyewear (contact lenses or glasses) | | | |
| History of eye surgery (*LASIK, cataracts, lens replacement) | | | |
| Glaucoma | | | |
| Other eye disorder | | | |
| **Psychiatric conditions** | | | |
| Substance abuse (use of alcohol or illicit substances) | | | |
| Current use of psychiatric medications | | | |
| Anxiety or depression (including history of suicidal behaviors or hospitalizations) | | | |
| Other psychiatric conditions | | | |

| Part IV: Past history of high altitude illness<br>*Have you ever been diagnosed with any of the following altitude conditions? Check all that apply.* | Yes | No | Elevation if known | Provider Notes (Administrative use only) |
|---|---|---|---|---|
| Acute mountain sickness (AMS) | | | | |
| High altitude pulmonary edema (HAPE) | | | | |
| High altitude cerebral edema (HACE) | | | | |

| Part V: History of other acute medical conditions occurring in the backcountry or wilderness.<br>*Check any condition you have experienced while in the wilderness in the past.* | Yes | No | Provider Notes (Administrative use only) |
|---|---|---|---|
| Carbon monoxide poisoning | | | |
| Frostbite | | | |
| Gastroenteritis (diarrhea, vomiting) | | | |
| Skin condition or wound issue | | | |
| Heat related | | | |
| Cold related | | | |
| Vision (sun or snow related, blindness, other) | | | |

*DOB: date of birth, CABG: coronary artery bypass graft, CAD: coronary artery disease, COPD: chronic obstructive pulmonary disease, CPAP: continuous positive airway pressure, LASIK: laser assisted in situ keratomileusis.

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# EXERCISE PRESCRIPTION AND PHYSIOLOGY

# 17

O. Josh Bloom • Karl B. Fields • Todd M. McGrath • Timothy R. Draper

## EXERCISE PHYSIOLOGY

- Exercise can be defined as "bodily exertion, especially for the sake of training, recreation, or fitness."
- Science of processes and mechanisms of skeletal muscle contraction and the corresponding interaction of other body systems that facilitate and respond to skeletal muscle contraction
- Understanding the body's physiologic response to repetitive skeletal muscle contraction is imperative.
- Skeletal muscle contraction that exceeds physiologic limits, is inappropriate in duration or intensity, or for which the musculoskeletal system has been inadequately prepared can lead to injury or poor performance/adaptation to training.
- Clinical relevance lies in identification of the pathology, what triggered it, and how to correct, alleviate, and/or prevent this in both individuals and populations of active people.

### Terminology

- Metabolic equivalents (METs): expression of metabolic cost of exercise in terms of oxygen consumption
  - Rest defined as 1 MET, which is an oxygenation of 3.5 mL $O_2$/kg/min
  - MET chart (Table 17.1)
- $\dot{V}O_2$ max: maximal oxygen utilization
  - Gold standard for evaluating cardiorespiratory fitness
  - Measured in mL $O_2$/kg/min
- Anaerobic threshold: point at which oxygen demand exceeds oxygen availability
- Workload: amount of energy required to complete a specific task (Table 17.2)

### Basic Science

- The sarcomere, which is the basic unit of muscle, is composed of actin/myosin filaments (Fig. 17.1).
- This linkage between actin and myosin is facilitated via acetylcholine release at the motor endplate, which triggers depolarization and subsequent release of calcium from the sarcoplasmic reticulum; this is followed by a series of reactions, causing formation of an adenosine triphosphate (ATP)–myosin complex, subsequent change in the conformation of the myosin unit, and ultimately, traction on the actin filament. Subsequently, this pulls on the connective tissue components of the sarcomere, resulting in contraction of the muscle (Fig. 17.2), which leads to movement.
- The motor nerve center directs timing and sequencing of motor unit recruitment and firing, which facilitates coordinated movement.
- Continued muscle contraction relies on an adequate supply of ATP in each sarcomere.
- ATP is provided by three interlinked, overlapping energy systems that synthesize ATP for both short periods of intense vigorous activity and longer periods of lower-level sustained activity.
- Exercise results in increases in protein synthesis within the muscle with long-term/cumulative changes to the steady-state level of protein synthesis with repetitive exercise.

### Energy Systems

#### PHOSPHAGENS (ATP–CREATINE PHOSPHATE SYSTEM)

- Anaerobic system used in maximum-intensity exercise lasting only seconds
  - Composed of ATP and creatine phosphate stored in the cytoplasm of each sarcomere
  - Phosphagens provide rapid resynthesis of ATP on the myosin head, facilitating brief high-intensity bursts of muscle activity.
  - Particularly important during very-high-intensity exercise and at the beginning of exercise

#### GLYCOGEN TO PYRUVATE ("LACTIC ACID SYSTEM")

- Anaerobic process that degrades muscle glycogen to pyruvate
  - Pyruvate is an essential substrate that can be oxidized to ATP via the aerobic energy system (see Oxidative System).
- Also directly provides some ATP, which becomes part of the phosphagen pool
- Energy source in high-intensity, short-burst exercise (typically ≤3 minutes)
- Pyruvate can be reversibly converted to lactate, which can be transported out of the cell for use by other tissues.
  - Also serves as a negative feedback loop because increased lactate causes metabolic acidosis, which increases the rate of ventilation and causes muscle discomfort; these reactions will eventually encourage an individual to reduce the intensity of exercise

#### OXIDATIVE SYSTEM (RESPIRATORY RESPONSE TO EXERCISE)

- The aerobic energy system
- Quantitatively the most important system
- Typically used in activities lasting >3 minutes
- Dependent on oxygen availability at the cellular level
- Accordingly, much of the body's physiology is structured to facilitate transport of $O_2$ to tissues via the cardiorespiratory system.
- Dependent on oxidation of *pyruvate*, *acetyl-CoA* (formed directly from glucose), or *free fatty acids*
- A low-power but extremely high-capacity energy system

### Adaptations to Chronic Exercise ("Training")

- Musculoskeletal and cardiorespiratory systems are highly adaptable to regularly performed exercise.
- Adaptation to repetitive exercise can be understood in terms of several factors, which can be represented by the mnemonic **P-ROIDS**.
  - **P**rogression: Gradual increase in intensity, duration, and difficulty of physical activity to improve strength, endurance, and sport-specific skills
  - **R**eversibility: Training is generally continuous or cyclic in nature, owing to the rapid loss of benefits from conditioning when people stop exercising
    - Inactivity for as little as several days can lead to reduction in work capacity secondary to a decrease in protein synthesis from baseline with loss of exercise stimulus.

**TABLE 17.1 METs IN COMMON COMPETITIVE AND RECREATIONAL SPORTS**

| | |
|---|---|
| Basketball (comp) | 7–12 |
| Biking (rec) | 3–8 |
| Dancing | 3–7 |
| Football | 6–10 |
| Golf (cart) | 2–3 |
| Jogging (5–6 mph) | 7–15 |
| Skiing (downhill) | 5–8 |
| Soccer | 5–12 |
| Tennis | 4–9 |
| Volleyball | 3–6 |

Modified from Mead WF, Hartwig R. Fitness evaluation and exercise prescription. *J Fam Pract.* 1981;13(7):1039-1050.

**TABLE 17.2 METs IN WALKING**

| Workload | Miles | Minutes |
|---|---|---|
| 5 METs | 1 | 15–18 |
| 6 METs | 1.5 | 21–25 |
| 8 METs | 2 | 24–29 |
| 10 METs | 4 | 50–54 |
| 12 METs | 5 | 70–80 |

Modified from Mead WF, Hartwig R. Fitness evaluation and exercise prescription. *J Fam Pract.* 1981;13(7):1039-1050.

- **O**verload: Exercising above normal levels through combinations of type of activity, intensity, duration, and/or frequency
- **I**ndividual **D**ifferences
- **S**pecificity: Specific training develops specific adaptations beneficial to a particular sport/activity.

- Training should focus on the energy system that is most important to that sport (e.g., distance runners spend more time working on the oxidative system).
- Modern training programs typically incorporate cross-training because training focused on optimizing all energy systems can improve sport-specific performance (e.g., dry-land and weight training can improve the performance of skiers).
- Concurrent training: integration of both aerobic/endurance and resistance training into a training plan

## Types of Exercise

- Aerobic/endurance exercise: fueled by an oxidative energy system and in place when oxygen supply/delivery to tissues is adequate for sustained; low- to moderate-intensity exercise
  - Increased lipid mobilization and oxidation by skeletal muscle
  - Increased number of mitochondria with adaptation
- Anaerobic exercise: typically high-intensity, shorter-duration exercise that exceeds capacity of the oxidative system and is fueled primarily by phosphagens and glycogen to pyruvate energy systems
- Resistance exercise: training for maximal contractile force and muscle hypertrophy
- Range of motion/stretching/warm-up/cool-down exercises (Fig. 17.3)
- Flexibility exercise: increases ability to lengthen muscle–tendon units and improve motion around specific joints

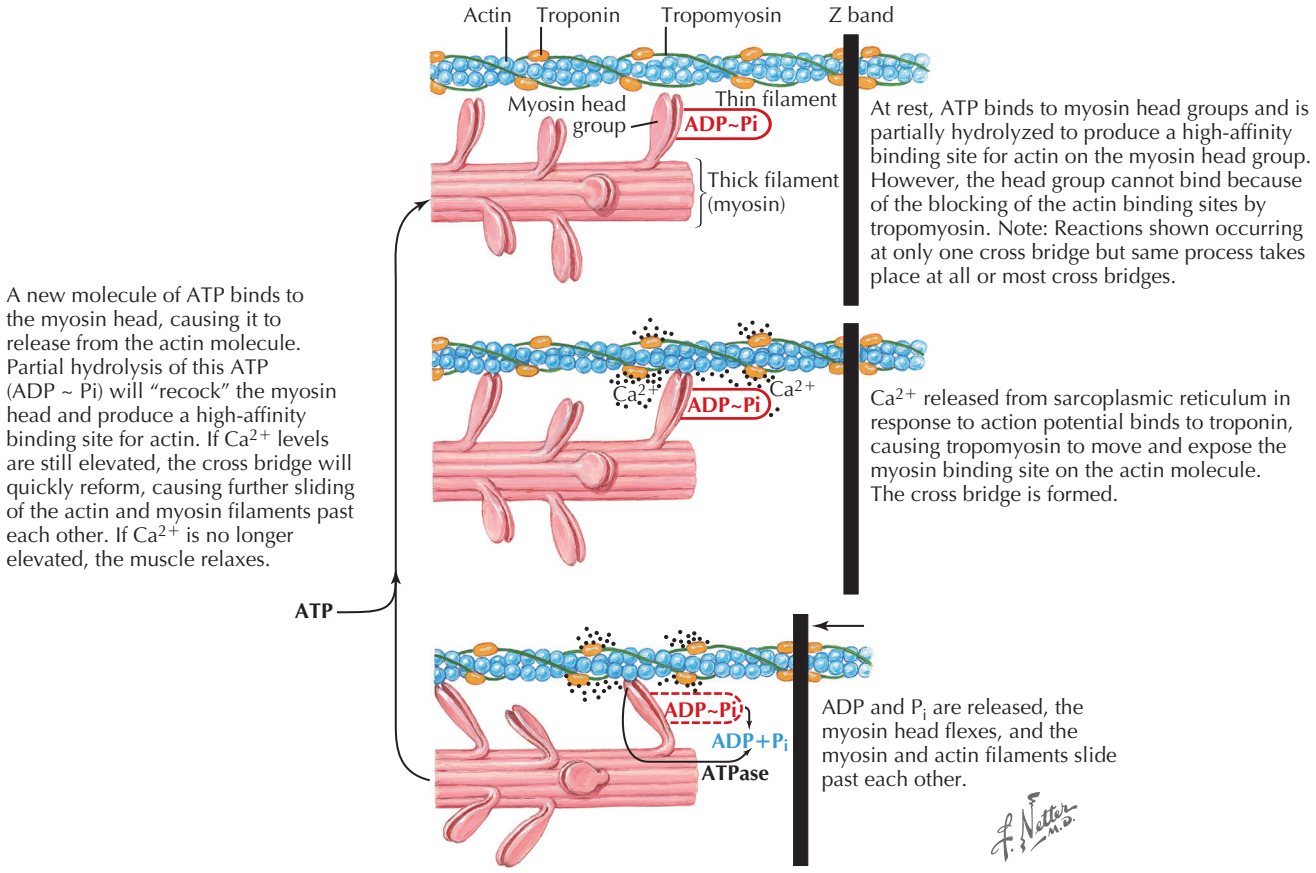
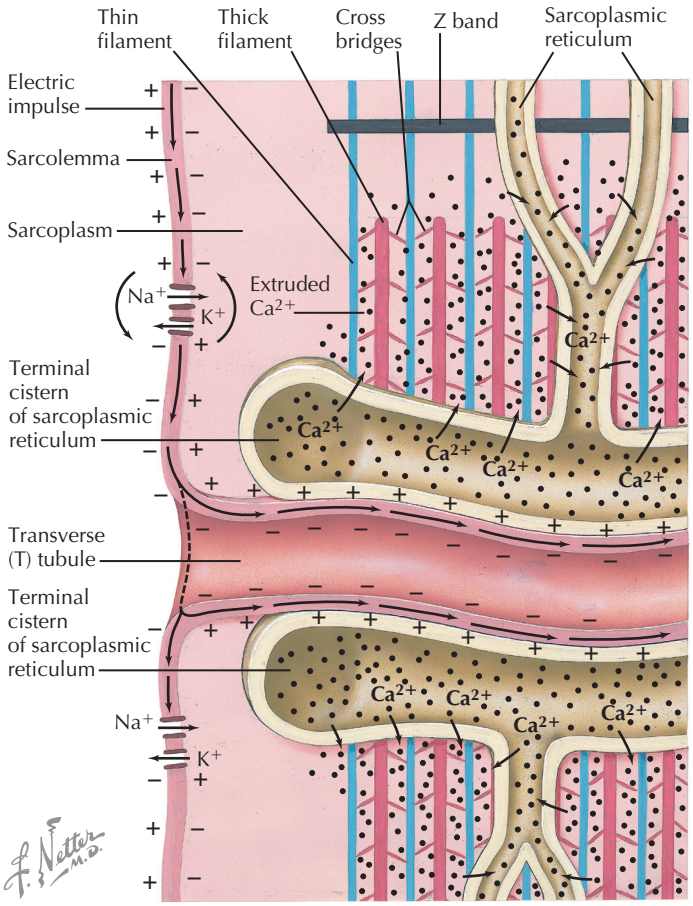



**Figure 17.1.** Biochemical mechanics of muscle contraction.




Electric impulse traveling along muscle cell membrane (sarcolemma) from motor endplate (neuromuscular junction) and then along transverse tubules affects sarcoplasmic reticulum, causing extrusion of $Ca^{2+}$ to initiate contraction by "rowing" action of cross bridges, sliding filaments past one another.

**Figure 17.2.** Initiation of muscle contraction by electric impulse and calcium movement.

- "Lifestyle activity": cumulative, non-structured, moderate-intensity physical activity wherein individuals partake throughout a typical day (e.g., taking the stairs and walking in from the parking lot)
  - 10 minutes of moderate-intensity exercise three times a day facilitates changes comparable to health benefits of 30 minutes of continuous exercise.
  - Increasing lifestyle activity is as effective as structured activity for improving cardiorespiratory fitness and reducing cardiovascular disease (CVD) risk factors.

## EXERCISE PRESCRIPTION

### General Considerations

- A tool to teach, coach, and educate patients regarding opportunities to improve overall health and well-being
  - Can also enhance exercise and sport-specific performance
- Exercise is safe for a majority of patients and has numerous health/fitness benefits.
- A written exercise prescription has been demonstrated to increase activity levels more compared with verbal advice alone.

### Implement as Part of Clinical Practice

- Identify those who would benefit from exercise prescription
- Identify conditions amenable to exercise therapy
- Assess patient's activity level
- Educate patient about the benefits of exercise
- Assess patient interest, motivation, and goals
- Strive to find activities that are sustainable
- Monitor progress; assess barriers

### Initiation of an Exercise Program

- Start slowly and allow fitness level to improve
- Goal is long-term lifestyle change
- Initially, prescribe shorter periods of light- to moderate-intensity exercise and build in duration, frequency, and intensity over time

### Safety

- Data are reassuring.
  - An overwhelming majority of medical-related problems are soft-tissue and overuse injuries.
  - Encourage appropriate warm-up and cool-down

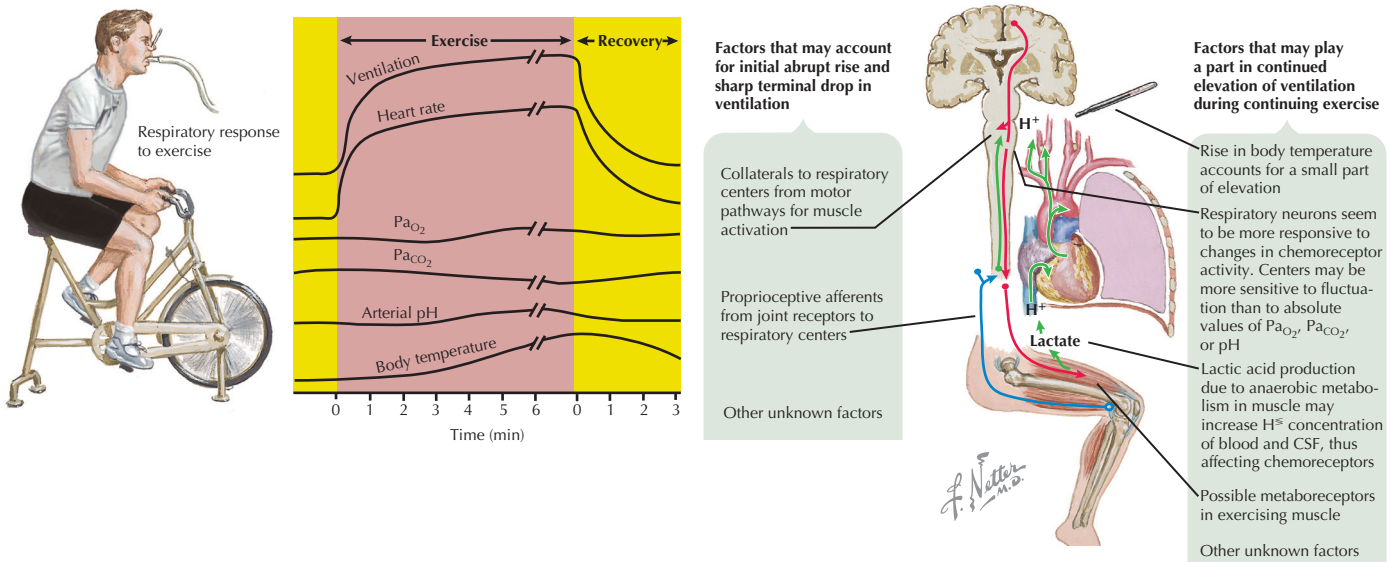



**Figure 17.3.** Physiological response to exercise and cool-down.

- Scientific evidence to support that stretching before activity prevents injuries is limited.
- Sudden death during exercise is rare.
  - A majority (approximately 80%) of instances are caused by coronary heart disease.
  - The risk is greater in sedentary people who start vigorous activity.
  - The relative risk of sudden death during vigorous activity is 2.1 times that of the risk during less strenuous activity.
- Regular activity is protective.
  - Exercise-associated cardiovascular (CV) risks lessen as individuals become more active/fit.
  - Exercise-related CV events are uncommon and frequently preceded by warning signs and symptoms.
  - Encourage slow start, consistency, and progress over time

## Basic Principles of Exercise Prescription

- Tailor prescription to individual patients based on their goals, physical/financial/time considerations, medical conditions, and motivation.
- Prescreen as indicated:
  - History and physical examination is the most important tool.
  - Exertional chest pain, syncope, near-syncope, disproportionate dyspnea on exertion, and marked decrease in exercise capacity are red flags warranting further evaluation (Box 17.1).
- Conduct functional/orthopedic evaluations as needed.

## Elements of Exercise Prescription (FITT-VP)

### Frequency

- Ideally five or more times per week

### Intensity

- Instruct on methods to assess exercise intensity:
  - Rating of perceived exertion
  - Borg scale (Box 17.2)
  - $\dot{V}O_2$ max
  - Maximum predicted heart rates can be calculated by subtracting age from 220
  - "Talk test"—defined as the ability to talk without significant breathlessness; rough marker of moderate- or low-intensity exercise

### Time/Duration

- Current guidelines recommend 30–60 minutes of exercise most days of the week.
- Obesity data call for 90 minutes of exercise daily.

### Type

- Accumulating evidence supports the benefits of cross-training in terms of injury prevention, improved function, fitness level, and compliance.
- Dynamic flexibility exercises and disciplines such as yoga or Pilates may be most useful in individuals who wish to pursue activities that require excellent flexibility and in older individuals who tend to lose mobility around joints with aging (Fig. 17.4).

### Volume

- Gradually increase volume of activity

### Progression

- Progress from lighter levels of exertion to more vigorous and more complex exercise over time as patient improves fitness level, tolerance, and functional capacity

## Screening/Exercise Testing

- Current evidence-informed model for pre-exercise screening is based on three factors:
  1. Individual's current level of physical activity
  2. Presence of signs or symptoms and/or known CV, metabolic, or renal disease
  3. Desired exercise intensity
- Current recommendations no longer endorse *low/moderate/high*-risk classification or use of CVD risk profile as indication for referral to a medical care provider before initiating moderate- to vigorous-intensity exercise programs.
  - The high prevalence of CVD risk factors among adults combined with the rarity of exercise-related sudden cardiac death and acute myocardial infarction argues that the ability to predict these rare events by assessing CVD risk factors is relatively poor.
- Current recommendations advocate *medical clearance* versus specific recommendations for *medical exams* or *exercise stress tests (ESTs)*.
  - Manner of clearance left to discretion of the medical care provider
  - Decisions about referral to a healthcare provider for *medical clearance* before initiation of an exercise program should be made on an individual basis based on clinical judgement.
- 2015 ACSM algorithm: see Recommended Readings

### BOX 17.1 MAJOR CARDIAC RISK FACTORS (AMERICAN HEART ASSOCIATION)

**Nonmodifiable Factors**

- Male gender
- Increasing age
- Heredity

**Modifiable Factors**

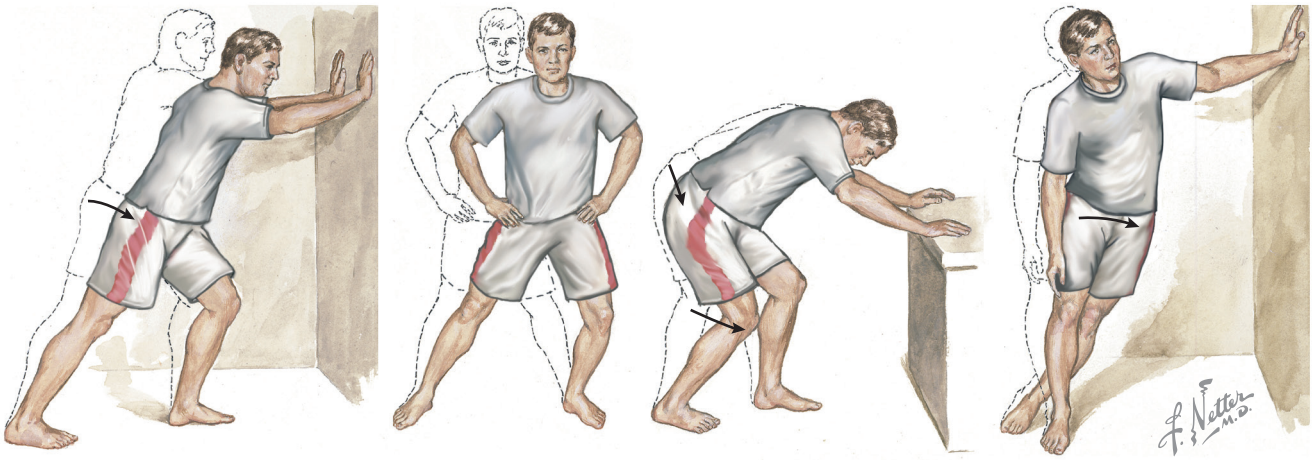
- Smoking
- Hyperlipidemia
- Hypertension
- Diabetes mellitus
- Physical inactivity
- Obesity and overweight

Modified from The American Heart Association. *Risk Factors and Coronary Heart Disease,* AHA Position Statement.

### BOX 17.2 LINEAR 6-TO-20 BORG SCALE OF PERCEIVED EXERTION OF PAIN

| | |
|---|---|
| 6 | |
| 7 | Very, very light |
| 8 | |
| 9 | Very light |
| 10 | |
| 11 | Fairly light |
| 12 | |
| 13 | Somewhat hard |
| 14 | |
| 15 | Hard |
| 16 | |
| 17 | Very hard |
| 18 | |
| 19 | Extremely hard |
| 20 | |

From Morrison C, Norenberg R. Using exercise test to create the exercise prescription. *Prim Care.* 2001;28(1):137-158.




Straight-leg stretch for gastrocnemius and plantar fascia

Adductor stretch

Bent-knee stretch for soleus, achilles tendon, posterior tibial tendon, and plantar fascia

Abductor and iliotibial tract stretch

**Figure 17.4.** Stretching exercises.

## Mortality Data

- Exercise capacity (fitness level) is an independent powerful predictor of clinical outcomes.
- In healthy patients and those with known CVD, peak exercise capacity is a stronger predictor for all-cause mortality than hypertension, diabetes, smoking, obesity, or hypercholesterolemia. All-cause mortality in patients who achieve <5 METs is double the all-cause mortality in those who achieve >8 METs.
- There is an inverse relationship between fitness level and mortality (i.e., higher fitness level correlates with lower mortality).
- Patients with low fitness levels have a four-fold increased relative risk of cardiovascular death and an eight-fold increased relative risk of cancer death, relative to peers with high fitness levels.
- Individuals who are unable to achieve 5–6 METs during EST are at an increased risk of coronary events and all-cause mortality.
- Inability to achieve 6 METs during EST places an individual at an increased risk of triple vessel or left main coronary artery disease and at least four times the age-adjusted risk of a cardiac event.
- Poor fitness level is a modifiable risk factor.
- Every 1 MET increase in exercise capacity confers increased survival rates of 8%–18%.
- Performing some amount of exercise is better than being sedentary.
- Greatest improvements in survival rates are typically between the least and the next-to-least fit quintiles.

## Special Populations

### Diabetes Mellitus (DM)

#### EXERCISE PRECAUTIONS

- Strongly consider baseline EST if:
  - Age >40 years with or without CVD risk factors other than diabetes
  - Age >30 years and:
    - Type 1 or 2 diabetes since >10 years
    - Hypertension
    - Cigarette smoking
    - Dyslipidemia
    - Proliferative or preproliferative retinopathy
    - Nephropathy, including microalbuminuria
  - Any of the following regardless of age:
    - Known or suspected CAD, cerebrovascular disease, or peripheral artery disease
    - Autonomic neuropathy
    - Advanced nephropathy with renal failure
- Retinopathy—avoid strenuous exercise and weight training (increased risk of vitreous hemorrhage and retinal detachment)
- Significant peripheral neuropathy—risk of ulcerations/skin breakdown and fracture
  - Appropriate socks/footwear important
- Caution during extremes of weather
- Ensure adequate hydration
- Skin issues and trauma around insulin pump site
- Caution regarding hypoglycemia and hyperglycemia
  - Higher-intensity aerobic exercise increases the risk of hypoglycemia.
  - Exercise induces a spike in endogenous insulin secretion; hence, individuals may develop hypoglycemia relatively early during exercise.
  - Delay exercise if glucose is >250 mg/dl with ketones or >300 mg/dl (with or without ketosis).

#### EXERCISE IMPROVES INSULIN SENSITIVITY

- Improvements decline rapidly (typically within 48 hours after exercise).
- Accordingly, patients with diabetes are recommended to exercise daily and not skip exercise for >1 day.

#### IMPROVES GLYCEMIC CONTROL

- Meta-analysis (of 14 studies, 12 addressing aerobic exercise and 2 resistance exercise, at moderate intensity, performed only 2.5–3.4 times/week) shows a 0.66% drop in hemoglobin A1C (irrespective of weight change or body mass index [BMI] change).

#### PREVENTS PROGRESSION

- Exercise prevents progression of glucose intolerance from "prediabetes" to diabetes.
  - Inverse relationship between level of physical activity and risk of type 2 diabetes

- Each 500 kcal expended in exercise per week is associated with a 6% decline in the risk of diabetes.
- Improved fitness level decreases mortality at each level of glycemic control.

GENERAL EXERCISE RECOMMENDATIONS

- Regular exercise is roughly comparable to taking one oral hypoglycemic medication.
- Encourage 150 minutes/week of moderate (brisk walking) to vigorous aerobic exercise (more if obese) spread across the week (at least 3 days).
- In addition to aerobic activity, encourage moderate- to vigorous-resistance training at least 2–3 days/week.
- Milder forms of exercise (such as yoga) have shown mixed results.
- Avoid exercise if blood glucose is >250 with ketosis or >300.

## Hypertension (HTN) (see also Chapter 36: The Hypertensive Athlete)

- Higher fitness level correlates with lower current and subsequent risk of hypertension independent of other risk factors.
- Numerous studies have demonstrated various modes of exercise to be effective in lowering blood pressure (BP) independent of weight changes.
  - Meta-analysis of 127 randomized clinical trials (RCTs) irrespective of baseline showed systolic BP reductions of 3.4–4.7 and diastolic BP reductions of 2.4–3.1 mmHg.
  - Higher initial BPs confer larger reductions (average BP reduction in hypertensive patients is 7.4 mmHg in systolic BP and 5.8 mmHg in diastolic BP).
  - Meta-analysis of 54 RCTs conducted in hypertensive and normotensive patients revealed that all frequencies, intensities, and types of aerobic exercise-lowered BP (regardless of race, BMI, weight loss, or baseline BP).
- Hypertensive physically fit men and women had 60% lower mortality rate than physically unfit normotensive men and women.
- As little as 4 months of an exercise program positively affects left ventricular remodeling, along with other benefits typically being achieved by 3 months.
- BP benefit is lost with cessation of exercise.
- Moderate-intensity exercise performed for >150 minutes per week showed the best results.
- In sedentary adults, "lifestyle activity" was as effective as a structured exercise program for BP reduction.
- Consistent aerobic exercise:
  - Effective in lowering BP, resting and exercise HR, and myocardial oxygen demand
  - Increases vagal tone and decreases plasma norepinephrine
  - Decreases resting sympathetic tone
  - BP has been shown to be reduced for up to 22 hours after endurance exercise—this phenomenon is known as *postexercise hypotension* (PEH)
- Resistance training also effectively lowers BP.
  - Regular resistance training lowers BP by an average of up to 5 mmHg.
    - Circuit training at 30%–50% maximum heart rate, with 15–30 seconds of rest
  - Regular resistance training decreases BP and HR response to any given workload (may be cardioprotective).
- High-intensity lifting can considerably increase BP (SBP >300 mmHg documented in power lifters).
  - Much milder increases in BP with less intensity
  - No documented clinical evidence of increased risk from BP increase during static exercise
- Consistently, circuit-type low- to moderate-resistance strength training appears most beneficial in hypertensive individuals.

PRECAUTIONS/RECOMMENDATIONS/MEDICATIONS

- BP should be <180/105 before initiating an exercise program.
- Exercise is safe in patients with uncomplicated mild HTN and who are not on any antihypertensive medications.
- Angiotensin-converting enzyme inhibitor (ACEI) and angiotensin receptor blocker (ARB) are the least likely to affect exercise tolerance.
- Beta-blockers will impede the heart rate response and can decrease exercise tolerance.
- Calcium-channel blockers (CCBs) are generally well tolerated but can cause postexercise orthostasis (a cool-down period helps).
- CCBs may be preferred in African-American patients.
- Exaggerated BP response to exercise occurs even in well-conditioned athletes; this may aid in identifying a subgroup of athletes who are at a greater risk of developing CV complications in the future.
- Caution should be exercised with regard to heat and dehydration, particularly in patients who are on diuretics.

## Osteoarthritis

- Aerobic exercise, resistance training, and physical therapy can reduce pain and disability in people with osteoarthritis (OA).
- Benefits observed in terms of improvements in strength, symptoms, function, and balance
- Systemic reviews assessing various exercises recently have shown benefits on pain, self-reported disability, walking speed, function, and overall patient global assessment of effect.
  - Walking, biking, swimming, and low-weight/high-repetition resistance training have demonstrated beneficial effects.
  - Isometric exercises are particularly helpful in lower-extremity OA.
  - Range-of-motion exercises have not been shown to be beneficial.
- Moderate-intensity exercise is safe (including in the elderly).
- Increases in exercise intensity should be kept to a minimum during flare-ups of OA.
- According to the EBM, the benefits of high-intensity versus low-intensity exercise in individuals with hip or knee OA remains unclear.
- Exercise does *not* cause OA in previously normal joints.
  - Runners and to a lesser extent walkers have a lower risk of developing hip OA and THR.

## Chronic Obstructive Pulmonary Disease (COPD)

- Although exercise has not been shown to reverse the physiologic changes of chronic obstructive pulmonary disease (COPD), numerous studies have confirmed that exercise improves dyspnea by reducing the rate of ventilation and improving exercise tolerance.
- Pulmonary rehabilitation programs relieve dyspnea and fatigue and improve emotional status of patients with COPD. Improvements are moderately large and also have clinical importance.
- Regular (daily) moderate-intensity exercise is most beneficial in improving functional capacity.
- Strong evidence demonstrates that in COPD patients, supervised physical exercise training improves exercise capacity when combined with aerobic and strength exercises, showing greater benefits than when either are performed alone.
- Cardiopulmonary testing and periodic pulmonary function tests can be used to assess if athletes with alpha-1 antitrypsin deficiency, cystic fibrosis, or other chronic pulmonary diseases can achieve the MET level required for a particular sports activity without developing acidosis, hypoxia, or other complications that would limit participation.
- Athletes with COPD from sarcoidosis and cystic fibrosis have competed at elite international levels in basketball and other

demanding sports, underscoring that mild to moderate pulmonary limitation rarely prevents participation.

### Obesity

- Negative caloric balance (less caloric intake than caloric expenditure) is required for weight loss. Increasing physical activity is crucial to establish this negative caloric balance.
- Diet is necessary for weight loss in obese patients. Once overweight, physical activity without control of caloric intake does not control weight gain.
- However, several studies have demonstrated that physical activity is critical to maintaining weight loss.
- Regardless of weight loss, improvements in body composition (loss of body fat and increase of muscle mass) can be expected with increased physical activity.
- Increasing lean muscle mass will increase the basal metabolic rate, allowing increased baseline calorie utilization. Because resistance training has been demonstrated to substantially improve lean muscle mass, it should be considered as part of an exercise prescription for overweight and obese individuals.
- A large observational study of adult men clearly demonstrated that obese but fit men had considerably lower all-cause mortality than unfit, normal-weight men.
- Current data suggest that longer duration of exercise (60–90 minutes/day) is necessary for sustainable weight loss.
- Increased lifestyle activity has been shown to be as efficacious as formal exercise for weight control.
- Specific weight-related considerations for overweight and obese patients:
  - Overweight and obesity are major risk factors for coronary artery disease. Accordingly, appropriate evaluation/screening is warranted before initiating an exercise prescription.
  - Low-impact or nonweight-bearing exercises may have to be considered for certain overweight and obese individuals because of the increased risk and prevalence of orthopedic problems.
  - Swimming and water aerobics, biking, and circuit-type weight programs should be considered in these populations.
- Moreover, thermoregulation can be an issue in overweight and obese individuals.
  - Exercising during cooler time periods of the day, wearing loose-fitting clothing, and ensuring adequate hydration may reduce the risk of heat-related problems.

## KEY CONSIDERATIONS

- A basic understanding of exercise physiology guides the physician in preparing a safe exercise prescription.
- Written exercise prescriptions appear to improve compliance with exercise recommendations.
- An exercise prescription should incorporate frequency, intensity, time, type, volume, and progression (FITT-VP).
- Physicians should decide which patients merit EST testing before initiating an exercise program.
- Patients with chronic disease greatly benefit from exercise, but their exercise prescription and medications must be individualized to maximize their health outcomes.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 18 AEROBIC TRAINING

John C. Hill • Inigo San-Millan

## INTRODUCTION

- In the early days of sporting events, coaches and athletes learned through trial and error that they could not simultaneously develop maximal endurance and maximal power. They found that by first establishing an aerobic endurance base and later adding faster training, they could peak at appropriate times.
- It was not until the 1960s that the study of exercise as a science became widespread, and gradual changes in training methods occurred in the 1970s. By the 1980s, exercise science grew by quantum leaps, and the explosion in scientific information continued even during the 1990s. Now, we can use evidence gleaned from studies involving top cyclists, runners, swimmers, rowers, and triathletes to better understand which physiologic components can be pushed to allow performance at a higher and more economical aerobic potential than ever thought possible.
- Aerobic physical fitness is measured in two basic components: cardiorespiratory adaptations (central) and muscle metabolic adaptations (local).
- Elite endurance athletes have excellent values for these two physiologic traits; however, this is not true for recreational athletes. A very high measure of maximal cardiorespiratory capacity may be a genetic phenomenon that only few individuals possess, but muscle metabolic adaptations can be significantly improved with appropriate training techniques (Fig. 18.1, see also Fig. 17.3).

## CENTRAL VS. LOCAL ADAPTATIONS TO EXERCISE

- Skeletal muscles possess a very high demand for oxygen during exercise.
- Both the cardiovascular and respiratory systems must work together to achieve the delivery of oxygen to muscles.
- This cardiorespiratory response to exercise reflects central adaptations to exercise. However, there are other elements in muscles that are crucial for athletic performance.
- Once oxygen is delivered to muscles, it must be appropriately utilized.
- Moreover, muscles must synthesize adenosine triphosphate (ATP) to produce muscle contraction and function. ATP is synthesized from carbohydrates (carbon hydrogen oxygen [CHO]), fats, and proteins in the mitochondria.
- Muscle metabolism during exercise elicits different byproducts such as lactate that need to be efficiently metabolized; these metabolic responses in skeletal muscles represent local adaptations to exercise.
- Over the past decade, there has been substantial research regarding and improvement in understanding muscle metabolism, and local adaptations during exercise may be most important when it comes to athletic performance.

### Cardiorespiratory Adaptations—Central

- The cardiovascular system has central and peripheral components (Fig. 18.2):
  - **Central components** include heart rate (HR), stroke volume (SV), and cardiac output (CO = HR × SV).
  - **Peripheral components**
    - Arterial–mixed venous oxygen difference (a $-\dot{V}O_2$ difference)
    - Ability of tissues to extract and use oxygen for ATP resynthesis is another primary component of the cardiovascular system during physical activity.
- Maximal oxygen consumption ($\dot{V}O_2$ max)
  - $\dot{V}O_2$ max is the maximum capacity that the body has to transport and use oxygen during exercise, and it represents cardiorespiratory adaptations to exercise as well as the **aerobic capacity** of an individual.
  - $\dot{V}O_2$ max measurement is considered the "gold standard" for aerobic capacity in athletes.
  - In general, it is expressed in milliliters of $O_2$ consumed per kilogram of body mass per minute (ml/kg/min) and may also be expressed in liters per minute (L/min).
- Largely determined by genetics and is limited by certain structural components such as heart size, SV, and lung capacity
- Because oxygen is used by mitochondria, a direct relationship exists between mitochondrial capacity and mitochondrial oxidative enzymes, which are part of local components of an athlete's physiology.
- Although considerable attention has been paid to $\dot{V}O_2$ max over the years, more recent knowledge and insights in muscle metabolism indicate that $\dot{V}O_2$ max is a poor indicator of performance, particularly in competitive athletes.
- In addition, it has been documented that numerous elite athletes greatly improve their performances throughout their careers without improving their $\dot{V}O_2$ max.
- It is well documented that local changes at the metabolic level in skeletal muscles are largely responsible and are crucial for improving athletic performance.

### Muscle Metabolic Adaptations—Local Adaptations

- The ability of humans to exercise ultimately depends on the ability to transform chemical energy into mechanical energy, which ultimately takes place in the mitochondria of skeletal muscles. Thus, mitochondria (Fig. 18.3) are one of the most important players in skeletal muscle metabolism and athletic performance.
- Fats, CHOs, and proteins are oxidized in mitochondria for ATP synthesis. Although at very high rates of cellular glycolytic flux and under anaerobic conditions, glucose is oxidized into cytosol, synthesizing two ATPs and producing lactate.
- Moreover, characteristics of skeletal muscle fibers play a vital role in muscle metabolism, substrate utilization, and ATP synthesis.
- Skeletal muscle is composed of three kinds of muscle fibers:
  - Slow twitch/red/oxidative muscle fibers also called *Type I*
  - Fast twitch/white/glycolytic muscle fibers also called *Type II* that are divided in two subgroups called *Types IIa and IIb*
- Muscle fiber contraction obeys a sequential recruitment pattern related to exercise intensity, wherein Type I muscle fibers are first to be recruited at lower intensities.
- As exercise intensity increases, Type IIa and finally Type IIb fibers are recruited.
- Each muscle fiber has different biochemical properties and thus different metabolic behaviors during exercise (Fig. 18.4).

### Type I Muscle Fibers

- Small motor neuron and fiber diameter, high mitochondrial and capillary density, and high content of myoglobin and oxidative enzymes
- Possess a high capacity to oxidize free fatty tissue from subcutaneous adipose tissue and from intramuscular triglycerides
- Although they have the slowest contraction time and force production of all fibers, they have the highest resistance to fatigue.
- Type I muscle fibers are mainly contracted during endurance events.

### Type IIa Muscle Fibers

- Possess a larger motor neuron and fiber diameter, high glycolytic capacity, and a good or intermediate oxidative capacity although not as much as Type I muscle fibers do
- Mitochondrial density and fat oxidation capacity are lower than that of Type I muscle fibers.
- Have a higher content of glycogen and glycolytic enzymes; thus, glucose oxidation is higher
- Contraction time is longer and resistance to fatigue is lower than that of Type I muscle fibers.
- Because most competitions are performed at high intensities, in the glycolytic state, type IIa muscle fibers play an important role in most athletes, including endurance athletes.

### Type IIb Muscle Fibers

- Also called *fast glycolytic* or *fast white*
- Have fastest contraction time and greatest force among all fibers
- Mitochondrial density is very low, and most ATP synthesis in these fibers comes from anaerobic glycolysis and from stored ATP and phosphocreatine (PC), which binds to ADP hydrolyzed from ATP to regenerate ATP.
- Type IIb muscle fibers are pure anaerobic fibers and are extremely crucial in athletic events of very short duration and high intensity, such as sprinting a 400-m race.

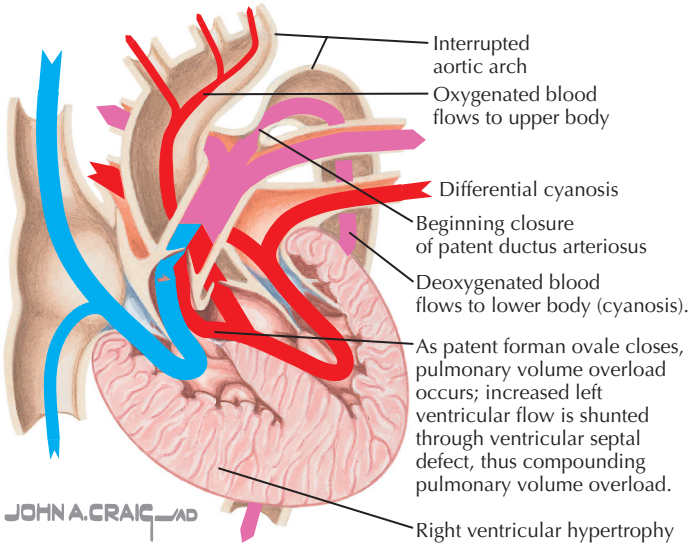



**Figure 18.1.** Physiologic adaptation to exercise.

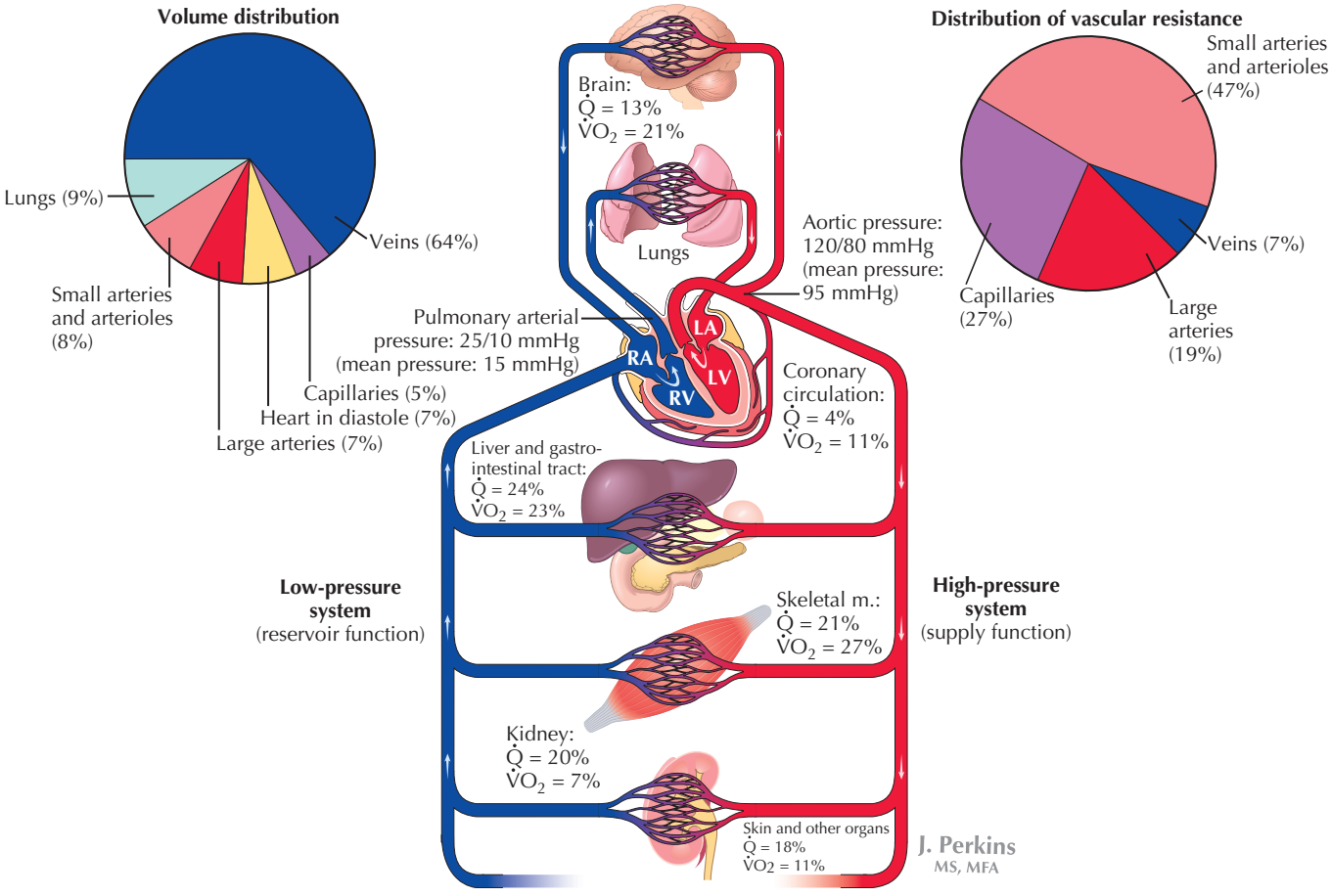
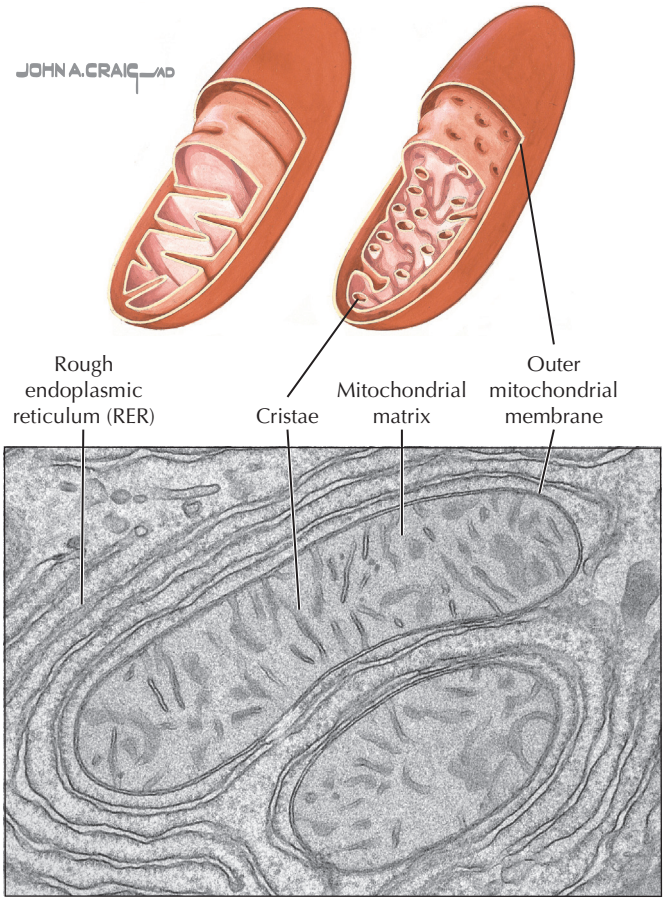



**Figure 18.2.** Components of the cardiovascular system.




Reprinted with permission from Ovalle W, Nahirney P. *Netter's Essential Histology*. 2nd ed. 2013.

**Figure 18.3.** Schematic presentation of mitochondria and EM of mitochondria in a hepatocyte.

## MUSCLE METABOLISM AND BIOENERGETICS

- ATP synthesis is generated by anaerobic and aerobic metabolism, primarily from fats and CHOs, with some contribution from amino acids derived from proteins.
- Fat it is primarily stored in adipose tissue but is also stored in small amounts in skeletal muscles.
- CHOs are stored in the form of glycogen in skeletal muscles (approximately 80%) and in the liver (approximately 15%).
- Exercise intensity, physiologic stress, and muscle fiber recruitment patterns dictate the energy system and the substrates utilized.

## FAT METABOLISM DURING EXERCISE

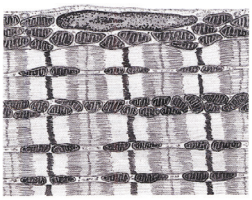
- Fats or lipids are the preferred energy source during endurance exercise.
- Most utilization occurs in Type I muscle fibers.
- Highly orchestrated process, which starts with adipose tissue and ends with mitochondria in skeletal muscles
- Fat stored in adipose tissue is broken down by lipolysis into free fatty acids (FFAs), which must subsequently travel through the blood to skeletal muscles.
- Once in the skeletal muscles, FFAs are attached to coenzyme A (CoA) to form fatty acyl-CoA, which is then transported across the outer mitochondrial membrane by carnitine–palmitoyl transferase I (CPT-I) and finally transported to the mitochondrial matrix by carnitine.
- Once inside the mitochondrial matrix, FFAs undergo β-oxidation where fatty acyl-CoA is degraded to acetyl-CoA, which can then enter the citric acid cycle for ATP synthesis.
- Moreover, skeletal muscle cytoplasm stores some fat called *intramuscular triglycerides* (IMTGs), which are stored close to the mitochondria and can contribute to energy generation. Based on the level of physical condition, IMTGs can contribute up to 15%–30% of all fat oxidation through exercise.
- Advantage of fat metabolism is that fat stores are unlimited, and even the leanest individual will never run out of fat stores.
- Therefore, fat is the preferred substrate for endurance activity during lower-intensity exercises.
- However, as exercise intensity increases, ATP must be synthesized faster, and fat cannot be oxidized fast enough to meet high ATP demands. At this point, the body starts utilizing a faster type of substrate: CHOs.

## CARBOHYDRATE METABOLISM DURING EXERCISE

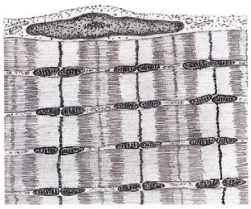
- As exercise intensity increases CHOs are mobilized and eventually become the preferred source of fuel by skeletal muscle.
- CHOs are stored in skeletal muscles (~80%), liver (~15%), and in the form of glycogen (~5% in the form of glucose in the blood).
- Glycogen storage is limited (300–600 g) depending on the size and weight of an athlete.
- Glycogen cannot be directly utilized by skeletal muscles to obtain energy and needs to be broken down to glucose through a process called *glycogenolysis*.
- In muscles, glucose is oxidized to ATP through a multistep process called *glycolysis* that results in the formation of pyruvate, which is then further oxidized to acetyl-CoA for ATP synthesis through the citric cycle in mitochondria.
- At higher intensities, fat cannot synthesize ATP quick enough, and all ATP synthesis is achieved through glycolysis.
- Considering the importance of CHOs for ATP synthesis during high-intensity exercise, appropriate glycogen stores are key for athletic performance.
- Fat is the preferred fuel up until exercise intensities of approximately 55%–75% of $\dot{V}O_2$ max, although CHO is also used at a low rate.
- At higher-intensity exercise beyond 75% of $\dot{V}O_2$ max, ATP must be synthesized at a faster rate in order to meet muscle contractile demands.
- Fat cannot synthesize ATP quick enough, and thus, CHO becomes the major energy source used by skeletal muscles up to 100% of $\dot{V}O_2$ max.
- When exercise intensities are close to maximal, ATP cannot be generated by neither fat nor CHO, and anaerobic metabolism takes over through the PC system and mobilizing ATP stored in skeletal muscles (Fig. 18.5).

## PROTEIN METABOLISM

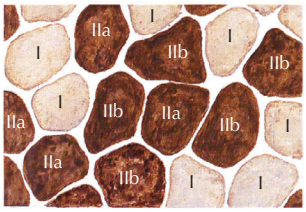
- Protein contributes minimally to energy generation during exercise.
- Total energy produced could range 3%–10% depending on exercise intensity and duration.
- Protein (mainly skeletal muscle protein) is degraded to amino acids for oxidation.
  - Branched-chain amino acids (BCAAs) play an important role during exercise.
  - Leucine, isoleucine, and valine make up BCAAs and can play vital roles during exercise, particularly under low glycogen stores, where BCAA utilization for ATP synthesis increases.
  - Another important amino acid is alanine, which is synthesized in skeletal muscles and then exported to the liver to be



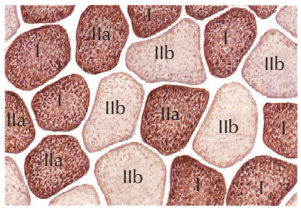
**Type I:** Dark or red fiber. Large profuse mitochondria beneath sarcolemma and in rows as well as paired in interfibrillar regions. Z lines wider than in type II.



**Type II:** Light or white skeletal muscle fiber in longitudinal section on electron microscopy. Small, relatively sparse mitochondria, chiefly paired in interfibrillar spaces at Z lines.



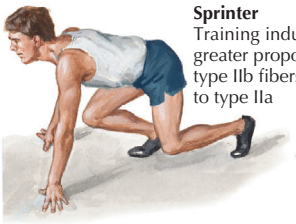

Cross section of skeletal muscle fibers stained for ATPase




Identical section stained for SDH

**Histochemical classification**

| Fiber type | ATPase stain | SDH stain |
|---|---|---|
| **1. Fast-twitch, fatigable (IIb)** Stain deeply for ATPase, poorly for succinic acid dehydrogenase (SDH), a mitochondrial enzyme active in citric acid cycle. Therefore fibers rapidly release energy from ATP but poorly regenerate it, thus becoming fatigued. | | |
| **2. Fast-twitch, fatigue-resistant (IIa)** Stain deeply for both ATPase and SDH. Therefore fibers rapidly release energy from ATP and also rapidly regenerate ATP in citric acid cycle, thus resisting fatigue. | | |
| **3. Slow-twitch, fatigue-resistant (I)** Stain poorly for ATPase but deeply for SDH. Therefore fibers only slowly release energy from ATP but regenerate ATP rapidly, thus resisting fatigue. | | |



**Sprinter** Training induces a greater proportion of type IIb fibers relative to type IIa

**Marathon runner** Type IIa fiber is increased relative to type IIb

| Characteristics of the Three Muscle Fiber Types | | | |
|---|---|---|---|
| **Fiber Type** | **Type I** | **Type IIa** | **Type IIb** |
| | **Slow Twitch (ST)** | **Fast Twitch (FTIIa)** | **Fast Twitch (FTIIb)** |
| Contraction time | Slow | Fast | Very fast |
| Resistance to fatigue | High | Intermediate | Low |
| Metabolism | Oxidative Aerobic | Oxidative Aerobic | Anaerobic |
| Force production | Low | High | Very high |
| Mitochondrial density | High | High | Low |
| Capillary density | High | Intermediate | Low |
| Oxidative capacity | High | High | Low |
| Glycolytic capacity | Low | High | High |
| Major fuel used | FAT | CHO | CHO+ATP+CP |

**Figure 18.4.** Muscle fiber types.

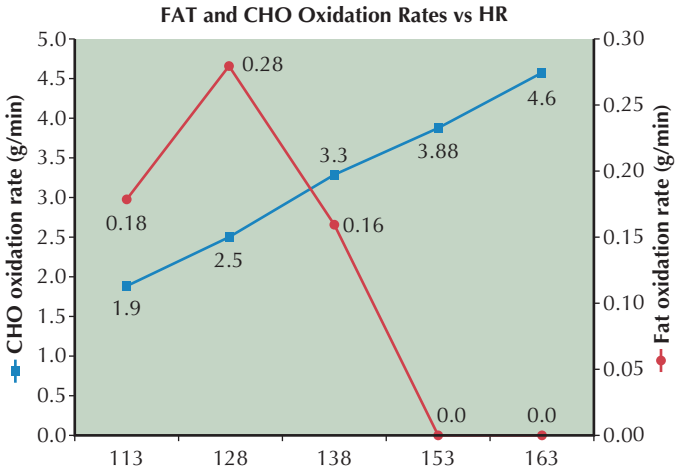



**Figure 18.5.** Fat and carbohydrate utilization during exercise. As exercise intensity increases, fat utilization decreases and eventually disappears, and carbohydrate utilization increases.

converted to glucose through the glucose–alanine cycle in a process called *gluconeogenesis*.

## LACTATE METABOLISM

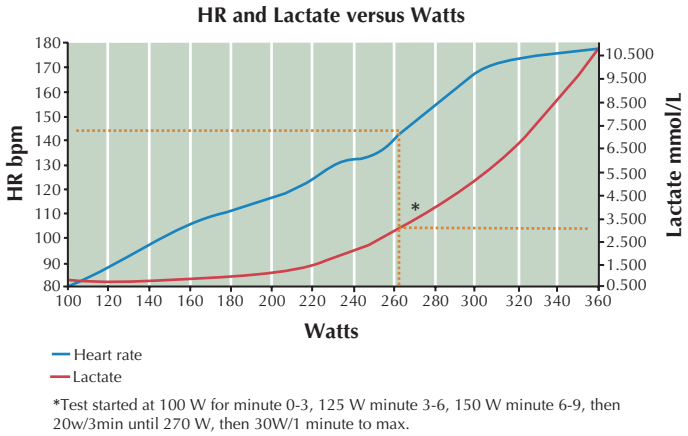
- Lactate is the byproduct of glucose utilization by muscle cells.
- The higher the glucose flux into the cell, the higher the lactate production, independent of oxygen availability.
- Historically, lactate was simply understood to be a waste product of anaerobic exercise, a waste product that may even crystalize and cause muscle soreness.
- Lactate studies date back from the 19th century when in 1863, a Nobel Laureate, Louis Pasteur, proposed that lactate was produced by lack of oxygen during muscle contraction.
- It was not until late in the 20th century when the importance of lactate and its key role in exercise and metabolism became clear, largely because of Dr. George Brooks, from the University of California at Berkeley, who extensively studied lactate for over 40 years. His work illustrates that lactate formation occurs under aerobic conditions, that lactate is not a waste product, and that lactate is the most crucial gluconeogenic precursor (new glucose generator) in the body. Approximately 30% of all the glucose used during exercise is derived from lactate "recycling" to glucose.
- High-intensity exercise is associated with high glycolytic activity in Type II muscle fibers, which results in the production of large amounts of lactate, even under aerobic conditions.
- During intense exercise, lactate production is manifold higher than resting levels, buildup exceeds clearance, and the release of hydrogen ions ($H^+$) associated with lactate can cause an important reduction of contractile muscle pH resulting in acidosis.
- This excessive accumulation of $H^+$, not only from lactate but also from ATP breakdown for muscle contraction (ATP hydrolysis), may interfere with muscle contraction at different sites competing with calcium ($Ca^{++}$) for troponin C binding site (a protein involved in muscle contraction regulation).

**TABLE 18.1 DIFFERENCES IN BLOOD LACTATE BETWEEN COMPETITIVE CYCLISTS OF DIFFERENT LEVELS***

| Workload | Junior Cyclist | Top Amateurs | Avg. Pro-Tour | World Class |
|---|---|---|---|---|
| w/kg | Blood La (mmol/L) | Blood La (mmol/L) | Blood La (mmol/L) | Blood La (mmol/L) |
| 3 | 1.3 | 1.1 | 1.1 | 0.8 |
| 3.5 | 1.8 | 1.3 | 1.2 | 0.8 |
| 4 | 3 | 2.3 | 2 | 0.96 |
| 4.5 | 6.6 | 3.5 | 3.2 | 1.8 |
| 5 | 10 | 7.6 | 5.8 | 3.1 |
| 5.5 | | 9.2 | 8.2 | 5.2 |
| 6 | | | | 8.9 |

Modified from San Millán I, González-Haro C, Sagasti M. Physiological differences between road cyclists of different categories. A new approach. *Med Sci Sports Exerc.* 2009;41(5):64-65.

*As observed, the lactate clearance capacity in world-class athletes is remarkable compared with even their professional peers.




*Test started at 100 W for minute 0-3, 125 W minute 3-6, 150 W minute 6-9, then 20w/3min until 270 W, then 30W/1 minute to max.

**Figure 18.6.** Physiologic characteristics and lactate threshold of a road cyclist. As the effort increases, the heart rate increases but serum lactate levels remain stable until at an effort of 264 Watts and a heart rate of 145, when lactic acid levels begin to exponentially rise. This indicates the lactate threshold.

- In addition, $H^+$ may inhibit $Ca^{++}$ release and reuptake from sarcoplasmic reticulum, which is involved in muscle contraction.
- These effects can result in decreased muscle contraction capacity, which can cause a considerable decrease in peak twitch force as well as maximum muscle shortening velocity and performance.
- Lactate is oxidized through a highly developed complex called *mitochondrial lactate oxidation complex (mLOC)* that comprises monocarboxylate transporter-1 (MCT1), its chaperone (CD147), a mitochondrial lactate dehydrogenase (mLDH), and cytochrome oxidase (Cox).
- The ability to clear lactate is probably the most important capacity in endurance athletes and the physiologic parameter that discriminates most athletes (Table 18.1).

## LACTATE THRESHOLD

- **Lactate threshold (LT)** is probably the most used training term by coaches and athletes worldwide.
- Commonly known as the *exercise intensity* or *blood lactate concentration* an athlete can sustain during a high-intensity effort for a specific period of time
- **Is sometimes referred to as *aerobic threshold*, which is inaccurate because lactate is constantly produced under aerobic conditions**
- LT is the point at which the rate of lactate production exceeds clearance and begins to accumulate in skeletal muscle, resulting in exportation to the circulatory system. At this point, energy production is dependent on glucose and glycogen.
- Higher LT as a percentage of $\dot{V}O_2$ max allows athletes to generate more power and sustain longer efforts.
- LT may be as low as 40%–50% $\dot{V}O_2$ max in sedentary individuals and may be 80%–90% $\dot{V}O_2$ max in highly trained aerobic athletes, who also achieve higher $\dot{V}O_2$ max.
- If two cyclists have identical maximal aerobic capacity, but one has LT at 70% $\dot{V}O_2$ max and the other has LT at 90% $\dot{V}O_2$ max, the latter has a significant physiologic advantage in a head-to-head endurance test.
- Compared to aerobic capacity, which is relatively fixed and related to genetic capabilities, LT is highly trainable. **Exercise physiologists have proven in multiple studies and in highly trained athletes that programs based on improving lactate thresholds are highly effective.**

### Determining the Lactate Threshold

- An exercise test can determine an athlete's LT.
- Lactate accumulates in the blood when requirements of energy production are so high that the body can no longer effectively clear the lactate from skeletal muscles.
- When LT is crossed, lactic acid builds in muscles and fatigue ensues. **The level of exercise intensity at which contractile forces in muscle begin to shut down is LT** (Fig. 18.6).
- The best way to determine LT for a cyclist is to ride a stationary bike at a given intensity for 45 minutes in the laboratory. The same type of test can be conducted for a runner using a treadmill. A small sample of blood is collected from the finger or earlobe and analyzed for lactic acid. The workload is then slightly increased. The sampling process is repeated every few minutes after the workload is increased (Fig. 18.7). Lactic acid concentration values are plotted against oxygen consumption and heart rate. When lactic acid levels precipitously rise, it is noted as the lactate threshold. Rise in respiratory rate corresponds to increasing lactate levels. Exercise is continued to a point of absolute exhaustion, which indicates $\dot{V}O_2$ max. Subsequently, a range of heart rates or power outputs is calculated to guide the cyclist in their training intensity (see Table 18.2).

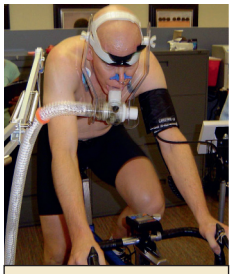
### Training Lactate Clearance Capacity and Lactate Threshold

- Lactate primarily produced in fast twitch muscle fibers as the byproduct of excessive glucose utilization is preferably cleared by mitochondria of slow twitch muscle fibers in the mitochondrial lactate oxidation complex (mLOC).
- Robust mitochondrial capacity in Type I muscle fibers is critical for optimal performance.
- Typical training mistake involves training at the LT to improve lactate clearance capacity. This is not quite accurate because mitochondria in slow twitch muscle fibers clear lactate through mLOC. Accordingly, it is important to train slow twitch muscle fibers to increase mLOC.
- Endurance training, in particular Z2 (see Table 18.2), is essential to improve Type I muscle fiber function, mitochondrial capacity, and mLOC.

- Training at LT is essential to improve glycolytic fibers (Type IIa) and their machinery ("Turbo"). LT training should be a key part of any comprehensive and competitive training program.

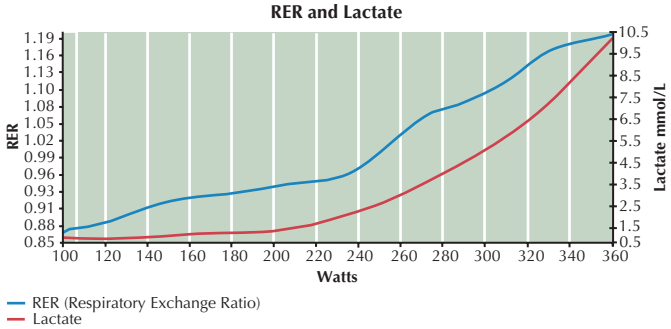
## Field Estimate of Lactate Threshold Heart Rate

- To determine lactate threshold heart rate (LTHR) for either cycling or running, complete a 30-minute time trial as follows. Find a course that is relatively flat. Warm-up as you would before a short race and then begin time trial. Start heart rate monitor immediately, preferably a monitor with an average heart rate mode. The effort of this time trial should be race-like—maximum intensity. Ten minutes into the time trial (20 minutes to go) press the "lap" button on the heart rate monitor so you have the average heart rate for the last 20 minutes when finished. This value is an approximation of LTHR.
- Pay attention to the heart rate whenever a burning sensation is experience in legs with onset of heavy breathing. LTHR refinement will occur with test repetitions, particularly by observing heart rate relative to breathing during workouts.



Performing lactate threshold and $\dot{V}O_2$ max on cycle ergometry in the performance lab.

Obtaining lactate blood sample from earlobe.




Rise in respiratory rate corresponds to a rise in lactate. As lactic acid builds in bloodstream, you must increase breath more rapidly to neutralize its effects.

**Figure 18.7.** Determining lactate threshold for cyclist.

## ECONOMY

- Compared to recreational runners and cyclists, elite runners and cyclists use less oxygen to hold a steady submaximal pace.
- World-class runners appear to move effortlessly, and elite athletes use less energy to produce the same power output as nonelite athletes.
- Economy refers to lower oxygen and caloric use to perform the same amount of work.
- Becomes more significant in long endurance events and racing (Fig. 18.8)
- **Fatigue can also negatively affect economy**. The longer the race, the more critical economy becomes in determining the eventual winner.
- **Just as with lactate threshold, economy is highly trainable**. Economy will increase with all aspects of endurance training, but sport-specific activity is critical: the only way to train swimming economy is by swimming, and the only way to train cycling economy is by riding a bicycle; cycling economy cannot be improved by rollerblading.

## Perspective

A word of caution relative to aforementioned discussion: it implies that fitness and race results can be easily quantified and predicted. You might think that scientists can take a group of athletes, predict who has elite potential, train them in a state-of-the-art facility, prod, probe, and analyze their performance. At the end of the day, they should be able to predict outcomes of all major sporting events. This could not be further from the truth. Although information about lactate threshold training is effective, it does not represent the "real world" of racing. Several variables are beyond scientific explanation. The incredible drive of certain athletes

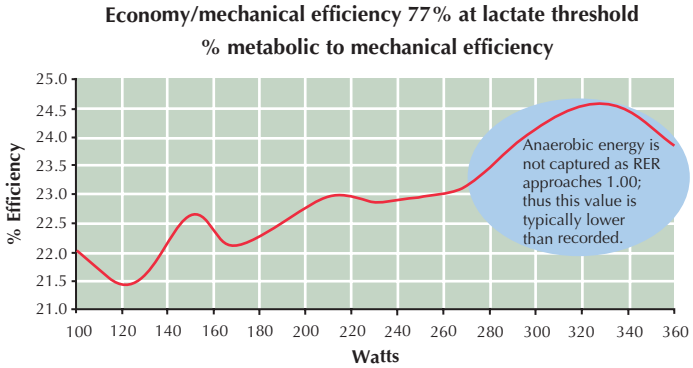



**Figure 18.8.** Mechanical efficiency. Mechanical efficiency reached a steady state of 23% before the lactate threshold. This indicates that overall economy is 77%.

**TABLE 18.2 EXAMPLES OF HEART RATE AND POWER TRAINING ZONES**

| RPE | Zone | Zone Description | HR | Power | % of LTHR |
|---|---|---|---|---|---|
| <10 | 1 | Active Recovery | <102 | <150 | 65%–81% |
| 10–12 | 2 | Endurance | 103–125 | 151–200 | 82%–88% |
| 13–14 | 3 | Tempo | 126–141 | 201–245 | 89%–93% |
| 15–16 | 4 | Lactate Threshold | 142–159 | 246–286 | 94%–100% |
| 17–18 | 5 | $\dot{V}O_2$ max | 160–165 | 286–350 | 101%–105% |
| 19–20 | 6 | Anaerobic Capacity | 165+ | 350+ | 106%+ |

Heart rate and power meter zones as % of lactate threshold for an athlete (see Fig. 18.7) whose lactate threshold occurred at HR of 145 and power of 264 watts; RPE (Borg Relative Perceived Exertion).

allows them to perform far beyond their abilities, while other genetically gifted individuals never seem to win any races.

## TRAINING GUIDELINES AND STRESS

- The *American College of Sports Medicine's Guidelines for Exercise Testing and Prescription* takes abstract concepts of aerobic training and puts them into practical guidelines that can be easily followed.
- **All successful training programs require a balance in frequency, duration, and intensity, also described as volume or workload.**
- Improvement of fitness never occurs in an unbroken continuum, and fatigue is the principal reason why. We will define each of these terms and discuss the interaction each has with the other.

### Frequency

- Refers to how often training sessions are conducted. Studies have revealed that training three to five times per week provides the greatest aerobic gains for time invested, but as your overall fitness improves, so must your training frequency. When training begins, three to five workouts per week will produce rapid improvements in fitness. Aerobic conditioning frequently improves by 10%–20% within a few weeks. If the same novice athlete were to train more frequently, instead of more rapid improvements, his/her fitness would decrease due to overtraining. Elite athletes training for a spot on the Olympic team may need to schedule 12–15 workouts each week and may only improve their fitness by 1% because they are so close to achieving their full potential.
- Ideal frequency of workouts is dependent on current adaptations of the body. For example, an experienced cyclist who has not trained for 3 weeks should restart his/her training regimen at a lower frequency than where he/she left off 3 weeks back to avoid the risk of overtraining.

### Duration

- Individual training sessions may vary considerably in length. Some may last several hours with a goal of improving aerobic fitness, whereas others are short in length and will promote either speed or recovery. As with frequency, the duration of a training session is dependent on an athlete's conditioning. As fitness improves, the athlete's body will be able to tolerate longer training sessions.
- The appropriate length of a workout is directed by the length of a race. If a runner never competes in a race longer than 5000 m, there is no real benefit of 4-hour runs. However, if the runner competes in ultra-marathons, which last 12–30 hours, he/she may train less frequently but will need to train for a much longer duration.

### Intensity

- Frequency and duration are much easier to quantify than intensity. Intensity is often referred to as volume or workload or simply, "hardness" of a workout. **Poor understanding of intensity is the primary reason that training programs are ineffective.** If you are performing your workouts with precise frequency and duration, but you allow them to become too hard, your aerobic fitness will suffer due to overtraining. If the training sessions are too easy, again your aerobic fitness will suffer due to inadequate stress.
- Athletes often describe their training in terms of volume. For example, if they rode 5 days last week for 1 hour each day, then they would relate that they trained for 5 hours. This actually describes the volume of training (frequency × duration) but does not quantify intensity. **A better summary of your training is workload, defined as a combination of volume and intensity**. Understanding how much effort or power went into each workout gives you a better understanding of training stress. Table 18.2 presents three individual ways to assess intensity. For example, if your goal is to ride at a tempo pace (Zone 3), at 90% of your LT for 30 minutes, then you could quantify this by using a heart rate monitor, a power meter, or the Borg Relative Perceived Exertion scale. Planning your workload around one of these scales is critical.
- Athletes need to determine their systematic training objectives to be able to appropriately plan for intensity training. Optimal endurance adaptations take place with long workouts at lower intensities. Numerous seasoned athletes use an easy "over-distance" pace ("Active Recovery" in Table 18.2) for endurance training, while a majority of less-intense athletes train at a slightly higher intensity ("Endurance" in Table 18.2), which can preclude optimal adaptations that occur at the slightly lower level. At least half, and usually more, of most training regimens should be spent at these lower intensities for most of the training cycle. A smaller proportion of training occurs at higher intensity levels, and the amount of time spent training at these higher levels increases as the training cycle moves toward peaking for competition. Tempo pace, hill workouts, and intervals are examples of higher intensity workouts. Race pace and racing occur at and above LT; hence, very little time is spent here until nearing peak performance, where 5%–15% of training may be performed at this level, with adequate recovery.
- Training intensity is the stressor that most athletes get wrong. Many athletes overtrain or train without appropriately varying intensity levels. If you exercise too intensively when you should be taking it easy, you will be tired on days when you should perform high-intensity training. All training days begin to look alike and shift toward mediocrity as easy runs become too hard and hard runs become too easy.

### Fatigue

- Fatigue is the reason we are all not world champions. When and to what extent we experience weariness is an optimal predictor of our fitness level. **Delaying the onset of fatigue is the primary reason for training**. The fittest athletes are those who can best resist the crippling effects of fatigue. As previously discussed, the causes of fatigue are lactate accumulation, glycogen depletion, and muscle failure; each of these causes of fatigue can be addressed by understanding the principles of lactate threshold training.

## TRAINING PRINCIPLES

### Overload

- Overload is to impose work stresses that are greater than those normally encountered. The purpose of training is to cause your body to positively change in order to better manage physiologic stresses of competition. **In order to stress your body, you must present a load that challenges its current level of fitness**. This load will initially cause fatigue, followed by recovery, and eventually an increased level of fitness or overcompensation. If workouts are of the right magnitude, i.e., slightly more than the body can handle, adaptation occurs and fitness steadily progresses (Fig. 18.9).
- **It is important to remember that overload occurs during workouts, but adaptation and overcompensation occur at rest. In other words, the potential for fitness is produced during training sessions, but the realization of fitness occurs during rest**. If you repeatedly deprive your body of rest, you will not continually improve, and you will also lose fitness—this is called *overtraining*. The biggest mistake most athletes

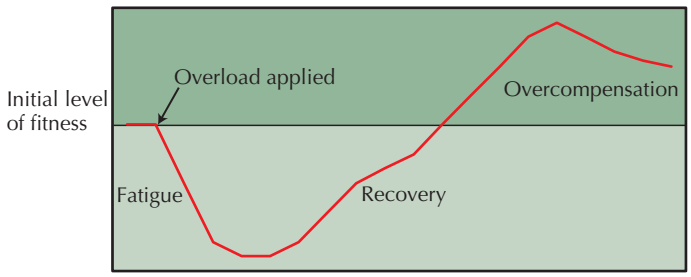



**Figure 18.9.** Overcompensation and improved fitness resulting from training workload.

make is disregarding their need to rest or reduce the intensity of training. Smart athletes will know when to back off in training or rest. However, when the load of training is reduced for prolonged periods, your body will adapt to this as well; this is called "being out of shape."

## Progression

- Progression involves a gradual and systematic increase in training intensity or volume as improvement occurs. If you have ever performed a workout or a race so intense that for days afterward you were too sore to even walk, much less run, then you have slowed your training progression. Such workouts violate the progression principle. Your body does not get stronger, rather you lose fitness and waste time and energy.
- You must gradually increase workloads with intermittent periods of rest and recovery. The stresses must be greater than your body is accustomed to handling. As the intensity of the workload is increased by small increments, usually 5%–15% every week to week and a half, you can avoid overtraining and injury and provide enough stress for adaptation to occur. Cumulative training volume should not exceed 5%–15% per year, and athletes training over 600 hours/year should probably not increase volume by more than 10% per year. Workload increases are primarily based on individual needs, particularly in terms of intensity (Fig. 18.10).

## Periodization

- **Periodization** is the structuring of training hours for a given cycle to produce a progressive increase of training stress and performance. Several athletes use training cycles, frequently dividing the competition year into 4-week cycles (meso or microcycles), although experienced athletes may individualize the lengths. Each cycle is planned to stimulate the appropriate training response for that part of the year relative to peak performance and competition. Many athletes will increase intensity in a stepwise manner over the first 3 weeks of a microcycle, then scale back hours and intensity during the final week of the cycle. Alternatively, experienced athletes individualize this pattern. On the other hand, a training year may be divided into different stages or cycles (macrocycles) such as base, building or intensity, and peak and/or race (Fig. 18.11). Periodization takes place from cycle to cycle (micro and macro).
- The basic premise of all periodization programs is that training should progress from general to specific. Early in the season, training is focused on maintaining weight, strength training, and general aerobic fitness. Later in the season, more time is spent focusing on a specific sport. The reason for dividing the year into specific periods is to emphasize specific aspects of fitness while maintaining others developed during earlier periods (Fig. 18.11). Trying to simultaneously improve all aspects of training is impossible for an athlete to handle. Periodization

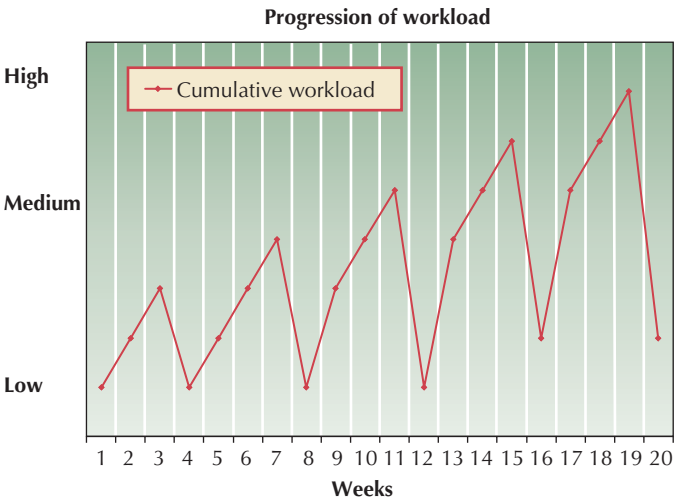



**Figure 18.10.** Example of weekly cumulative workload progression.

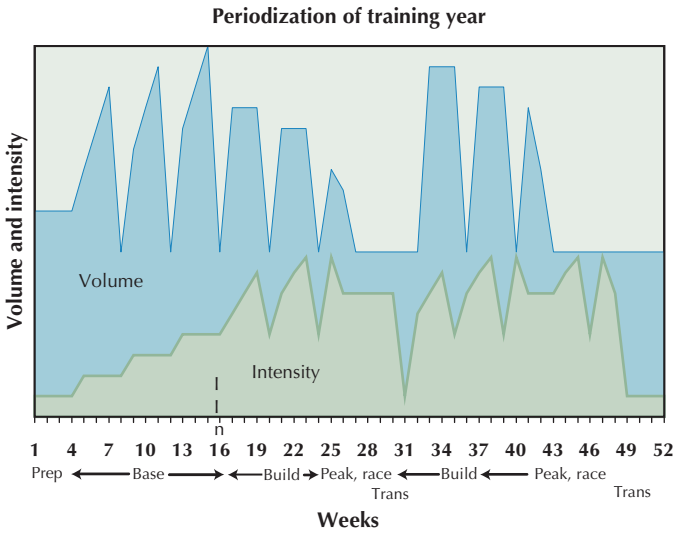



**Figure 18.11.** Hypothetical training year divided into periods showing the interplay between volume and intensity. Prepare to train. Base is to establish speed, strength, and endurance; Build is to increase intensity and improve weaknesses; Peak is to taper for race readiness; Race is for priority races; and Transition is the recovery period.

of training helps to reduce injury, maintain flexibility, and limit burnout. Scientific evidence to support such training programs is limited, but logically, it does make sense. In addition to the logic, a majority of the world's elite aerobic athletes follow these principles.

## Individualization

- Many factors contribute toward an athlete's capacity to handle a given workload. These factors are not purely physiologic but are psychological, socioeconomic, environmental, and genetic. Each of these factors has the ability to improve or impede fitness. Periodization of training addresses many of these individual variables. Several books have been written on the subject of periodization. Simply put, two athletes should not perform exactly the same workouts. Also, an athlete should not perform the same training each day, or each week, or each month. Constantly varying workouts during the week and over the year is the key to progressing in your aerobic fitness.

### Principle of Specificity

- Training specificity is best accomplished through a training program that involves movement patterns and speeds similar to a given exercise task or competition goal (e.g., bikers must bike and runners must run). It contributes to exercise economy, has both neuromuscular and metabolic components, and is one of the most important conditioning principles.

## SUMMARY

- Fatigue is a significant determining factor of our fitness level. The primary reason for training is to delay the onset of fatigue. The fittest athletes are those who can best resist its crippling effects. We know that lactic acid is always being produced, even at rest. A consistently low level of lactic acid in the serum during moderate exercise implies that the body can clear it from the blood as fast as it is generated. The point at which lactic acid levels begin to rise in the serum depends on $\dot{V}O_2$ max and LT; this is the point at which lactic acid production is greater than the body's ability to clear it and when effort becomes disabling.
- $\dot{V}O_2$ max alone is not a good predictor of performance. It is the person who can maintain the highest value of $\dot{V}O_2$ max for an extended period of time who stands the best chance of winning a race. This sustainable high value is equivalent to LT. The purpose of aerobic conditioning is to cause your body to positively change in order to better manage the physiologic stresses of competition. In order to appropriately stress your body, you must present a load that challenges its current level of fitness. This load will initially cause fatigue, followed by recovery, and will eventually lead to an increased level of fitness or overcompensation. If the workouts are of the right magnitude, slightly more than the body can handle, adaptation occurs and fitness steadily progresses.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# RESISTANCE TRAINING

# 19

William J. Kraemer • Gwendolyn A. Thomas • Disa L. Hatfield

## GENERAL PRINCIPLES

- Resistance training is the most potent form of exercise to strengthen tissues and help prevent injury and improve sports performance. Appropriately prescribed and implemented resistance training programs are necessary to achieve these goals.
- The size principle of motor unit recruitment demonstrates that the recruitment of motor units and their associated muscle fibers occurs in an orderly manner from low- to higher-threshold motor units, and as one climbs the recruitment ladder, greater amounts of force can be produced.
- Strength (maximal force production) is a key factor in highly trained athletes; however, other trainable variables (e.g., power [force ÷ time] or rate of force development, local muscular endurance, and hypertrophy of muscle) are equally important.
- For athletes, a total conditioning program (i.e., resistance training, aerobic conditioning, flexibility, speed and agility, plyometrics, nutritional components, body composition, and psychological aspects) is needed; however, such a program must be appropriately designed or else, it could result in incompatibility (e.g., too much endurance training can interfere with power development).

### The Needs Analysis

- The development of a resistance training program depends on matching individual needs of an athlete with demands of the sport.
- A sport must be analyzed by its biomechanical movements (e.g., isometric or dynamic; concentric or eccentric) that have to be reflected in the resistance training program. If a range of motion is not trained, then the tissues become more susceptible to injury owing to a lack of adaptation.
- The metabolic profile of a sport can range from a cross-country runner to a wrestler. What is the basic metabolism that predominates? Most sports played at high school or collegiate levels have high adenosine triphosphate–phosphocreatinine (ATP–PC) and glycolysis involvement supported by a basic aerobic endurance component.
- Understanding the injury profile of a sport is important to strengthen tissues and movements of the affected joint.
- Testing is needed for informed decisions regarding needs of an exercise prescription.

### Specificity of Training

- The specificity of training is related to the recruitment of muscle tissues reflected in the "size principle." Considering the need for greater force and power demands, more motor units must be recruited. Recruitment of motor units always occurs in an orderly manner from lower-threshold motor units composed of Type I slow twitch muscle fibers to higher-threshold motor units (i.e., Type II muscle fibers, fast twitch).
- The amount of force or power needed determines how much of the muscle is activated and thus trained in a given exercise. Heavier resistances in a program ensure that all muscles have been trained.
- It is important to use resistances and velocities across the entire force–velocity spectrum (i.e., light to heavy, reflecting high to low velocities). Training for maximal power should be performed with no deceleration except for gravity in the exercises performed (e.g., Olympic-type lifts).
- Ultimately, the specificity of training is related to the physiologic systems that are used to support the motor unit recruitment demands of an exercise.

## DEVELOPMENT OF A WORKOUT

- A key factor in the prescription of exercises in resistance training is the acute program variable and choices made because these determine the exercise stimuli.

### Choice of Exercise

- Exercise choice involves the type of weight-training equipment and type of muscle action that will be used in a workout.
- Exercises are classified as structural (i.e., involving multiple joints) or body part (i.e., involving an isolated joint).
- **Structural exercises** include whole-body lifts that require coordinated actions of muscle groups and joint movements (e.g., closed kinetic chain exercises). Most primary or core exercises are structural (e.g., squats or power cleans). These should be included in the training program of every athlete.
- **Body-part exercises** attempt to isolate a particular muscle group or joint (e.g., bicep curl). Most assistance exercises can also be classified as body-part or single-joint exercises.

### Order of Exercise

- Larger muscle group exercises should be performed first to allow higher resistances to be lifted.
- New or complex exercises should be performed first in a workout to allow development of better exercise techniques owing to less fatigue.
- When creating a circuit weight-training workout, one has to decide whether the order and choices are going from arm to or arm or leg to leg exercises or arm to leg exercises or upper- to lower-body exercises.
- The fitness level of an athlete and exercise tolerance are important considerations while designing a workout or training program.

### Number of Sets

- The number of sets in a workout is part of a volume calculation (e.g., sets × rep × resistance = total kgs) and is related to the training goals of a program (e.g., low or high workout volume for different workouts or training cycles).
- Three to six sets are used to achieve optimal adaptations. Not every exercise in the workout must have the same number of sets.
- To date, no single-set system has been shown to be superior to multiple-set programs and should only be used to reduce the volume of a workout or training cycle.
- Training for muscular power is accomplished with more sets and fewer repetitions (e.g., six sets of three repetitions each) to optimize the power output of each set.

- During needs analysis, the practitioner will evaluate the athlete and decide the primary goal of the resistance training program; this training goal will be used to determine specific loads, rest, and repetition arrangements.

## Length of Rest Periods

- The length of rest periods between sets and exercises is crucial for all exercise prescriptions.
- Rest periods determine how much of the ATP–PC energy source is recovered.
- Lactate is a buffer and not responsible for the acid–base disruption or fatigue and does not play any role in muscle soreness; in fact, lactate is essentially just a good marker of metabolic demands. Nausea and dizziness caused by excessive physiologic stress should not be equated with a "good workout."
- Short rest periods can lead to greater psychological anxiety and fatigue and higher metabolic demands (e.g., high lactate production, pH decreases, and $H^+$ increases). As a result, certified strength and conditioning specialists (CSCS) may have to gradually expose athletes to such workouts and use no more than 2 per week.
- Rest periods of <1 minute is classified as being very short; 1.5–2 minutes as short to moderate; 3–5 minutes as long; and >5 minutes as very long rest periods. As the resistance gets heavier, longer rest periods are needed.
- Popular extreme commercial programs have a tendency to repeat too many of such short rest periods per week, and this can promote overreaching/overtraining.

## Amount of Resistance

- The amount of resistance used for each specific exercise is the most important variable (i.e., size principle).
- Resistance is a major stimulus related to changes in measures of strength, power, and local muscular endurance.
- The amount of resistance must be selected for each exercise in a given resistance training program.
- A repetition maximum training zone **(RM zone),** allows only a specified number of repetitions to be performed targeting a 3-repetition range (i.e., zone) for loading. Changes can be made for each set in a workout. If an RM zone is 3–5 RM, and the athlete can only perform 2, the resistance is lowered; similarly, if s/he can perform 6, the resistance is increased to stay within the zone. It is a way to set a resistance load for an exercise.
  - Six or fewer very-heavy RM resistances appear to have the greatest effect on strength, which contributes to the force component of a power equation. In addition, it recruits the maximum number of motor units and thus stimulates hypertrophy of the entire motor unit array of a muscle.
  - Resistances in the 8–10 RM range contribute to both strength and hypertrophy.
  - Twenty or more RM resistances improve local muscular endurance measures (e.g., repetitions at 75% of 1 RM).
  - This RM zone continuum makes it possible to develop a specific feature of muscular performance to varying degrees over a range of RM resistances.
  - The RM zone cues loading and should not be done to failure as a target end point because this can cause increased compression on joints and fosters overreaching.
- Another standard method of determining resistance for specific exercises is to use **percentages of 1 RM** (e.g., calculating 70% or 85% of 1 RM tested to determine the training load). This method requires regular testing of maximal strength in various lifts used in the training program or prediction of 1 RM by using an equation (e.g., Epley Eq. 1 Repetition Maximum Prediction = 0.033 (reps) × [repetition weight] + [repetition weight]; used for major muscle group exercises).
- The relationship between possible RM and percentages of 1 RM varies with the amount of muscle mass needed to perform an exercise (e.g., leg presses require more muscle mass than do knee extensions).
- When athletes use machine resistances with 80% of 1 RM (previously thought to be primarily for strength-related prescriptions), the number of repetitions that can be performed is >10, particularly for large muscle group exercises such as leg presses.
- Percentages were developed for free-weight exercises and do not translate very well to machine exercises, particularly as the muscle mass used increases.

## Frequency of Training

- The frequency of training can range 1–6 days per week and is related to the required volume of work or recovery time.
- Elite athletes may require periodized training frequencies of 4–5 days to achieve gains over short periods of time.
- Athletes may train twice daily with 4–6 hours of rest between workouts to reduce volume within a single workout so that the quality (intensity) of workout can be maintained at the highest level.
- Training frequencies of more than twice a week typically involve different training programs and do not repeat the same program during each workout.
- A minimal frequency of two sessions per week for a given exercise is needed for any improvement.
- Certain types of variation (i.e., periodization) must be employed when consecutive training days are used.
- Progression in frequency depends on the phase of the training cycle, fitness of the athlete, goals of the program, training history, exercise selection, training volume, intensity, recovery ability, and nutrition.
- Excessive soreness indicates a lack of recovery, and subsequent workouts must be adjusted to reduce this stress.
- Training with heavy loads increases the required recovery time before subsequent exercise sessions.

## Considerations for Women

- The same general principles apply to the training of women and men. By understanding specific gender differences (e.g., weaker upper body in women), the required elements can be added to a training program. The maximal mean total body strength of an average woman is 63.5% of that of an average man; moreover, the isometric upper body strength of an average woman is 55.8% of that of an average man, and the isometric lower body strength of an average woman is 71.9% of that of an average man.
- Some initial evidence indicates that strength gains in women may plateau after 3–5 months of training. This plateau may be more pronounced in the upper body, where the absolute muscle mass is less than that in men. Thus, emphasis on development of upper lean body mass in women may be warranted for sports wherein upper body strength is a limiting factor of performance.
- In certain cases, women may need much more maximal strength to optimize power development.
- Body somatotype can have a dramatic influence on muscle strength and size gains, which is reflective of the number of muscle fibers in various muscles. Target goals should be set by accounting for not only muscle but also the importance of strengthening other tissues such as connective tissues as well as training other physiologic systems and their organ structures (e.g., cardiovascular and endocrine systems).

### Training Cycles

- In classic program time frames, most sport coaches consider training as comprising three basic time frames: off-season, preseason, and in-season. Certified strength and conditioning specialists (e.g., NSCA-CSCS) view such older forms of training delineations to be defied by a periodized training schedule. It consists of a macrocycle (the annual cycle or training phase), a mesocycle (3–6 month phases), and a microcycle (1 day to 4 weeks). Each of these depend on the periodization model used, which is based on the need for quality training and the required rest and recovery times in order to mitigate any overreaching or overtraining syndromes.
- Sport and practices can induce physical and mental trauma that can be problematic to optimal training. For targeted goals in a strength and conditioning program, this can contribute to the development of nonfunctional overreaching, and in extreme cases, overtraining, which can last for over a year and can permanently diminish a competitive career.
- Quality of training is vital for effective use of time and variation in the training stimulus (e.g., resistance used or volume of workouts) along with rest days allow effective adaptations and resilience. It is important to educate coaches regarding the value of rest days in improving performance.

### Variation in Training

- The principle of variation relates to changes in characteristics of a training program to match the changing program goals as well as to provide a changing target for the body's adaptation. For experienced lifters, it is prudent to design resistance training program cycles that vary as often as every 2–4 weeks (linear microcycles).
- New terminology: overreaching—an intentional hard training phase to push an athlete's performance for a short period of time, followed by a remarkable reduction in stress, which produces an increase rebound (super compensation)
- Nonfunctional overreaching—mistakes are made in training that diminish an athlete's performance, but the exact cause is not known and recovery is possible in days or weeks only if rest is allowed
- Overtraining—is less common than previously claimed and most of the time is due to non-functional overreaching that can be caused by mistakes in exercise prescription and/or a medical issue causing excessive stress (maladaptive state); performance remains decreased for a few weeks to a few months
- Certain variables do meet a genetic maximum for development, and not all plateaus in training are a sign of overreaching or overtraining because there is a limit to training progress; this underscores the importance of realistic training goals and targets for improvements.

## GENERAL PREPARATORY CONDITIONING

- Acute program variables must be carefully used to design a workout by using the program concepts outlined below. Effective strength and conditioning programs must be designed, implemented, and monitored by experienced and certified strength and conditioning specialists (NSCA-CSCS) who work closely with the sports medicine team.
- A general preparatory conditioning phase (2–4 weeks) is typical of linear periodization programs (crucial at the start of a training program for beginners); it can also be helpful for beginners who are using a nonlinear program.
- Appropriate progression considers progressive overload, specificity, and variation to match dynamic training goals and fitness levels of an athlete.
- A major issue is doing "too much, too soon" in a strength and conditioning program and overshooting an athlete's ability to recover from a workout or to mentally cope with a training program.
- Educational aspects of resistance training, appropriate exercise techniques, aerobics, and nutrition should be stressed. Resistance loads should be light to moderate because the ability to concentrate with less fatigue helps learning.

### Periodization of Training

- Over the past 25 years, the concept of periodization of training has taken several different forms, and each of them has been effective in its own way by meeting the needs for variation, rest, and recovery. Each individual adapts and recovers differently to a resistance training program. The most effective programs monitor progress with testing and develop individualized programs.
- Regular monitoring and testing is needed to evaluate day-to-day progress and progress over each training cycle. If testing is performed, one has to adhere to the credo "if you test or monitor it, you have to manage it," indicating that the program must be altered or not based on testing results.
- This requires integration of sports medical teams (physicians, dieticians, athletic trainer, physical therapists, and sport psychologists) and strength and conditioning teams, along with the use of evidence-based practices as well. Integration with sports medicine elements is vital for full program connections.
- Again, periodized training programs are bracketed by time, as noted by a macrocycle (1–4 years), mesocycle (3–6 months), and a microcycle (1 day to 1–4 weeks depending on the periodization model used).

### Classic "Linear" Format

- Decreases training volume and increases training intensity as a program progresses
- Used in athletes who are peaking for a single performance in a strength/power sport (e.g., weightlifting or field events such as shot and discus) in a major competition (e.g., World Championships or Olympic Games)
- Classic periodization methods use progressive increases in intensity with a small variation in each 2–4-week microcycle. In order to move though a microcycle faster to get to other stimuli linear periodization now has also cut the length of the microcycle down to 2-week segments and some even to 1 week. This allows even more variation that is typical of what can be noted in nonlinear programs. A classic 2-week microcycle linear periodized program is outlined in Box 19.1. Certain variations can be observed within each microcycle considering the repetition range of each cycle, but the general trend noted in these programs is a steady linear increase in intensity of a training program.
- In addition, resistance loading can take the form of percentages of the 1 repetition maximum 9 RM during a lift. However,

**BOX 19.1 GENERAL EXAMPLE OF A LINEAR PERIODIZED PROGRAM WITH 2-WEEK MICROCYCLES**

**Microcycle 1:** 3–5 sets of 12–15 RM zone
**Microcycle 2:** 4–5 sets of 8–10 RM zone
**Microcycle 3:** 3–4 sets of 4–6 RM zone
**Microcycle 4:** 2–3 sets of 1–3 RM zone
**ACTIVE REST CYCLE** of approximately 1–2 weeks before the start of a new mesocycle of training

*RM*, Repetition maximum zone.

this is typically used in exercises and in athletes who are very familiar with their maximal 1 RM limits as with competitive weightlifters and powerlifters. Otherwise, an RM zone can be used.

### Special Consideration for Athlete Populations

- Each 8–16-week program is called a *mesocycle* (composed of microcycles), and a 1-year training program is typically composed of several mesocycles. Each mesocycle attempts to increase muscle hypertrophy, strength, local muscular endurance, and/or power toward an athlete's theoretical genetic maximum. Thus, the linear method of periodization is theoretically based on the development of muscle hypertrophy and improved nerve function and strength/power. This process is repeated with each mesocycle in order to make progress in training programs.
- Training volume starts with a higher initial volume, and as the intensity of the program increases, volume gradually decreases. Drop-off between intensity and volume of exercise can decrease as the training status of an athlete advances. Advanced athletes can tolerate higher starting intensities and volumes of exercise during heavy and very heavy microcycles.
- Increases in the intensity of periodized programs begin the development of required adaptations in nervous system for enhanced motor unit recruitment. Adaptations develop as a program progresses, and heavier resistances or velocities of movement are used. Heavier weights demand involvement of high-threshold motor units in the process of force production. Moreover, exercises with higher power output and training velocities use specialized motor unit recruitment patterns.
- Athletes must be careful to not progress too quickly to high volumes using high intensities. Pushing too hard can lead to serious overreaching or at the worst overtraining syndromes. Although it takes a great deal of excessive work to produce true overtraining effects, highly motivated athletes can easily make mistakes out of sheer desire to achieve gains and see what is called *nonfunctional overreaching*, where performance diminishes but can be recovered within days if discovered by a strength and conditioning professional. However, in case of true overtraining, it may take weeks to recover performance levels from mistakes made during an exercise prescription program and see any progress in training and performance. It is important to monitor the stress of workouts. Rest between training cycles (active rest phases) allows time for required recovery so that overreaching to overtraining issues are reduced if not eliminated; 1–2 days of complete rest during a training week has been shown to reduce the chance of overtraining.
- High-volume exercise in early microcycles marks the initiation of a linear periodized program; thus, late cycles of training are linked to early cycles by changing the stimulus of the training. Once basic strength has been developed, specialized power training programs allow separate development of power capabilities in muscles.
- Maintenance or continued improvement in strength is needed for power development as loading is reduced. A major error in certain training programs, particularly in those for women, is to drop-off the high resistance loading that is required for strength development for prolonged periods while focusing on power by using lighter and more ballistic loading schemes. This has led to the use of 2-week microcycles for power development and different linear progression strategies.

## "Nonlinear" or Undulating Format

- Developed out of the need to periodize throughout multiple competition sports wherein one peak does not necessarily produce success; shown to be very effective in college and high school settings wherein schedule influences are dramatic
- Useful in sports with long seasons wherein success during the season and qualification for major tournaments and competitions are important
- Varying training volumes and intensity so that fitness gains are achieved over long training periods
- Training volume and intensity are varied using different workouts for each day to address a specific trainable feature (e.g., strength, power, muscle size, and local muscular endurance); this is achieved by using percentages of 1 RM or near RM training zones, varying volumes of work, varying rest periods, in varying orders, and varying supplemental exercises.
- Nonlinear or undulating periodized programs are designed to maintain variation in training stimuli without holding an athlete to strict phasing of linear periodized programs.
- Nonlinear programs facilitate easier implementation of a program when schedule or competitive demands over long periods limit sequential variation in a linear manner. In addition, they allow variation in intensity and volume within each 7–14-day cycle over the course of a training program (e.g., 16 weeks).
- Typically, changes in intensity and volume of training vary within each 7–14-day cycle. For example, in 12–16-week programs, an active rest phase of 1–2 weeks may be followed by another nonlinear cycle or even by a linear program, if desired. Box 19.2 offers an example of loading or intensity variation over a 5-day rotation during a 12–16-week mesocycle.
- Important to this concept is the fact that one can also have a different priority for each mesocycle, thereby allowing additional workouts to emphasize one style and at the same time, keeping a lot of variation in the program (e.g., a power mesocycle or a strength mesocycle wherein additional workouts will be directed toward such types of workouts over 16 weeks).
- Variation in training is much greater within each 5-day workout over 10 days. Intensity spans a maximal range of 15 RM (possible 1-RM sets versus 15-RM sets). In addition, workout cycles may include other types of training protocols (e.g., plyometric protocols or 6–10 sets of 2–3 repetitions at 30% of 1 RM for power and velocity training days). The key element is variation during an acute 1–2-week cycle within a 12–16-week mesocycle.
- An athlete trains different components of muscle within each microcycle. Although nonlinear programs attempt to train various components of the neuromuscular system within the same cycle, only one feature (e.g., power or strength) is trained during a single workout.
- The mesocycle is completed within a certain number of workouts (e.g., 48) instead of a certain number of training weeks. Workouts rotate between different styles for each training session. If an athlete misses a Monday workout, the rotation order is simply pushed forward. In this way, no workout stimulus is missed during a training program.
- Primary exercises are typically periodized. Similarly, supplemental exercise movements (e.g., hamstring curls or abdominal exercises) can be periodized as well.
- Nonlinear programs accomplish the same effects and are superior to linear programs in certain situations. The key to

### BOX 19.2 GENERAL EXAMPLE OF A NONLINEAR PERIODIZED PROGRAM PROGRESSION

This program uses a 5-day rotation:
**Monday:** 2 sets of 12–15 RM zone
**Wednesday:** 6 sets of 1–3 RM zone
**Friday:** 3 sets of 4–6 RM zone
**Monday:** Power day: 10 sets of 2–3 repetitions at 30% of 1 RM
**Wednesday:** 4 sets of 8–10 RM zone

*RM*, Repetition maximum.

success in all training programs appears to be variation. Different approaches can be used over the year to reach the training goals of an individual athlete in his/her specific sport.
- The concept of "flexible" nonlinear periodization was created to individually optimize the quality of each workout and not just go through paces. Not all athletes are always ready to perform a scheduled workout during a training program owing to a lack of recovery from the previous day, sport demands of practice or competition, illness, or injury, and therefore, the workout to be used is decided on each single day to determine if a scheduled workout will work in a nonlinear periodized schedule. This can involve some preworkout testing (e.g., vertical jump to see if one can train power), progression of a workout log to see if the number of reps for each set with a prescribed resistance can be performed and if not, the workout must be typically changed to a lower volume or lighter resistance or a rest day. This places additional responsibility on the strength and conditioning professional to develop a system to accomplish this but is more effective in producing more "quality" workouts in a program.

### Training Adaptations

- Different programs result in different training adaptations and physiologic responses. Specificity of training applies to all features of each exercise used in the conditioning program (e.g., each specific workout has varying effects on various physiological systems, signaling molecules, and gene expression).
- Development of muscle size is dependent on the number and type of muscle fibers. Moreover, body somatotypes (ectomorph, mesomorph, and endomorph) influence the gain potential for strength and size, whereas limb lengths and body dimensions affect several performance outcomes.
- Heavier weights have a higher neuroelectric Hz stimulus to all muscle fibers that form different motor units and promote greater hypertrophic responses across the fiber array in a muscle and thus support the need for heavier loadings as part of a periodized program for both men and women.
- Strength increases are specific to the mode of muscle action used during training.
- Most sport skills develop in <0.2 milliseconds (rate of force development), which makes the ability to produce force in short periods of time vital to performance, which requires power training.
- Speed repetitions, wherein an athlete holds on to weight (e.g., bench press or military press), should not be used to train for muscular power because an athlete's body tries to decelerate mass by activating antagonist muscles and reducing activation of agonist muscles. Instead, power exercises such as Olympic high pulls, power cleans, isokinetic exercises, pneumatic exercises, and medicine ball exercises should be used to promote power development.
- Typically, velocity of movement is associated with the type of resistance used, choice of exercise, and exercise modality. Terms such as speed, strength, and power relate to rapid development of force at high speeds of movement. Continuum of velocities is used in conventional resistance training, from very slow concentric movements (e.g., 1 RM lift) to higher-speed power movements (e.g., 30%–50% of 1 RM). Slower, high-resistance training enhances development of high force but has little carryover to faster velocities or rate of force development; conversely, high-speed training has little carryover to development of slower speed force.

## PHYSIOLOGIC COMPATIBILITY OF EXERCISE TRAINING MODES

- Aerobic endurance exercise (e.g., running and cycling), when concurrently performed with resistance training at a substantially high intensity and volume, has the potential to compromise increases in strength and power and limit Type I muscle fiber size increases.
- For example, training incompatibility for strength and power is noted with extremely high-intensity long-distance running.
- For most sports, the phenomenon of interference can be accommodated by prioritization of goals. Prioritization involves working on the most important training goals first and maintaining others. For example, one training phase may focus on power development, but high levels of strength also must be maintained to optimize power.

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Available at www.ExpertConsult.com.

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# 20 FLEXIBILITY

Ian Shrier

## GENERAL PRINCIPLES

- The term *flexibility* is often clinically used as a synonym for range of motion (ROM) around a joint.
- Both muscles and ligaments can limit ROM.
- When ligaments limit ROM, it is referred to as a decrease in mobility; flexibility is usually reserved to refer to limited ROM caused by the muscle–tendon unit.
- Flexibility depends on both muscle stiffness (force required to stretch a muscle) and the stretch tolerance of an individual (amount of discomfort a person feels when a muscle is stretched).

### Definitions of Related Terms

**Stretching:** an activity wherein a person purposefully attempts to increase ROM by applying a longitudinal force to a muscle

**Elastic effects:** increase in tissue length that immediately returns to original length when stress is removed

**Viscous effects:** increase in tissue length that is dependent on time and returns to the original length at a slow rate (i.e., it is reversible); viscous effects occur because molecules move when force is applied over time, and thus, the return to original length is not immediate

**Viscoelastic effects:** a combination of viscous and elastic effects

**Plastic effects:** a permanent change in the molecular structure of a tissue as that which occurs when force is applied to a plastic sheet without completely tearing it. Plastic deformation indicates that damage has occurred to a tissue—it does not occur with appropriate stretching (i.e., the ROM returns to normal within a reasonable time frame after appropriate stretching).

**Flexibility training:** program of stretching exercises designed to increase ROM of targeted joints to a desired level or to maintain that level once it is attained

### Specificity of Flexibility Training

- The immediate gain in ROM with stretching is mostly limited to the muscle being stretched, but there is some increase in ROM in the contralateral limb as well. This suggests that a neurologic reflex is one component of the mechanism for the effects of an acute stretch.
- If one stops moving a joint, one loses flexibility. It is unknown how much movement is necessary to maintain flexibility, but it would seem logical that any muscle that is not moved through a particular ROM for a long period of time will lose that ROM.

### Effects of Temperature on Flexibility Training

- Most studies have suggested that the effectiveness of stretching increases when the tissue is warmer.
- The most effective way to increase muscle temperature is with muscle activity, although deep heating methods (e.g., ultrasound) can be effective as well.
- Superficial heat is not an effective method to warm up deep muscles.

### Age and Gender Differences

- Flexibility decreases during the first year of life and then begins to increase until approximately 5–8 years of age. This is followed by a steady decline in flexibility in both boys and girls until 12–14 years of age (near puberty). Subsequently, flexibility increases again until approximately 20–24 years of age, which is followed by a slow but steady decline. These changes appear to coincide with changes in levels of activity, but whether or not this relationship is causal remains to be determined. For example, one begins to walk at approximately 1 year (flexibility begins to increase), and one starts school around 5–8 years of age, which is associated with increased sitting (decreases in flexibility). Flexibility may increase again at the age of 12–14 years as general activity increases with walking to secondary school and social relationships but may decrease by the age of 20–24 years as people enter the workforce and again become less active.
- Although females are generally more flexible than males, differences within the general population are small. The general perception that females are much more flexible may occur because females participate in activities and sports that include a large amount of flexibility training (e.g., dancing or gymnastics). There are two additional reasons why the increased flexibility in females may not be caused by sex hormones: (i) it is seen before puberty and (ii) the increase in flexibility that occurs around the time of adolescence occurs earlier in boys than in girls (but girls enter puberty earlier than boys).

### Stretch Reflex

- The stretch reflex is a protective reflex mediated by muscle spindles; it causes stretched muscles to contract and thus prevents excessive ROM.
- The stretch reflex is the primary reason certain people advise against ballistic stretching (bounce stretching). As stretch is released during the bounce, muscles contracts and then subsequent stretch occurs against an eccentrically contracting muscle. This theoretically increases the risk of injury, but there are no studies that actually compare rates of injury with ballistic stretching versus other types of stretching. An alternative view is that a force involved with appropriate ballistic stretching is very small and much less than what occurs during regular sport. Therefore, if an injury occurs during a ballistic stretch, some argue that it would likely have occurred during the sport as well.

## ROLE OF FLEXIBILITY IN INJURY PREVENTION

### Optimal Flexibility

- A graph of the relationship between flexibility and the risk of injury would be U shaped. Both individuals who are inflexible and those who are extremely flexible are at a higher risk of injury than are those with an intermediate level of flexibility. Individuals with increased flexibility may be representative of a hypermobility group (because of their ligaments) rather than a hyperflexible group (because of their muscle–tendon unit) because these two features are sometimes difficult to differentiate.
- The U-shaped curve is based on cross-sectional data and does not mean that inflexible individuals are at a reduced risk of injury if they begin to stretch before exercise. First, there may be some other factor associated with inflexibility that is responsible for the risk of injury, and this other factor might not be affected by stretching. Second, the *immediate effects of stretching*

*are opposite to the long-term effects of stretching* (see following text). In general, cross-sectional data on flexibility refer to long-term effects and thus provides minimal information on effects of stretching immediately before exercise.

- Stretching immediately before exercise: Immediately after an acute bout of weightlifting, muscles are fatigued and weaker. Similarly, but perhaps through a different mechanism, muscles are also weaker after stretching. This would not be expected to reduce the risk of injury, and most studies have shown no change in the risk of injury when a pre-exercise stretching intervention is initiated.
- Regular stretching: If a person does weightlifting over weeks to months, the muscles become stronger. Similarly, but perhaps through a different mechanism, muscles are also stronger after weeks to months of stretching. This is expected to reduce the risk of injury. Three studies examining the effects of regular stretching on the risk of injury have reported beneficial effects, but only one was statistically significant. Beneficial effects of yoga may work through this mechanism, although certain types of yoga also include strengthening and balancing exercises in addition to stretching exercises. The potential psychological benefits of yoga (and stretching in general) could be explained through other possible mechanisms.
- This area warrants a lot of additional research. For example, most studies have examined lower-intensity activities such as jogging. Whether these results can be generalized to higher-intensity sports such as basketball remains to be determined.

## ROLE OF FLEXIBILITY IN PERFORMANCE ENHANCEMENT

- As one must differentiate the effects of different types of stretching on the risk of injury, one must differentiate the effects of an acute stretch with those of regular stretching.
- An acute stretch weakens the muscle, and it cannot produce as much force or contract as rapidly.
- If one stretches regularly over weeks, both force and velocity of contraction increase.
- Different sports have different requirements, and performance does not solely depend on force and velocity of contraction.
- In the running gait, energy is lost with each step; however, the energy lost is less when the gastrocnemius complex is stiff.
- The performance of a ballerina depends much more on esthetics than on the height of a jump. If stretching improves esthetics, the performance is improved, even if the jump height is ≤2 cm. In addition, if a hurdler cannot get the leg over a hurdle without immediately stretching before the race, then stretching will improve performance, even if "running speed" between the hurdles is less.

## ROLE OF STRETCHING IN REHABILITATION FOLLOWING INJURY

- One of the dogmas of sport medicine is that ROM must be restored before strengthening begins. In fact, there is no evidence to support this belief for a vast majority of injuries wherein weakness is a considerable part of the pathology.
- To our knowledge, no studies have compared a rehabilitation program with stretching to a rehabilitation program without stretching after an acute injury. Because stretching affects Type III and IV fibers that transmit pain, stretching should have an analgesic effect just as it does for other conditions. Moreover, if stretch-induced hypertrophy occurs as it does in healthy tissue, stretching should increase the strength of the tissue and improve healing.
- If the purpose of stretching is to increase strength, then it would be more logical to use a strengthening program. Additional studies are needed in this regard for acute injuries. With regard to chronic injuries, two studies that compared a strengthening program to a stretching program without strengthening have suggested that strengthening is much more important (see Recommended Readings):
  - For chronic groin pain (Holmich and colleagues), the strengthening group exhibited 23 excellent results versus 4 excellent results in the static stretching group.
  - For lateral epicondylitis (Svernlov and colleagues), the eccentric strengthening program had a 71% success rate at 1 year compared with only 39% for the proprioceptive neuromuscular facilitation (PNF) stretch group.
- Strengthening exercises to restore tissue strength are the foundation of most rehabilitation programs. Therefore, the most relevant clinical studies would be those that compare stretching plus strengthening with strengthening alone in order to determine if there is a clinically meaningful benefit in adding stretching exercises.

## TECHNIQUES FOR IMPROVING FLEXIBILITY

- Daily stretching improves flexibility; however, the minimum frequency required remains to be determined.
- The optimal way to stretch is unknown and likely depends on each individual person, the muscle (e.g., the angle of the muscle fibers or muscle pennation may influence the optimal duration of a stretch), and the baseline stiffness of a muscle. This hypothesis is supported by studies showing that the optimal duration of a stretch for an individual is different for different muscles and different for different individuals.
- Considering the variability from one individual to another, one proposed method is to individualize the treatment. A person begins by stretching the muscle to an acceptable length and holding the stretch. Once the sensation of force decreases, the stretch is increased so that the sensation felt during initial stages of the stretch is resumed. As long as there is an increased ROM over time, the stretching technique is effective. There are no studies that evaluate the relative effectiveness of this method.
- Some examples of types of stretching are mentioned below:
  - **Static stretching:** One holds a stretch for a given period of time. It is important to feel the sensation of the stretch but not pain.
  - **Active stretching:** One performs a stretch by contracting the antagonist muscle. For example, one may contract quadriceps to stretch the hamstring, with no other passive force being applied to the hamstring. Although this technique gained a lot of popularity several years ago, studies have suggested that it is no more effective at increasing ROM than static stretching.
  - **PNF stretching:** One uses a combination of muscle contraction and passive force to improve the effectiveness of the stretch. Certain forms of PNF contract the muscle being stretched, and certain other forms contract the antagonist muscle. PNF stretching is the most effective type of stretch. The original hypothesis that PNF stretching would decrease muscle activity/tone has been disproved in several studies, and the effect is more likely an increase in stretch tolerance (because there is also an effect on the contralateral limb).
  - **Ballistic stretching:** One moves to the end ROM, then relaxes and returns to the end ROM using a bouncing technique. The amount of force used and how much movement occurs during the relaxation phase (i.e., how close to the neutral position the individual returns to) vary according to various recommendations. Certain authors advocate a full return to the neutral position, whereas other advocate small oscillations at the end ROM. In general, traditional ballistic stretching is not superior to static stretching. The sport medicine community often considers ballistic stretching dangerous. There are theoretical reasons why it might be

dangerous and other theoretical reasons why it would not be more dangerous; however, to our knowledge, there are no published studies clarifying this.

- **Dynamic stretching:** Different authors use this term differently. One common use refers to one moving the joint through a regular ROM without applying force at the end of the motion (i.e., a form of ballistic stretching). Because muscle contractions are involved and because muscle contractions increase the temperature of the muscle, it would not be surprising if this technique were superior to a stretching technique that does not first warm up the muscles. Because there are no studies comparing its effectiveness against other forms of stretching in terms of risk of injury or performance, all claims should be interpreted with great caution.

## PERSONALIZING A STRETCHING PROGRAM

### Objectives

- As with any intervention, one must clearly understand the objectives. Is the objective to reduce injury, improve performance tests, improve performance, or something else? Do not confuse performance tests (e.g., force achieved on a maximal voluntary contraction test) with performance (e.g., vault in gymnastics). Performance requires a much more sophisticated level of muscle coordination, often has an esthetic value, and is greatly influenced by an athlete's psychological state of mind. The effect of a stretching program on performance is a combination of multiple factors.
- Different sports require different ROMs for different joints. Any stretching program should target the specific muscles that require flexibility for that specific sport.
- Most sports require an increase in ROM with increasing velocity of contraction. For example, jogging *generally* requires greater ROM than walking (more specifically, increased knee flexion but decreased knee extension), and running requires greater ROM than jogging.
- Because different muscles have different optimal stretching times, and different individuals have different optimal stretching times, it is important to evaluate the effectiveness of any program shortly after it is initiated. Regardless of the program, a person should improve his/her ROM for the muscles being stretched. If this does not occur within 2 weeks (or sooner), the program should be modified. This may mean changing how long the stretch is held, how many times the stretch is applied in one session, how many sessions are conducted per day or week, and how much force is applied with each stretch. As a starting point, I ask patients to stretch a muscle so that they feel a slight pulling or tension. I ask them to hold this position until they no longer feel the stretch, and then to stretch the muscle a little further so that the sensation of tension is felt again. During the second time, the stretch is held for approximately the same amount of time as the first stretch. In general, most people will end up stretching each muscle for approximately 20–40 seconds using this method; thus, it should be acceptable if patients wish to stretch more than once a day.
- Several stretch positions have been deemed "dangerous" by the sport medicine community:
  - As with any exercise, injury will occur when the stress applied to a tissue exceeds the stress it can withstand.
  - A stretch should not place stress on tissues that are not targeted for increased ROM. For example, the target muscle for the hurdler's stretch is the hamstring muscle when leaning forward and the quadriceps muscle when leaning backward; however, there is also an increased stress to the medial aspect of the knee that appears unnecessary. Therefore, this stretch should be replaced using positions that do not stress the medial aspect of the knee, unless stressing the medial aspect of the knee is a specific objective for that person.

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Available at www.ExpertConsult.com.

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# EXERCISE IN THE HEAT AND HEAT ILLNESS

# 21

Jon Divine • Stephen Dailey, Jr. • Keith Cameron Burley

## HEAT PRODUCTION

### Exercise: The Body's Furnace due to the Inefficiency of Work

- The body utilizes energy in the form of energy-rich chemical compounds to perform work that is required to maintain normal function (Fig. 21.1).
- Conversion from chemical bond energy to gross motor energy is inefficient; *a majority of reactions produce heat.*

### Resting Heat Production

- Sources of nonexercising heat production include involuntary smooth muscle contraction, myocardial contraction, muscle group contraction to maintain posture, shivering, digestion, and baseline cellular metabolism.

### Heat Production During Exercise

- Heat production *increases to 15–20 times of the resting rate* as a result of increased muscular contraction and cellular metabolism.
- At maximum exercise, the work efficiency of the body is only 15%–30%; thus, 70%–85% of muscle energy consumption is converted to heat and *must be dissipated.*
- The thermoregulatory system is unable to maintain a homeostatic environment when heat production is increased beyond the body's ability to effectively transfer heat, resulting in an elevated core temperature.
- The consequence is heat injury.

## HEAT DISSIPATION AND HEAT TRANSFER

### Thermoregulation

- During excess heat production, the body must constantly dissipate heat in order to maintain core temperature.
- Without regulation, the heat generated by the body at rest would increase core temperature by 1°C every 5 minutes; the rate of core temperature elevation accelerates in a hot environment.
- Thermoregulation requires complex interactions among the central nervous system (CNS), cardiovascular system, and integumentary system to maintain a core body temperature of approximately 37°C (98.6°F).
- The CNS temperature regulatory center is located in the hypothalamus.
  - The hypothalamus receives inputs regarding the core body and skin temperature from peripheral skin receptors and circulating blood volume.
  - Increases in core temperature are sensed and centrally regulated by thermal detectors in the hypothalamus, which then provide stimulus via the sympathetic nervous system to initiate sweating and increased peripheral blood flow (Fig. 21.2).
- Core body temperature is determined by the net balance of metabolic heat production and the transfer and exchange of heat to/from the surrounding environment. This can be expressed in the heat balance equation described below (21.1).

$$S = M(\pm \text{work}) - E \pm R \pm C \pm K \qquad \text{(Eq. 21.1)}$$

S = amount of stored heat
M = metabolic heat production
E = evaporative heat loss
R = radiant heat loss or gain
C = convection heat loss or gain
K = conductive heat loss or gain

- Heat loss can be divided into:
  - Nonevaporative heat loss
  - Evaporative heat loss

### Nonevaporative Heat Loss by Conduction, Convection, and Radiation

- Nonevaporative heat loss dissipates most body heat when the ambient temperature is below 20°C (68°F).

#### Conduction Heat Loss

- A warmer body that is in direct contact with a colder body will result in heat transfer to the colder body.
- The rate of conductive heat loss follows Fourier's law: the rate of conductive heat loss is directly dependent on the heat transfer area, the thermal conductivity of materials, and the temperature difference between materials and is indirectly related to material thickness.

#### Convection Heat Loss

- Convection heat loss is heat transfer as a result of forced fluid flow (usually cooler) across a warmer, relatively stationary surface.
- *The rate of convection heat loss follows Newton's law of cooling:* the rate of heat transferred is directly related to the difference between the temperature of an object and that of its surroundings.
- Every object has a **convective heat transfer coefficient (k)**
  - The heat transfer coefficient (k) of water is 50–100 times greater than that of air.
  - Blood has a slightly higher k than water: i.e., a relatively large amount of heat energy is transported with only a moderate increase in the temperature of blood.
- When the difference between the temperature of blood and that of the tissue through which it flows is significant, convective heat transfer will occur, altering both the temperature of the tissue and the blood and follows Pennes' model (1948).
- Convective heat transfer depends on the rate of tissue perfusion of blood and the "local" vascular anatomy.
  - Convective heat loss is directly related to
    - The amount of body surface exposed to circulating air
    - The speed of air circulation
    - At the skin surface level, convective heat loss improves when cutaneous blood flow (transferring heat) increases through dilated peripheral vessels near the skin surface that come in contact with circulating air.
    - The increase in convective heat loss is directly proportional to the speed of circulating air in contact with the skin.
    - Convective heat loss is inversely related to the thickness of the skin between peripheral veins and skin surface.
  - Convective heat loss is the primary principle guiding the use of **cold-water immersion (CWI)** for individuals being treated for hyperthermia.

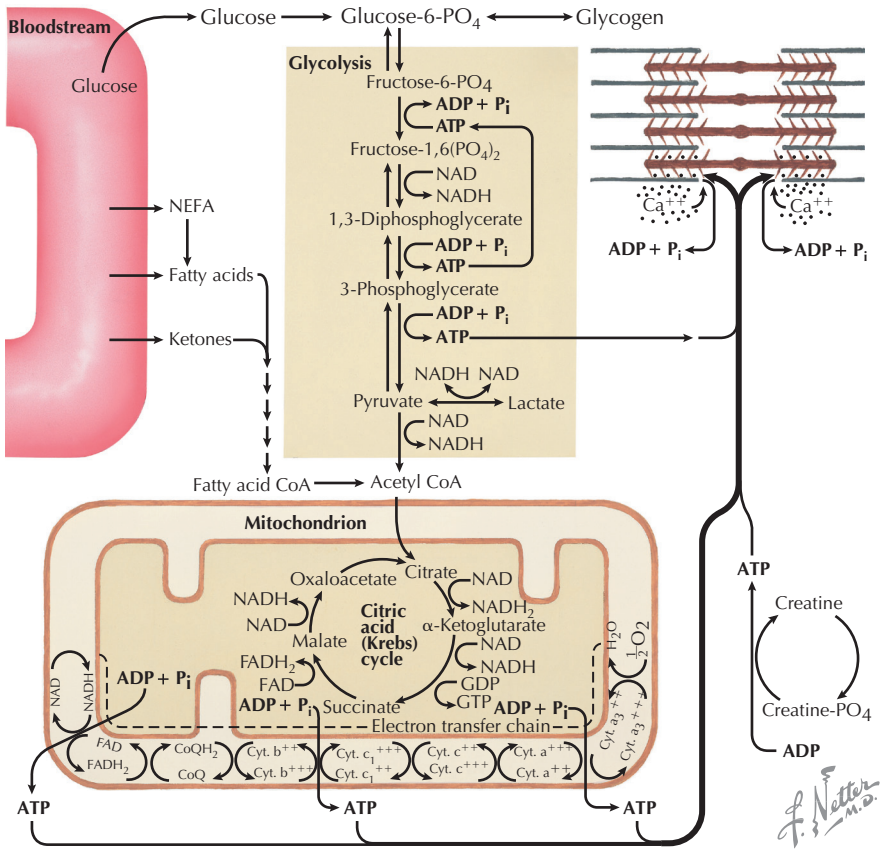


**Figure 21.1.** Regeneration of ATP for source of energy in muscle contraction. The body utilizes energy in the form of energy-rich chemical compounds to perform the work that is required to maintain normal body function.

### Radiation Heat Loss

- Radiation heat loss occurs via the transfer of electromagnetic waves when energy (heat) flows from high temperature to low temperature.

### Evaporative Heat Loss

- Evaporation accounts for a majority of heat loss when the temperature is above 20°C (68°F).
- Evaporation occurs when liquid converts to gas.
- Heat is transferred through the evaporation of sweat and respiratory moisture.
  - Insensate loss of moisture equals 600 mL per day.
- Sweating
  - Sweating usually begins when the body temperature is above 37°C (98.6°F).
  - Amount is proportional to body surface area
    - Rate is dependent on acclimatization and level of conditioning.
    - Sweat rates vary between 600 and 3500 mL per hour.
  - **Heat of vaporization** of water governs the cooling effect of perspiration.
    - Normal rate of heat vaporization of water is 540 calories per gram but from the skin surface is 580 calories per gram.
- Cooling as a result of the evaporation of perspiration is directly related to
  - Sweat rate (L/h): multiplied by 580 calories per gram (heat of vaporization of water on skin)
  - Total body surface area (TBSA)
  - Velocity of air crossing the skin surface area
- Problems with heat dissipation in a hot and humid environment:
  - Evaporative cooling is indirectly related to humidity.
  - Evaporation can account for 98% of heat loss in a hot dry environment.
  - At temperatures above 35°C (95°F), convection and radiation do not contribute to heat loss, whereas sun radiation causes heat gain.
  - In heat-acclimated athletes, equilibrium between heat production and heat dissipation results in core temperatures during exercise ranging between 37°C (98.6°F) and 40°C (104°F) without diminished performance.
  - If heat-dissipating mechanisms fail or if there is overwhelming heat stress, core temperature will continue to rise to dangerous levels.

### Cardiac Output (CO) and Plasma Volume for Heat Dissipation

- Specific demands for cardiac output (CO) and plasma volume must be met for heat dissipation during exercise in a hot environment.
- 15% of CO is shunted to working muscles early during exercise.
- In addition, 15%–25% of CO is shunted from the central circulation to the skin for cooling, which effectively lowers the central plasma volume.
- A bout of exercise decreases plasma volume, venous return, and stroke volume.
  - Conversely, a known training effect of regular exercise is an increase in plasma volume, venous return, and stroke

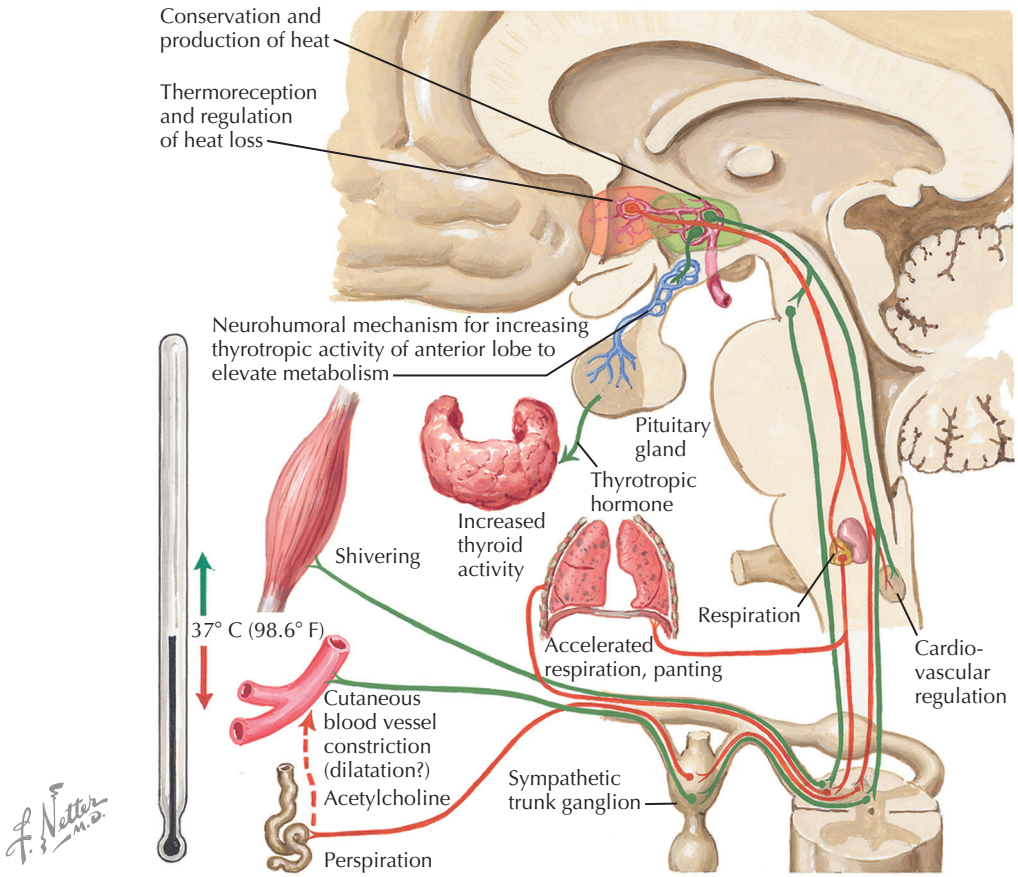


**Figure 21.2.** Temperature regulation. Increases in core temperature are sensed and centrally regulated by thermodetectors in the hypothalamus, which then provide stimulus via the sympathetic nervous system to initiate sweating and increase peripheral blood flow.

volume—all of these result in improved heat regulation with exercise.

- Sweat rate during exercise results in 500–2000 mL loss of plasma volume per hour.
- To maintain the necessary CO in order to maintain exercise intensity (and core temperature), heart rate must increase to compensate for the reduced stroke volume.
- If plasma volume is not maintained while exercise intensity and environmental conditions remain constant, work and exercise performance will be adversely affected, cooling efficiency decreases, and hypotension occurs.
- Process can be favorably changed by reducing exercise intensity (slowing down), building plasma volume (drinking), and improving nonevaporative heat loss mechanisms (e.g., improving convection by increasing skin surface exposure or circulating local wind/water speed and reducing radiation exposure).

## EXERTIONAL HEAT ILLNESS

### Exercise-Associated Muscle Cramps (EAMCs)

**Signs and Symptoms:** Progressive, painful involuntary muscle contraction of skeletal muscles during or after intense, prolonged exercise; large lower limb muscles are commonly affected, but any muscle may be involved, including abdominal and intercostal muscles

**Etiology:** Although commonly referred to as heat cramps, they are not directly related to an elevated core body temperature. Exercise-associated muscle cramps (EAMCs) can occur in warm, cool, or temperature-controlled environmental conditions. EAMCs often begin with early muscle fasciculation in an affected muscle group and may progress to severe and diffuse muscle spasm. Although the exact mechanism is not known, muscle fatigue, dehydration, high sweat rates, high sodium sweat concentration, electrolyte imbalance, and altered neuromuscular control have been implicated in the development of EAMCs.

**Predisposing Factors:** Lack of acclimatization and/or conditioning, ongoing negative sodium balance ("salty sweaters"), history of EAMCs, faster competition performance times, and prior local muscle or tendon injury

**Treatment:**

- Rest and cooling
- Passive, static muscle stretching
- Ice and muscle massage
- Oral hydration with an electrolyte and carbohydrate solution
- Oral intake of salt-containing foods
- In refractory individuals who are nonresponsive to oral hydration or in those who are unable to consume oral fluids, intravenous (IV) normal saline bolus may be indicated.
- Refractory muscle cramps may require IV medications
  - Diazepam 1–5 mg IV
  - Midazolam 1–2 mg IV
  - Magnesium sulfate 2 g intravenous piggyback (IVPB)

**Prevention:** Conditioning and heat acclimatization, maintaining fluid and salt balance; recurrent crampers, particularly "salty sweaters," may benefit from liberal use of salt within their diet, particularly in warmer environments

**Complications:** Rare, but may be a warning sign for impending heat exhaustion; development of rhabdomyolysis should be

expected in severe, prolonged episodes of muscle cramping that involves multiple muscle groups

## Heat Syncope

**Signs and Symptoms:** Syncope, near-syncope, or lightheadedness often seen in unfit or nonheat-acclimatized individuals who stand for extended periods of time in a warm environment

**Etiology:** Often attributed to dehydration, venous pooling in peripheral-dependent extremities, decreased preload, and reduced cardiac output with resultant decreased cerebral blood flow leading to syncope; is generally a self-limited condition

**Treatment:** (General treatment of heat illness)

- Assess ABCs
- Lie down and elevate legs (Trendelenburg position)
- Rest in a cool, shaded environment
- Oral hydration

## Exercise-Associated Collapse (EAC)

**Signs and Symptoms:** Difficulty with standing and/or ambulating without assistance, faintness, dizziness, or syncope following the completion of a bout of prolonged, strenuous activity; accounts for approximately 85% of medical visits following marathons, ultramarathons, or triathlon events

**Etiology:** Normal response to aerobic exercise is an increase in cardiac output in response to the increase in $O_2$ demand and redistribution of blood flow to working muscles. During exercise, the repetitive contraction of skeletal muscles helps to maintain venous return to the heart: often referred to as the "second heart" mechanism. As the intensity of muscle contraction decreases during the end of the event, vasodilatation persists, leading to blood pooling in the peripheral circulation and decreased preload. This combined with inadequate sympathetic tone results in decreased overall cerebral perfusion pressure, which results in an altered mental status.

**Predisposing Factors:** Heat stress, abruptly ending exercise without cool down, dehydration, lack of acclimatization, and advanced age

**Evaluation and Treatment:**

- General treatment of heat illness (3.b.11)
- Rest in a cool, shaded environment with air movement
- Initiate oral hydration
- *Before confirming the diagnosis of EAC, exertional heat stroke (EHS) and exercise-associated hyponatremia (EAH) must be ruled out.*
  - Core temperature <40°C, normal mental status, and normal sodium levels are consistent with EAC.
- With treatment, symptoms should resolve in <30 minutes.
- Follow vital signs closely

**Complications:** Rare, but may be a warning sign for impending heat exhaustion or another more ominous cause of syncope

## Exercise-Associated Hyponatremia (EAH)

**Definition:** Hyponatremia (serum sodium ≤135 mEq/L) observed in athletes that is usually associated with endurance/ultraendurance exercise

**Incidence:** Observed in 5%–13% of marathon participants and 0.3%–27% of ultraendurance participants, most of whom are minimally symptomatic or asymptomatic

**Presenting postevent symptoms:** Complaints of "not feeling right," nausea, lightheadedness, malaise, lethargy, and cramps; vomiting is an extremely common postmarathon symptom of EAH

**Signs of fluid overload:** Edema (e.g., ring or wrist band fitting more tightly), weight gain, and emesis; **more ominous signs of fluid overload are indicative of early pulmonary and cerebral edema and include tachypnea, tachycardia, and mental status changes (confusion, seizure, coma, and death)**

**Etiology:** Thought to be caused by a combination of excessive intake of hypotonic fluids, loss of sodium in sweat, and an inappropriate exercise-elevation of antidiuretic hormone (ADH)

**Predisposing Factors:**

- Endurance activity lasting >4 hours
- Body mass index (BMI) <20 kg/m$^2$
- Weight gain during endurance event: Runners who *fail to lose 0.75 kg of body weight* during a marathon are ***seven times*** more likely to develop hyponatremia than are those who lose >0.75 kg.
- Female gender (likely secondary to lower BMI): Women in the postluteal phase of their menstrual cycle have also been found to be at an increased risk of EAH because of the influence of progesterone on fluid retention and increased sensitivity to ADH during this phase.
- Inexperience with endurance events
- Use of nonsteroidal anti-inflammatory medications (NSAIDs) and selective serotonin reuptake inhibitors (SSRIs): Etiology remains controversial; may result from decreased glomerular filtration rate
- Prolonged activity in a hot and humid environment

**Evaluation/Treatment** (Houston Marathon Protocol):

- Level 1 prerace assessment:
  - Prerace education includes:
    - Stress importance of measuring prerace and postrace weights
    - Prerace questionnaire to self-assess the risk of hyponatremia
    - List of postrace symptoms associated with hyponatremia
    - Encourage salty food and fluid intake in a cool environment
    - Dilute urine production offers favorable prognosis
- Level 2 assessment (serum sodium <135 mEq/L with few or no symptoms):
  - Participant presents with weight gain or postevent symptoms of hyponatremia and no mental status changes.
  - Obtain vital signs, weight, serum electrolytes, and glucose
  - Observe closely while encouraging oral fluid intake
  - IV fluids are typically not needed unless oral fluids cannot be tolerated—consider 3% NS (hypertonic saline) if mental status is deteriorating.
    - Production of dilute urine indicates progress toward normal sodium levels.
- Level 3 assessment (serum sodium <135 mEq/L with markedly mental status changes—seizures, coma, or increasing delirium):
  - Usually not seen unless sodium levels are <128 mEq/L
  - Usually, worsening mental status is followed by rapid deterioration of respiratory status.
  - Establish and monitor airway; be prepared for intubation
  - Establish IV access and use hypertonic saline: **3% NS, 100 mL for over 10 minutes, and follow with 1 mL/min/kg, up to 70 mL/hour, until symptoms improve**
  - Arrange for immediate transport to a medical facility
  - In a critical care setting with central pressure monitoring, loop diuretics and mannitol may be needed to reduce pulmonary and cerebral edema.

**Prevention of EAH:** Educate athletes not to drink more than their sweat rate (usually 600–1200 mL/hour). Athletes can be taught the following method to predetermine their own sweat rate (see **"calculate sweat loss under similar conditions"** in the Prevention of Heat Illness section below). Hypotonic carbohydrate

(6%–8%) fluids may be more beneficial for prevention of EAH than water. Event organizers can reduce the distance between drinking stations for endurance events.

## Heat Exhaustion

**Overview:** ***Heat exhaustion is the most common exertional heat-related illness***. It is characterized by the inability to effectively exercise in the heat secondary to a combination of cardiovascular insufficiency, hypotension, energy depletion, and central fatigue.

**Signs:**
- Elevated core temperature usually ≤40.5°C (≤105°F)
- Decreased cardiac output (tachycardia, orthostatic hypotension, tachypnea, and syncope)
- Mild mental status changes (mild confusion, agitation, and incoordination)
- ***If a majority of mental status changes are severe, proceed to heat stroke evaluation and management.***

**Symptoms:**
- Fatigue/inability to continue exercise
- Headaches
- Nausea and vomiting
- Muscle cramps
- Chills or piloerection
- If dehydration is present as well, thirst may be present, but it is not a reliable indicator of hydration status.

**Etiology:** Failure of the cardiovascular system's response to workload. Usually, a combination of exertional heat stress and dehydration, which results in inability to adequately dissipate heat; occurs most frequently in hot and humid conditions but can occur in normal environmental conditions with intense physical activity; heat exhaustion most commonly affects unacclimatized or dehydrated individuals with a BMI of >27 $kg/m^2$.

**Evaluation and Treatment:**
- General treatment of heat illness (3.b.11)
- Obtain vital signs and core body temperature
- If core body temperature is <40°C (104°F)
  - Remove clothing and sports equipment that restrict heat dissipation
  - Use ice towels and ice bags to assist cooling
  - Initiate oral hydration with an electrolyte-containing solution
  - If unable to tolerate oral fluids, IV fluids may be necessary.
  - Closely monitor vital signs, core temperature, and mental status
  - Initiate rapid cooling protocol if status deteriorates
  - Symptoms usually resolve within 2–3 hours. Slow recovery should prompt transfer to a medical facility for further evaluation.

**Complications:** No long-term sequelae of heat exhaustion have been reported; however, evidence suggests that one episode of marked heat illness may increase the risk of future episodes of heat illness or complications thereafter.

## Exertional Heat Stroke (EHS)

**Definition:** Exertional heat stroke **(**EHS) is a life-threatening condition characterized by extreme hyperthermia (***core temperature >40.5°C or 105°F)*** with eminent thermoregulatory failure, multiorgan failure, and profound CNS dysfunction.

**Signs and Symptoms:**
- Core temperature >40.5°C (105°F); may be as high as 41.7°C–43.3°C (107°F–110°F)
- Markedly impaired cardiac output (hypotension, tachycardia, and tachypnea)
- Pronounced mental status changes (irritability, ataxia, confusion, disorientation, syncope, hysterical or psychotic behavior, seizures, and/or coma)
- Diminished peripheral cooling ability
- Unlike classic heat stroke, which generally affects children and the elderly, patients with EHS will usually have hot and wet skin.
- Life-threatening conditions such as disseminated intravascular coagulation (DIC), acute renal failure (ARF), and neurologic sequelae are associated with prolonged elevated core body temperature.
- Morbidity and mortality increases as the duration of critically elevated core body temperature (>40.5°C or 105°F) is prolonged.
- **Rapid cooling has a marked impact on the survivability of critically ill individuals.**

**Etiology:** Total thermoregulatory failure that will not spontaneously reverse itself; the risk of EHS is greatest when the wet bulb globe temperature (WBGT) is >28°C (82°F) during higher-intensity exercises and when strenuous exercise lasts for >1 hour (Eq. 21.2). **WBGT index is calculated using the following formula:**

$$WBGT = 0.7(WB) + 0.2(BB) + 0.1(DB) \quad \text{(Eq. 21.2)}$$

WB = wet bulb temperature: humidity indicator
BB = black bulb temperature: measures radiant heat
DB = dry bulb temperature: normal air temperature

**Pathophysiology:**
- EHS occurs when core body temperature increases above a critical level, resulting in a cascade of cellular and systemic responses.
- Release of inflammatory cytokines and alteration of heat shock proteins, which decreases the body's ability to prevent thermal denaturation of structural proteins and enzymes, resulting in early circulatory collapse and systemic damage.
- Activation of the coagulation (clotting) pathway as a result of vascular endothelial damage
- At critical levels of hyperthermia, the degree of tissue injury and death is related to both the level and duration of thermal stress, resulting in thermoregulatory failure, heat stroke, and circulatory shock.

**Predisposing Factors:**
- Dehydration (acute and/or chronic)
- Lack of acclimatization to heat
- Negative sodium balance (over time)
- Frequently occurs near the finish line of a race when an already hot, potentially dehydrated athlete increases speed to reach a personal record (PR) performance, causing increased muscle heat production, increased muscle blood flow, secondarily decreased skin blood flow, and increase in core temperature
- Approximately 25% of patients have preexisting gastrointestinal or respiratory illness, and many experience warning signs of impending illness.

**Evaluation and Treatment:**
- General treatment of heat illness
  - Remove clothing and sports equipment that restrict heat dissipation
  - Obtain vital signs and core temperature
    - Determine core temperature by using a rectal thermometer before rapid cooling is initiated
    - Methods of measuring temperature, other than true-core (rectal) temperature, should not guide diagnosis and therapy.
    - Oral, tympanic membrane, aural canal, and axillary temperatures do not correlate with core temperature in heat-injured patients.

- Peripheral temperatures may be up to 1°C lower than the core temperature.

- **Critical Intervention:** CWI up to the neck is the most effective cooling modality for patients with EHS. Once the diagnosis is confirmed, CWI protocol should begin. *NATA 2015 Position Statement: Exertional Heat Illness. Strength of Recommendation Level A.* CWI protocol:
  1. **Initial response:** Once EHS is suspected, prepare to cool the patient and contact emergency medical services (EMS).
  2. **Prepare for ice-water immersion:** On the playing field or in close proximity, half-fill a stock tank or wading pool with water and ice:
     - Ice should cover the water surface at all times.
     - If the athlete collapses near the athletic training room, a whirlpool or cold shower may be used.
  3. **Determine vital signs immediately before immersion:**
     - Core body temperature by using a rectal thermometer
     - Airway, breathing, pulse, and blood pressure
     - Level of CNS dysfunction
  4. **Begin ice-water immersion:** Cover as much of the body as possible with ice water while cooling
     - If full body coverage is not possible owing to dimensions of the container, cover the torso as much as possible.
     - Keep the athlete's head and neck above water.
     - Place an ice/wet towel over the head and neck while the body is being cooled in the tub.
     - Water temperature should be approximately 1.7°C–15°C (35°F–59°F).
  5. **Circulating water:** During cooling, *water should be continuously circulated* to optimize cooling.
  6. **Continued medical assessment:**
     - Vital signs should be monitored at regular intervals.
     - Continuous rectal temperature monitoring is preferred.
  7. **Fluid administration:** An IV fluid line can be placed for hydration and to support cardiovascular function; however, this should not delay ice-water immersion.
  8. **Cooling duration:** Continue cooling until the patient's rectal temperature reduces to 39°C (102°F).
     - If rectal temperature cannot be measured and CWI is indicated, cool for 10–17 minutes and then transport to a medical facility.
     - An approximate estimate of cooling via CWI is 1°C for every 5 minutes and 1°C for every 3 minutes if water is aggressively stirred.
  9. **Patient transfer:** Remove the patient from the immersion tub only after rectal temperature reaches 39°C (102°F) and then transfer to the nearest medical facility via EMS.
     - Cooling should be the primary goal before transport. If EMS or the hospital is not equipped to cool via ice-water immersion, consider *continued cooling on-site* until a safe rectal temperature is achieved.

**Complications:** Extend to all major organ systems:

- CNS injury—may be permanent in up to 20% of cases
- Rhabdomyolysis—results in myoglobinuria, which may lead to acute tubular necrosis (ATN) and renal failure
- Acute renal failure—approximately 25% of EHS patients develop
- DIC—may lead to a circulatory collapse
- Hepatic failure
- Myocardial injury—may lead to arrhythmias, myocardial infarction, and cardiac arrest
- Pulmonary injury—may lead to pulmonary edema, pulmonary infarction, or acute respiratory distress syndrome (ARDS)
- Metabolic/laboratory abnormalities—hypokalemia (early), hyperkalemia (later), hypernatremia, hyponatremia, hypocalcemia, hyperphosphatemia, hypoglycemia, lactic acidosis, uremia, elevated creatine phosphokinase, elevated transaminase, leukocytosis, elevated coagulation studies, myoglobinuria, anemia, hemoconcentration

**Morbidity and Mortality:** Permanent morbidity and mortality rates vary directly with the time elapsed between core temperature elevation and initiation of cooling therapy. Mortality rates can approach 10%.

**Return-to-Play (RTP):** Evidence suggests that a bout of EHS may actually reset thermoregulatory adaptations, which may lead to an increased risk of subsequent heat injury for months after the initial event (Box 21.1). A graduated return-to-play (RTP) protocol should be followed. Although RTP guidelines vary, the primary considerations after EHS are treating associated sequelae and identifying the cause of EHS to aid prevention techniques. Exercise heat tolerance testing has been helpful in determining the appropriate time for an athlete to RTP.

**BOX 21.1 A GRADUATED RETURN-TO-PLAY PROTOCOL FOLLOWING HEAT ILLNESS**

**REST:** Before returning to activity, an athlete should rest for 7–21 days, allowing for recuperation and all blood work to return to normal.
**LOW AND COOL PROGRESSION:** Once an athlete has returned to baseline, he/she may begin with low-intensity exercise in a cool environment and gradually increase the intensity, duration, and level of heat exposure over a 2-week period.
**CONSIDER TESTING**: If return to pre-EHS activity levels seems challenging, consider laboratory-based exercise-heat tolerance test at 1 month after an EHS event.
**COMPETITION:** An athlete may be cleared for full competition if heat tolerance exists after 2–4 weeks of training.

## RISK FACTORS AND POPULATIONS AT INCREASED RISK OF HEAT-RELATED ILLNESS

- Healthy adults
  - Poorly acclimated to heat or humidity
  - Poorly conditioned
  - Inexperienced in activity (limited judgment about heat risk)
  - Salt or water depleted
- Large or obese adults
  - Generate more heat for the same level of activity; adipose tissue has lower specific heat than does lean tissue
  - Dissipate heat less efficiently
    - Lower body surface-to-mass ratio
    - Fewer heat-activated sweat glands
    - Poorer fitness levels
- Children
  - Produce more metabolic heat per mass unit compared with adults
  - Sweat less and require greater core temperature increases to trigger sweating
  - Acclimatize slower
  - Lower cardiac output at any given workload
  - May lack adequate blood flow for both muscle and cooling needs
  - Transition to adult thermoregulation begins after puberty

- Elderly individuals
  - Generally less efficient at cooling
    - Reduced vasodilator response, which may begin as early as 50 years of age
    - Lowered maximum heart rate leads to decreased maximum cardiac output
    - Reduced thirst response after water deprivation, which results in underhydration
    - Often have reduced fitness levels
- History of previous heat injury
  - Unknown mechanism: speculation that the CNS-controlled cooling mechanism can be irreversibly damaged, resulting in a higher threshold requirement to begin diaphoresis
- Women of reproductive age during the postovulatory phase of the menstrual cycle:
  - Heat dissipation may be reduced because of an increased temperature threshold to begin sweating or possibly a smaller plasma volume.
- Acute illnesses:
  - Increased metabolic demand for blood flow throughout the body; specifically, gastrointestinal illness:
    - Increased risk because increased splanchnic circulation competes with skin blood flow for cardiac output
    - Often associated with dehydration and electrolyte disturbances
- Chronic illnesses associated with heat illness:
  - Cystic fibrosis, diabetes mellitus (uncontrolled), and history of malignant hyperthermia
- Presence of sickle-cell trait (SCT): See Chapter 26: Hematologic Problems in Athletes
- Alcohol and other substance abuse:
  - Alcohol has residual effects on thermoregulation capacity, predisposing athletes to dehydration and reduced cooling efficiency.
  - Acute stimulant intoxication (cocaine or methamphetamine) may be difficult to differentiate from heat stroke because of increased metabolic activity.
- Use of certain medications:
  - Anticholinergic agents, antihistamines, beta-blockers, diuretics, tricyclic antidepressants and monoamine oxidase inhibitors, and stimulant agents for attention-deficit disorder predispose people to a higher risk of heat-related illness.

## PREVENTION OF HEAT-RELATED ILLNESS

### Adequate Preparticipation Medical History and Evaluation

Identify athletes who are at a risk of sustaining heat-related illness.

### Ensure Adequate Acclimation and Conditioning

- Physiologic effects of acclimation to exercise in heat require 7–10 days of exposure and include:
  - Improved cooling efficiency from sweating: earlier initiation of sweating, increased rate of sweating, increased maximum sweating capacity, and lower sweat sodium concentration
  - Increased cardiovascular efficiency: increased basal plasma volume, decreased heart rate at a given workload, and heat stress
  - Thermal effects: increased exercise capacity in heat, lower core and skin temperature at a given workload and heat stress, reduced perceived intensity of exercise, and increased subjective thermal comfort
  - Approximately 75% of effects occur in the initial 4–6 days.
  - Continuous daily aerobic exercise of 90–100 minutes may be optimal.
  - Acclimation effect persists for 1–4 weeks.
  - Individual variation is remarkable, and effects may persist longer in dry heat than in humid heat.

### General Points for Improving Heat Acclimation

- Preseason conditioning should include strength, endurance, and skills acquisition drills in a warm environment.
- Delay full participation until minimum conditioning levels are met.
- Heat acclimation generally requires 10–14 days.
- Avoid supramaximal efforts, such as performance testing, in heat—particularly those involving athletes with sickle cell trait.
- The historical practice of fluid restriction to improve heat adaptability or conditioning is extremely dangerous and should never be done.
- Provide education regarding heat-related illness to all athletes. This should include:
  - Providing awareness and counseling to at-risk populations and their coaches regarding signs and symptoms of early heat stress, dangers and sequelae of heat-related illness, and strategies to minimize the risk of developing these illnesses
  - The importance of staying appropriately hydrated, adequate sleep, avoiding drugs and alcohol, knowing each athlete's individual limitations, and avoiding exercise in dangerous environmental conditions should be emphasized to all athletes and coaches.

### Monitor Atmospheric and Environmental Conditions

- The American College of Sports Medicine (ACSM) and several high school athletic federations have guidelines on when to restrict activity during periods of high temperatures.
- **Monitor weather reports**
  - Simple and convenient sources of approximate temperature and humidity readings such as frequently updated Internet sites or mobile phone applications are helpful to calculate heat stress (Fig. 21.3) but may not allow for local variations of sun exposure, wind velocity, or local airflow.
  - The ACSM endorses the use of color-coded warning flags along the course of an endurance event, which is used in numerous endurance races to alert runners/participants regarding relative risks of continuing a given race based on relative heat stress (Fig. 21.4).

### Adjust Workout Schedule Based on Environmental Conditions

- Reschedule workouts, practices, and competitions to a cooler time of day or cancel altogether.
- Alter practices and workouts by (Table 21.1):
  - Decreasing their intensity
  - Shortening their duration
  - Providing more frequent breaks
  - Modifying practice clothing to increase evaporative cooling
  - Moving to a more shaded or breezy area

## APPROPRIATE CLOTHING FOR EXERCISE IN THE HEAT

- Short-sleeved, loose-fitting, open-weave, or mesh jerseys allow better evaporation.
  - Evidence to support or discourage wearing of newer, sweat-"wicking" shirts (made of fabric that absorbs sweat away from the skin to the outer surface to promote evaporative cooling) to reduce the risk of heat illness is lacking.

- Wearing no shirt has both benefits (better heat loss from evaporation and convection) and risks (more radiant heat gain).
- Athletes should practice or play in shorts when possible.
- Light-colored uniforms reflect sunlight and thus reduce radiation absorption.
- As they become sweat-soaked, uniforms or clothing should be changed to allow efficient evaporation.
- Avoid restrictive clothing and bulky protective equipment such as poorly aerated helmets, heavy long-sleeved uniforms, protective pads, and rubberized workout suits, which block skin surface area and reduce cooling by radiation, convection, and evaporation.
- Keep in mind the protective benefits of oil- or gel-based sunscreens as these may block evaporative cooling.
  - Using a water-based sunscreen, applied frequently, is the ideal means to provide adequate sun protection without adversely affecting heat dissipation.

Air temperatures (°F)

| Relative humidity (%) | 70 | 75 | 80 | 85 | 90 | 95 | 100 | 105 | 110 | 115 | 120 |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 0% | 64 | 69 | 73 | 78 | 83 | 87 | 91 | 95 | 99 | 103 | 107 |
| 10% | 65 | 70 | 75 | 80 | 85 | 90 | 95 | 100 | 105 | 111 | 116 |
| 20% | 66 | 72 | 77 | 82 | 87 | 93 | 99 | 105 | 112 | 120 | 130 |
| 30% | 67 | 73 | 78 | 84 | 90 | 96 | 104 | 113 | 123 | 135 | 148 |
| 40% | 68 | 74 | 79 | 86 | 93 | 101 | 110 | 123 | 137 | 151 | |
| 50% | 69 | 75 | 81 | 88 | 96 | 107 | 120 | 135 | 150 | | |
| 60% | 70 | 76 | 82 | 90 | 100 | 114 | 132 | 149 | | | |
| 70% | 70 | 77 | 85 | 93 | 106 | 124 | 144 | | | | |
| 80% | 71 | 78 | 86 | 97 | 113 | 136 | | | | | |
| 90% | 71 | 79 | 88 | 102 | 122 | | | | | | |
| 100% | 72 | 80 | 91 | 108 | | | | | | | |

**Figure 21.3.** Heat stress (apparent temperatures in °F): calculation of heat stress and heat stress risk. (Modified from National Weather Service: Heat Wave and U.S. Department of Commerce, National Oceanic and Atmospheric Administration, PA 85001, 1985. Color code adapted from ACSM Position Statement [1975] on prevention of heat injuries during distance running.)

## TABLE 21.1 ALTERATION OF PRACTICES AND WORKOUTS BASED ON HIGH HEAT AND HUMIDITY

| *WBGT* | | |
|---|---|---|
| °F | °C | Restraints on Activities |
| <75.0 | <24.0 | All activities allowed, but decrease intensity and be alert for symptoms of heat-related illness |
| 75.0–78.6 | 24.0–25.9 | Shorten practice duration and provide more frequent rest periods in the shade<br>Enforce drinking of fluids every 15 minutes<br>Maximize sweat evaporation with appropriate clothing choices and maximize wind exposure |
| 79.0–84.0 | 26.0–29.0 | Stop activity for unacclimatized and high-risk athletes and shorten activity duration for all others<br>Consider cancellation of long-distance races |
| >85.0 | >29.0 | Cancel all athletic activities |

*WBGT*, Wet-bulb globe temperature.

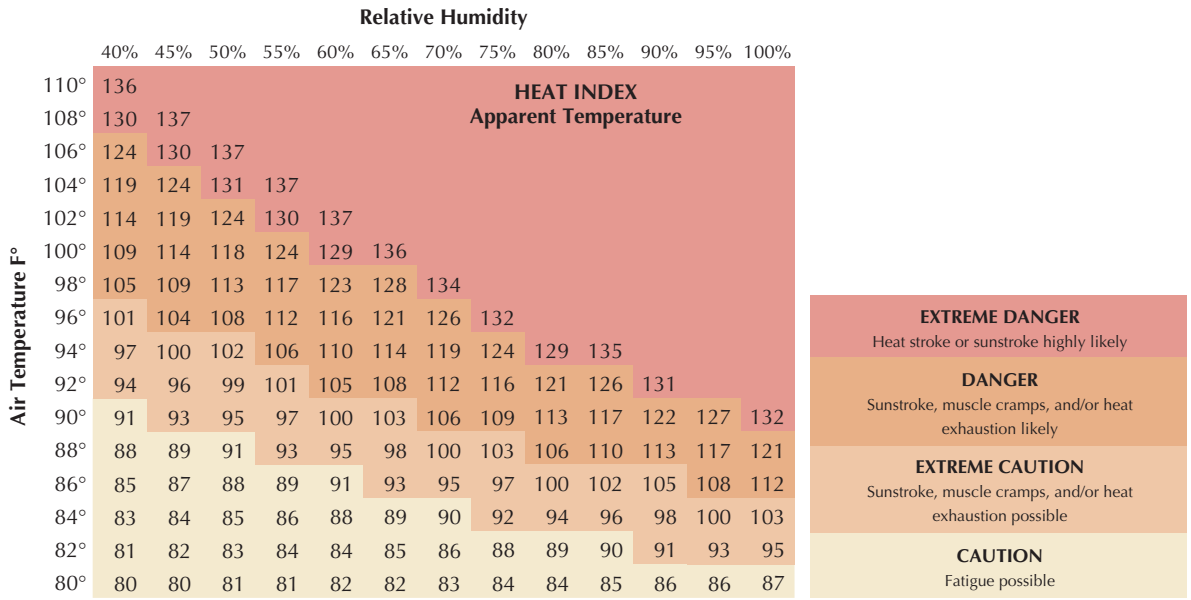


**Figure 21.4.** Color-coded warning flags used during an endurance event to alert runners/participants regarding relative risks of continuing the event based on relative heat stress. (From National Oceanic and Atmospheric Administration, National Weather Service [http://www.nws.noaa.gov/om/heat/heat_index.shtml].)

## Close Monitoring of Athletes

- Observe athletes who are at an increased risk for heat-related illness

## Prevent Athletes With Fevers and Acute Illnesses From Participating

- Core temperature elevations caused by fever and illness are additive to those caused by activity; therefore, both cardiac output and aerobic capacity are reduced in febrile athletes. *Exercise with fever may be dangerous, particularly in heat.*
- Aspirin, NSAIDs, acetaminophen, or other antipyretics cannot reduce core temperature elevations caused by exercise.
- When in doubt regarding participation, apply the "neck check" rule: *no participation if symptoms (cough, abdominal pain, nausea, vomiting, and/or diarrhea) are present below the neck or if fever is present.*

## Prevent Dehydration

- Dehydration reduces endurances exercise performance, decreases time to exhaustion, and increases heat storage.
- When fluid deficits exceed
  - By 2% of body weight, work capacity decreases by 15%–20%, and thermoregulatory function is impaired
  - By >3%–5% of body weight, sweat production and blood flow to the skin begin to decrease
  - By >6%–10% of body weight, cardiac output decreases considerably, resulting in lower sweat production and reduced blood flow to both skin and working muscles
- Dehydration *plus* heat stress of exercise cause:
  - Greater reduction in central volume, venous return, and consequently stroke volume and cardiac output, compared with *either factor alone*.
  - Performance reductions proportional to the level of dehydration and intensity of exertional heat stress
  - Reduced heat dissipation resulting from decreased skin flow and diminished sweating
- Monitoring body weight helps assess dehydration.
  - Acute weight loss implies dehydration.
  - *Caution is indicated when an athlete has a workout weight loss of >3% of body weight or fails to regain the previous day's weight loss by the time of the next day's workout.* During summer workouts, dehydration can be cumulative over several days, such as during two-a-day football workouts.
  - Athletes with large or persistent acute weight loss should be restricted from activity until rehydrated.
- For prolonged or repetitive endurance exercise in the heat, carbohydrate–electrolyte solutions (6%–8% carbohydrates) may limit dehydration, accelerate rehydration, and have been reported to maintain performance.
- **Thirst is not an adequate guide for fluid consumption in humans:** Calculation of individual fluid replacement rates should be encouraged for all athletes.
  - Athletes *may not feel thirsty* until they have depleted >5% of the volume in the euvolemic state.
  - Inexperienced athletes may automatically associate heat illness symptoms with being dehydrated and drink excessively, resulting in overhydration, which increases the risk of hyponatremia.
  - For optimal individual hydration, individuals should drink at regular intervals (every 15–20 minutes) and replace what they sweat. Fluid loss from sweat usually occurs at a rate of 600–1200 mL per hour but can be higher or lower in certain athletes and will vary based on weather conditions, exercise intensity, body surface area, and genetics.
- Calculating sweat loss under similar conditions is the ideal method to determine fluid replacement.
- The most accurate method to easily determine fluid requirement is to calculate sweat loss under similar conditions:
  - Weigh nude before an activity
  - Perform the activity at a competition level for 1 hour (recommended to achieve a reliable representation of expected sweat rate in an endurance event)
  - Track fluid intake (in mL) during the activity
  - Record nude weight after the activity, and subtract it from the starting weight. Convert the difference in body weight to mL
  - To determine hourly sweat rate, add the difference in body weight (in mL) to the volume of fluid consumed.
  - To determine how much to drink every 15 minutes, divide the hourly sweat rate by 4. This becomes the guideline for fluid intake every 15 minutes of the activity
  - Note the environmental conditions on this day and repeat measurements on another day when the environmental conditions are different. This will give the athlete an idea of how different conditions affect his/her sweat rate.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 22 EXERCISE IN THE COLD AND COLD INJURIES

Christopher C. Madden • Jessie R. Fudge

## GENERAL PRINCIPLES

### Physiology of Cold Exposure

#### Mechanisms of Heat Loss

RADIATION

- Radiation involves the direct emission or absorption of heat energy from the body (mostly infrared radiation).
- Radiation is the largest source of heat loss from the body.
- Clothed, sedentary individuals in a calm, temperate climate lose more body heat (approximately 60%) by radiation than active individuals (approximately 45%) in a thermoneutral environment where heat production equals heat loss.
- The human body constantly radiates heat to nearby solid objects with a cooler temperature, and the rate of heat loss increases with the difference in temperature between the body and the object.
- Radiant heat loss increases as temperature decreases but only becomes a significant concern in extremely cold environments.
- Clothing does not markedly affect radiant heat loss.

EVAPORATION

- Evaporation occurs when water or moisture is transformed into vapor.
- Evaporation occurs with perspiration on the body's surface and in respiratory passages as inspired air is warmed and moistened.
- High altitude increases heat and water losses from the lungs because breathing deepens and the respiratory rate increases.
- A small amount of "insensible" perspiration occurs at cold temperatures but may become significant in active individuals who are not appropriately dressed for that temperature.
- Vigorous exercise with increased sweating leads to greater evaporative heat loss than with normal evaporative loss in sedentary individuals in a temperate climate (a difference of approximately 20%–30%).
- Vapor barrier systems meant to decrease evaporative water loss are primarily ineffective in physically active individuals because they trap perspiration between the barrier and the skin and lead to increased heat loss by convection when the sweat rate exceeds the rate of permeability (Fig. 22.1).
- Wet clothing combined with wind leads to significant evaporative heat losses.

CONVECTION

- Convection involves the transfer of heat from the body to cold air or water in contact with the body surface.
- Ongoing heat loss occurs when warmed air or water is continually displaced from the body surface and is replaced by air and water of colder temperatures.
- The amount of heat loss that occurs by convection is determined by the temperature difference between the air and the body surface and by the speed of the air moving over the body.
- Convective heat loss increases greatly with **wind** moving over the body surface, particularly with submersion in cold water.
- In addition to **natural wind, wind moving over the body surface** created by cycling, skiing, running, windsurfing, or other activities that increase air flow over the body can cause marked heat loss, particularly at cold temperatures sustained over prolonged periods.
- **"Wind chill"** is wind combined with cold that causes a lower "equivalent" temperature (Fig. 22.2) than current environmental temperature and greatly accelerates heat loss by convection; wind chill is most significant with the first 20 mph increase in wind speed, with some additional effects at higher wind speeds.
- Cold-water swimmers lose significant heat by convection because continuous motion prompts the ongoing displacement of water away from the body, resulting in constant exposure of a "warm" body to cold water.
- Windproof clothing, particularly combined with appropriate insulation layers, can greatly reduce convective heat loss in most environments by trapping warm air and minimizing air displacement.

CONDUCTION

- Conduction involves the direct transfer of heat from the body when it is in contact with a surface colder than the body's temperature (e.g., water, snow, ice, or rocks).
- Water is an excellent conductor, and it has an exponentially greater volumetric heat capacity than air, which can lead to significant heat losses for anyone immersed in cold water, particularly without protective clothing.
- In addition, wet clothing increases conductive heat losses.

#### Mechanisms of Heat Production

- Metabolic and biochemical reactions (slight increase)
- Muscular activity
  - Involuntary shivering (increases heat production 2–6 times the basal levels; subsides after several hours when core temperature drops below 30°C [86°F])
  - Voluntary exercise (increases heat production up to 10 times the basal levels)
- Nonshivering thermogenesis
  - Increases thyroxine, epinephrine, and norepinephrine and a subsequent slight increase in overall tissue metabolism
  - Relatively ineffective in preventing cold injury; may increase heat production by 10%–15% in adults

#### Thermoregulation and Physiologic Adaptations

- The hypothalamus is the thermoregulatory center for maintenance of body temperature and physiologic response to cold.
- The body needs to stay between 34°C and 40.5°C (95°F and 105°F) to maintain normal organ function; core temperature normally ranges between 36.5°C and 37.5°C (97.7°F–99.5°F).
- The body dissipates heat relatively efficiently but is less effective at compensating for cold.
- The body initially responds to cold by regulating the **core** (brain and other major organs) and **shell** (skin, muscle, and extremities).
  - Exposure to cold stimulates thermoreceptors in the skin that signal the hypothalamus to activate shivering and nonshivering thermogenesis, constrict peripheral or "shell" circulation, and decrease sweating in an effort to increase heat production and maintain core temperature.

- The body shell acts as an interface between the body and environmental stresses—shell temperature may vary widely in contrast to core temperature.
- Athletic performance may be decreased with shell cooling as cold weakens and slows muscle contractions, decreases tissue elasticity, and slows nerve conduction.

- Prolonged cooling causes body core temperature to drop and results in several physiologic changes:
  - When core temperature falls below 35°C (95°F), rates of essential biochemical reactions decelerate.
  - Shivering gradually ceases at approximately 31°C–32°C (88°F–90°F) as muscles become cooler and stiffer.
  - Severe cold may affect all organ systems, particularly the central nervous system (CNS) and the cardiovascular system (Table 22.1).

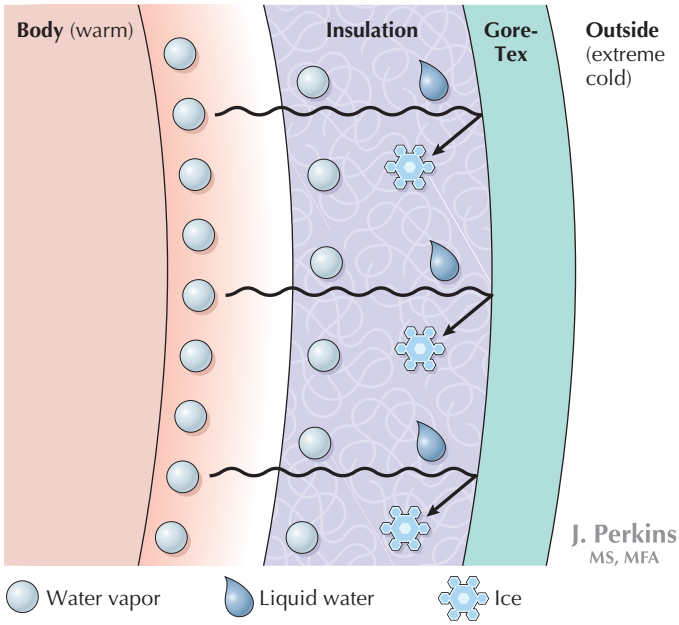


**Figure 22.1.** Vapor barrier systems.

- As temperature decreases, heart rate and cardiac output reduce, and arrhythmias may occur secondary to myocardial irritability.
- Cerebral blood flow decreases, leading to dilated pupils, obtundation, stupor, and eventually coma.
- Respiratory rate reduces and so does oxygen consumption.
- Diuresis occurs secondary to shunting of blood volume to the body core from the periphery.
- Lactic acidosis develops secondary to decreased shell tissue perfusion (decreased tissue oxygenation) and subsequent anaerobic glycolysis combined with carbon dioxide retention that occurs with reduced respiratory rate.
- Insulin activity reduces and hyperglycemia develops.

## Exercise in the Cold

### Sports Associated With Cold Injury

- Any cold-weather or cold-water sport can be associated with cold injury.
- Risk of cold injury corresponds with level of cold exposure, wind, precipitation, and athlete's preparation for cold exposure.
- Specific sports may include skiing (all types), snowshoeing, mountaineering and rock climbing, snowmobiling, canoeing, kayaking, white-water rafting, open-water swimming, and skating. Windsurfing and small-boat sailing may result in cold injury after unexpected capsizing. Moreover, unprepared runners, hikers, backpackers, and cyclists may experience significant cold injuries, particularly with prolonged and inadequate exposure combined with unexpected weather (e.g., blizzard, cold rain, high winds, dropping temperature, and wind chill).

### Prevention

Almost all cold illness is caused by exposure to cold, often unexpected, without adequate protection. Appropriate planning is imperative to prevent cold illness and injury.

#### INCREASE HEAT PRODUCTION

- Increasing heat production is the least effective way to prevent cold illness.
- Exercise: voluntary muscular activity will produce heat

| Wind (mph) \ Temperature (°F) | Calm | 40 | 35 | 30 | 25 | 20 | 15 | 10 | 5 | 0 | –5 | –10 | –15 | –20 | –25 | –30 | –35 | –40 | –45 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 5 | | 36 | 31 | 25 | 19 | 13 | 7 | 1 | –5 | –11 | –16 | –22 | –28 | –34 | –40 | –46 | –52 | –57 | –63 |
| 10 | | 34 | 27 | 21 | 15 | 9 | 3 | –4 | –10 | –16 | –22 | –28 | –35 | –41 | –47 | –53 | –59 | –66 | –72 |
| 15 | | 32 | 25 | 19 | 13 | 6 | 0 | –7 | –13 | –19 | –26 | –32 | –39 | –45 | –51 | –58 | –64 | –71 | –77 |
| 20 | | 30 | 24 | 17 | 11 | 4 | –2 | –9 | –15 | –22 | –29 | –35 | –42 | –48 | –55 | –61 | –68 | –74 | –81 |
| 25 | | 29 | 23 | 16 | 9 | 3 | –4 | –11 | –17 | –24 | –31 | –37 | –44 | –51 | –58 | –64 | –71 | –78 | –84 |
| 30 | | 28 | 22 | 15 | 8 | 1 | –5 | –12 | –19 | –26 | –33 | –39 | –46 | –53 | –60 | –67 | –73 | –80 | –87 |
| 35 | | 28 | 21 | 14 | 7 | 0 | –7 | –14 | –21 | –27 | –34 | –41 | –48 | –55 | –62 | –69 | –76 | –82 | –89 |
| 40 | | 27 | 20 | 13 | 6 | –1 | –8 | –15 | –22 | –29 | –36 | –43 | –50 | –57 | –64 | –71 | –78 | –84 | –91 |
| 45 | | 26 | 19 | 12 | 5 | –2 | –9 | –16 | –23 | –30 | –37 | –44 | –51 | –58 | –65 | –72 | –79 | –86 | –93 |
| 50 | | 26 | 19 | 12 | 4 | –3 | –10 | –17 | –24 | –31 | –38 | –45 | –52 | –60 | –67 | –74 | –81 | –88 | –95 |
| 55 | | 25 | 18 | 11 | 4 | –3 | –11 | –18 | –25 | –32 | –39 | –46 | –54 | –61 | –68 | –75 | –82 | –89 | –97 |
| 60 | | 25 | 17 | 10 | 3 | –4 | –11 | –19 | –26 | –33 | –40 | –48 | –55 | –62 | –69 | –76 | –84 | –91 | –98 |

Frostbite Times: 30 minutes; 10 minutes; 5 minutes

Wind Chill (°F) = $35.74 + 0.6215T - 35.75(V^{0.16}) + 0.4275T(V^{0.16})$

Where, T= Air Temperature (°F) V= Wind Speed (mph)

*Effective 11/01/01*

**Figure 22.2.** Wind chill chart. (From National Oceanic and Atmospheric Administration, National Weather Service [http://www.nws.noaa.gov/om/winter/windchill.shtml].)

**TABLE 22.1 CLINICAL MANIFESTATIONS OF HYPOTHERMIA**

| System | Mild Hypothermia (35°C–32°C [95°F–90°F]) | Moderate Hypothermia (32.2°C–28°C [90°F–82.4°F]) | Severe Hypothermia (<28°C [82.4°F]) |
|---|---|---|---|
| Central nervous system | Confusion, slurred speech, impaired judgment, amnesia | Lethargy, hallucinations, loss of papillary reflex, EEG abnormalities | Loss of cerebrovascular regulation, decline in EEG activity, coma, loss of ocular reflex |
| Cardiovascular system | Tachycardia, increased cardiac output, and systemic vascular resistance | Progressive bradycardia (unresponsiveness), decreased cardiac output, BP, atrial and ventricular arrhythmias, J (Osborn) wave on EKG | Decline in BP and cardiac output, ventricular fibrillation (28°C [82.4°F]) and asystole (20°C [68°F]) |
| Respiratory system | Tachypnea, bronchorrhea | Hypoventilation (decreased RR and tidal volume), decreased oxygen consumption and $CO_2$ production, loss of cough reflex | Pulmonary edema, apnea |
| Renal | Cold diuresis | Cold diuresis | Decreased renal perfusion and GFR, oliguria |
| Hematologic | Increase in hematocrit, decreased platelet and white blood cell counts, coagulopathy, and DIC | | |
| Gastrointestinal | | Ileus, pancreatitis, gastric stress ulcers, hepatic dysfunction | |
| Metabolic/endocrine | Increased metabolic rate, hyperglycemia | Decreased metabolic rate, hyper- or hypoglycemia | |
| Musculoskeletal | Increased shivering | Decreased shivering (32°C [89.6°F]) | Patient appears dead "pseudorigor mortis" |

From Mellion M. *Sports Medicine Secrets*. 3rd ed. Philadelphia: Hanley & Belfus; 2002.
*BP,* Blood pressure; *DIC,* disseminated intravascular coagulation; *EEG,* electroencephalogram; *GFR,* glomerular filtration rate; *RR,* respiratory rate.

- Shivering: involuntary muscular activity will also produce heat
- Eating: frequent meals or snacks are needed to replenish glycogen and fat stores, particularly with prolonged exercise in cold environments
- Hydration: consume adequate fluids to prevent dehydration and subsequent impairment in circulating blood volume

DECREASE HEAT LOSS

- Decreasing heat loss is the most effective way to prevent cold illness.
- Physiologic mechanisms for increasing heat production are much less effective than strategies applied to decrease heat loss.
- Heat loss is primarily prevented with adequate **layering** of clothing.
- Insulation and permeability are important properties of cold weather clothing.
- Most versatile cold weather clothing systems are usually composed of three layers:
  - Inner hydrophobic polyester fabric (e.g., Capilene, Coolmax, Thermax, Thermolite, or Thermostat) that allows wicking of moisture away from the body. Avoid cotton.
  - Middle insulating material can be a second light layer (similar to inner layer fabric) or a heavier layer (can cause overheating; best used during exercise warm-up, cool-down, or in extremely cold environments) such as wool and wool/synthetic blends (heavy when wet but excellent insulator even then), pile and fleece (varying weights offer versatility for exercising athletes, fleece replacing pile), synthetic fillers (e.g., Primaloft, Liteloft, Dacron, Hollofill, Quallofil, or Thinsulate), or down (good in cold dry conditions, loses insulating properties when wet); the middle layer can lead to overheating.
  - Outer protective shell that is windproof and water repellant (e.g., newer treated nylons):
    - Newer treated nylons are best because they offer highest breathability: fibers are tightly woven, and sprays are used to increase water repellency.
    - Laminates such as Gore-Tex and other brand-specific materials designed to mimic Gore-Tex are advertised as "waterproof" and breathable, but the amount of sweat produced during exercise can exceed the capability of the laminate to transmit water vapor.
    - To date, an ideal fabric that allows water vapor to pass outward but not inward has not been developed.
    - Breathability of the outer layer is inversely proportional to the degree of water repellency and waterproofing; adequate breathability is important during exercise.
- Middle and outer layers should each be slightly larger than the direct inner layer to allow a narrow space for warm air trapping; avoid "tight" layering.
- The primary goal with layering is to stay relatively warm without excessively sweating or overheating, and layers must be shed or added depending on current activity levels and environmental conditions.
- Ventilation ports in certain outer garments may be opened for adequate breathing and are particularly important when using laminates.
- Excessive sweating causes heat loss through increased evaporation and conduction and may affect the insulating property of certain fabrics.
- Wind chill can cause dangerously large amounts of heat loss through convection without windproof garments and appropriate insulation adjusted to activity levels.

SPECIAL PROTECTION

- Special protection and other measures to minimize heat loss and injury should be implemented.

- Prevent significant conductive loss by placing a "barrier" between the body and colder objects (e.g., foam or other sleeping pads such as Therma-Rest, wet or dry suits with cold-water exposure, or leather or plastic boots with an inner insulating layer).
- Maintain adequate trunk warmth to minimize vasoconstriction in hands and feet that may result in subsequent cold injury.
- Wear mittens instead of gloves, preferably with a protective and windproof outer shell and an inner insulating layer.
- Prevent radiant heat loss from the head (blood supply to the head is maintained in cold conditions) by wearing an insulating cap or balaclava and by using a hood when needed (high wind or wet weather).
- Protect the exposed skin of face and ears by using a balaclava or neck gaiter pulled up over the face (neck gaiters also decrease heat loss from relatively superficial, large vessels of the neck).
- Prevent genital injury by wearing undershorts with a windproof front panel.
- Protect eyes (corneal freezing) by using ski goggles.
- Avoid excessive wetting at all times, and if immersed in cold water, minimize movement and pull body into a "tight" position (assume the HELP posture—**h**eat **e**scape **l**essening **p**osture—which is similar to an "upright fetal" position) to decrease exposed surface area unless close to shore or a boat.
- Adequately warm-up before exercising in cold conditions (may warm-up indoors or use insulated protective clothing if outdoors).
- Avoid the use of emollients on the face because these have been shown to increase the incidence of frostbite and other cold injuries.

#### EXTERNAL WARMING SOURCES

See Treatment discussion in the Accidental Hypothermia section.

## SPECIFIC INJURIES

### Systemic Cold Injury

#### Accidental Hypothermia

**Definition:** Unintentional decline in core body temperature below 35°C (95°F) not due to organic disease

- **Mild** (35°C–32°C [95°F–90°F])
- **Moderate** (<32°C–28°C [90°F–82°F])
- **Severe** (<28°C [82°F])
- Certain authorities define these values more conservatively. The advanced cardiac life support (ACLS) algorithm for the treatment of hypothermia classifies severe hypothermia as below 30°C (86°F).

**Risk factors:** Old age, infancy, alcohol, CNS depressants, hypothyroidism, hypopituitarism, hypoadrenalism, diabetes mellitus, sepsis, CNS insult (e.g., cerebral vascular accident or trauma), malnutrition, and hypoglycemia

**Presentation:** Assume hypothermia until proven otherwise when evaluating an athlete with confusion, dysarthria, and ataxia who has been exposed to cold (see Table 22.1). Rectal temperature (estimation of core temperature) and clinical findings establish definitive diagnosis, but low-reading rectal thermometers are frequently not available. **Low-reading thermometers should be available in first-aid kits during cold-weather athletic events.** The degree of hypothermia can be estimated using clinical observation: quickly note the **level of consciousness** and **presence of shivering.** A conscious individual who is shivering likely has a core temperature above 31°C–32°C (88°F–90°F), whereas a severely confused or obtunded individual who is not shivering likely has a core temperature below 31°C–32°C (88°F–90°F).

- **Mild hypothermia:** Confusion, poor judgment, dysarthria, amnesia, dizziness, apathy and fatigue, mood lability, ataxia, increased shivering, tachycardia, tachypnea, elevated blood pressure, increased urinary frequency (cold diuresis), hyperreflexia, and hyperglycemia
- **Moderate hypothermia:** Lethargy, stupor, occasional unconsciousness; shivering slows, becomes intermittent, and then ceases; hallucinations, loss of pupillary reflex, bradycardia, atrial fibrillation and other arrhythmias, decreased ventricular fibrillation threshold, hypotension, decreased rate and volume of breathing, hyporeflexia or areflexia, muscle rigidity and decreased or absent voluntary motion, paradoxical undressing, and electrocardiographic changes (prolonged P-R, QRS, and Q-Tc intervals, and J [Osborn] wave) (Fig. 22.3)
- **Severe hypothermia:** May appear dead, profound coma, hypotension, respiratory depression, pupils fixed and dilated, severe muscular rigidity, areflexia (even to painful stimuli), atrial arrhythmias common, ventricular fibrillation easily induced with minimal patient movement, asystole, pulseless electrical activity and other arrhythmias, pulmonary edema, oliguria, and coagulopathy
- **Paradoxical undressing:** A conscious, profoundly hypothermic patient undresses, usually when near or just before death. Perception of warmth and cold relies on temperature receptors near the skin surface. When core temperature drops significantly, vasoconstricted vessels in the periphery may suddenly dilate, "fooling" the body into thinking it is warm. Combined with severely impaired consciousness and judgment, the feeling of warmth causes the affected individual to shed clothing. This is a poor prognostic sign.

**Treatment:**

**General Principles (all degrees of hypothermia)**

- **Preventing further heat loss** is the highest priority.
- Gently *remove* hypothermic individual *from the cold* to a shelter (tent, snow cave, or other natural or man-made shelters) and *insulate* against further heat loss.

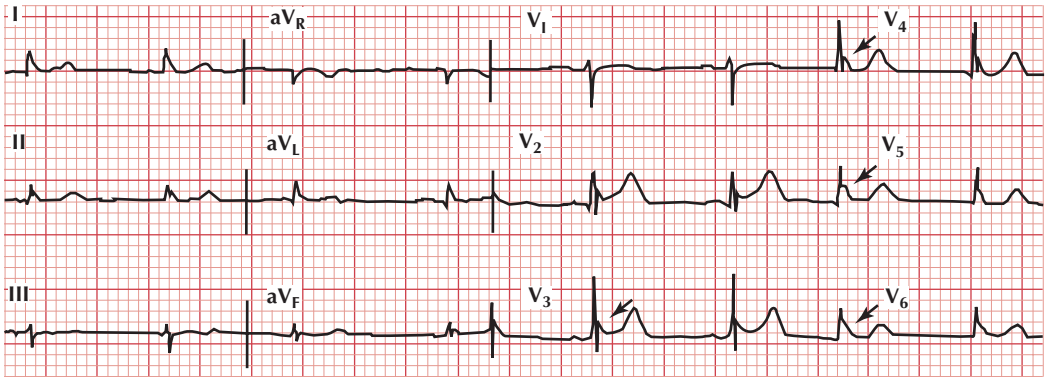


**Figure 22.3.** Systemic hypothermia. (From Goldberger A. *Clinical Electrocardiography: A Simplified Approach.* 7th ed. Philadelphia: Mosby; 2006.)

- Insulate using sleeping bags or by using a combination of sleeping bags and a vapor barrier such as a tarp, parka, or plastic bag.
- Minimize conductive heat loss by placing blankets or a sleeping pad between the patient and the ground.
- Affected individual should be kept in a horizontal position to minimize potential orthostatic worsening of hypotension.
- Remove all wet clothes by gently cutting them off.
- Hypothermia should always be treated before frostbite.

**Mild Hypothermia**

- Patients without impaired consciousness or severe confusion and dysarthria should be encouraged to eat warm food and to drink warm sugar- or calorie-containing noncaffeinated fluids to boost morale, supply calories, and curb dehydration.
- Once adequately protected from the cold and well insulated, the affected individual should be allowed to "shiver back to normal" (passive rewarming).
- Exercise is occasionally recommended in individualized circumstances.
- Certain practitioners advocate the use of active external warming measures (see following discussion) or noninvasive internal rewarming in the field with heated, humidified oxygen.
  - These rewarming measures are not likely to significantly increase the core body temperature and are controversial because they may hinder the shivering mechanism and consequently the warming process by warming the body peripheries.
  - Exogenous rewarming techniques are typically not needed in conscious, shivering individuals because shivering is more effective than exogenous rewarming, which inhibits shivering.

**Moderate and Severe Hypothermia**

- **Field Management**
  - Such patients are **unable to rewarm themselves.**
  - Field management involves preventing further heat loss, stabilizing the patient, and arranging for emergent transport to a medical facility for definitive care.
  - Basic life support principles should be applied. Cautious endotracheal intubation is indicated in patients with significantly impaired consciousness, coma, or respiratory distress. Moderately to severely hypothermic patients should be handled extremely gently because minimal motions may trigger ventricular fibrillation, which is often fatal.
  - After affected patients are adequately insulated, certain experts advocate exogenous rewarming in the field through various techniques, whereas other experts feel that any rewarming should be attempted in a hospital setting only.
  - Overall, when **shivering is absent** in moderately to severely hypothermic patients, some form of **active external rewarming** in addition to **noninvasive active internal rewarming** (e.g., heated humidified oxygen) is probably safe in the field.
  - Humidified warmed oxygen is not likely to have a significant effect on the core temperature but may prevent heat loss through the airways.
  - Slow administration of warmed intravenous D5 normal saline provides only a small amount of heat, but it may slowly correct intravascular volume depletion and may help stabilize the conduction system of the heart.
  - **Active external rewarming** involves the application of exogenous heat sources.
    - Forced heated air devices (e.g., Bair Hugger)
    - Hot packs or bottles placed over high-flow areas such as the neck, axilla, and groin
    - Hot pads or blankets
    - Radiant heat using heat lamps
    - Immersion of hands, forearms, lower legs, and feet in warm water (44°C–45°C [111°F–113°F])
    - Body-to-body contact
  - Active external rewarming techniques are relatively ineffective for core rewarming compared with active internal rewarming techniques. If used, care must be taken to avoid thermal burns with certain techniques.
  - Minimal warming of the cardiovascular and respiratory systems may help stabilize cardiorespiratory parameters even if the core temperature cannot be significantly increased.
  - Because the core temperature is essentially unaffected or at best minimally elevated, complications resulting from rapid rewarming do not usually occur. The amount of core rewarming, if any, achieved using these methods is controversial, but certain studies have claimed this as beneficial.
  - In conclusion, exogenous rewarming may be cautiously instituted in moderately to severely hypothermic patients, particularly if active internal core rewarming is not immediately available.
  - **Afterdrop considerations:**
    - The central perception of warmth caused by warm objects in contact with the body causes decreased vasconstriction in cold extremities.
    - Blood may be cooled rather than warmed as it flows through the periphery, particularly after exercise, and shunting of blood from the core to cold peripheries followed by the return of cool blood may cause a significant **"afterdrop"** in core body temperature in moderately to severely hypothermic patients.
    - Afterdrop, combined with dehydration, cold diuresis, relative peripheral vasodilation, fluid sequestration in tissues, and venous pooling, may lead to relative core hypovolemia and hypotension (rewarming shock).
    - There is no strong evidence that supports theoretical massive peripheral vasodilation and subsequent hypotension and shock resulting from exogenous rewarming. Recent studies have suggested that the "afterdrop" observed in temperature is not a true temperature drop and is related to differences in core temperature measuring techniques. The exception may be with whole body immersion in hot water, which must be avoided in all settings.
- **Cardiopulmonary Resuscitation (CPR)**
  - **Cardiopulmonary resuscitation (CPR) is indicated if there is no sign of pulse or breathing after assessing for 60 seconds.** It may be extremely difficult to detect pulse or respiration in profoundly hypothermic individuals.
  - **Do not initiate CPR in patients with a discernable pulse because a lethal cardiac rhythm may easily be precipitated.** A cardiac monitor should be used if available.
  - If ventricular fibrillation occurs at a core temperature below 30°C (86°F), do not administer medication, and limit defibrillation to one shock (200 J biphasic) because defibrillation rarely succeeds at these temperatures. Once temperature is 30°C (86°F) or above, medication may be administered (at longer-than-standard intervals), and defibrillation is repeated as indicated.
  - Historically, the most frequently used antiarrhythmic medication with hypothermia was bretylium tosylate; however, it is no longer included in the ACLS guidelines. Other medications are usually relatively ineffective and excessive pharmacologic intervention should be avoided

with a depressed, vasoconstricted cardiovascular system. Magnesium sulfate may help stabilize the ventricles and convert ventricular fibrillation in hypothermic patients.
    - The rate of chest compressions should be half normal.
    - No patient should be pronounced dead until he or she is "warm and dead."
    - CPR should not delay transfer to a medical facility for definitive management and should ideally be performed simultaneously with transfer, even if intermittent.
- **Hospital Management**
    - **Serious cardiovascular, CNS, and acid–base complications may occur with rewarming.**
    - All patients should be placed on a cardiac monitor, given warm humidified oxygen, and have intravascular access. Warm D5 normal saline (40°C [104°F]) is administered as indicated to correct dehydration and volume depletion (minimally elevates core temperature, <1°C per hour). Endotracheal and nasogastric tubes and bladder catheters are used in appropriate clinical situations.
    - Appropriate laboratory tests (complete blood count, comprehensive metabolic profile, coagulation studies, consider uncorrected arterial blood gas) must be performed, and close serial monitoring is required. Serum potassium >10 mEq/L in the presence of hypothermia is a strong indicator of death. ECG and chest radiograph should be performed.
    - Certain experts believe that limiting the application of external heat to the trunk may minimize the theoretical circulatory issues associated with active external rewarming. Nevertheless, several medical facilities use active external rewarming for most cases of mild and certain cases of moderate hypothermia.
    - Heated humidified oxygen raises the core temperature by 1°C–2°C per hour and may be a useful adjunct to other rewarming methods. Passive external rewarming alone in a warm environment will raise the core temperature by approximately 1.5°C per hour.
    - Active core rewarming techniques deliver direct heat to the body core and are the procedures of choice in severely hypothermic patients and in those with cardiovascular instability or poor perfusion. Temperature monitoring is best accomplished with an esophageal temperature probe.
        - The **gold standard** is **extracorporeal blood rewarming**—it may raise the core temperature by 1°C–2°C every 3–5 minutes.
        - Other effective methods include peritoneal lavage and, more recently, closed thoracic lavage (both raise the temperature by 2°C–3°C per hour).
        - Arteriovenous rewarming using a negative pressure device placed over the forearm is a promising technique to treat hypothermia. The device works by overcoming peripheral vasoconstriction by application of a significant vacuum pressure that "opens up" arteriovenous anastamoses and subsequently applies a direct thermal load to high-flow anastamoses through a chemical heating pad.
        - Less effective methods include gastric, colonic, and bladder irrigation.

COLD WATER IMMERSION

- Drowning or fatal cardiac arrhythmia may cause death earlier than hypothermia after cold water immersion.
- Hypothermia usually requires a significant duration of immersion (30 minutes to 2 hours) to kill a victim. On the other hand, drowning may occur relatively quickly as a result of the cold shock response.
- Sudden exposure to cold water causes an immediate and involuntary gasp that may result in aspiration of water and/or laryngospasm; this is followed by profound hyperventilation that results in respiratory alkalosis and subsequent muscle tetany and cerebral hypoperfusion.
- Breath-holding duration is significantly reduced (kayakers who roll may even have a hard time holding their breath for the duration of the roll).
- Severe tachycardia, increase in blood pressure, and vagal overload may induce lethal cardiac arrhythmias.
- Cold water-induced peripheral vasoconstriction facilitates rapid cooling of extremities and greatly decreases neuromuscular activity, making it almost impossible to tread water (without a flotation device), hold onto a flotation device, signal, or grasp anything (e.g., rescue line or hoist).
- If an arrhythmia does not occur, immersed individuals often drown because of excessive inhalation of water (particularly under turbulent conditions) and failure to initiate or maintain survival performance.

## Local Injury

### Frostnip

**Description:** Occurs before tissue freezing when cold induces local vessel constriction

**Symptoms:** Ice crystals may form on the skin surface, and the skin may appear whitish or pale.

**Physical examination:** Deeper tissues are soft and pliable, and affected skin is easily thawed by covering the face (with mittens or neck gaiter) or by placing affected hands in armpits or under other garments.

**Treatment:** Almost instant thawing may be followed by redness, pain, hypersensitivity, and occasionally swelling. Skin often peels a few days later. **Frostnip indicates inadequate protection** and **risky environmental conditions,** and attention should be directed toward preventing frostbite and further cold injury.

### Frostbite

**Definition:** Localized cold injury produced by freezing of tissues. Hands, feet, face (particularly nose), and ears are most frequently affected. Frostbites may occur in any environment where the temperature is below freezing (0°C [32°F]) and is frequently associated with hypothermia.

**Risk factors:** Environmental risk factors may include cold temperature, wind chill (wind velocity plus temperature), high altitude, high humidity, prolonged exposure, and direct skin contact with conductors such as cold metal, gas, or other liquids. Other risk factors may include previous frostbite, wet or inadequate clothing, dehydration, diminished mental capacity (e.g., drug-induced or other), associated trauma, alcohol use, tobacco use, use of drugs that cause vasoconstriction or alter thermoregulation, and comorbid medical conditions, particularly those that compromise circulation (e.g., atherosclerosis or diabetes mellitus).

**Athletes at risk:** Any athlete exercising or competing in cold weather, particularly without adequate protection, may sustain frostbite. Joggers are most frequently affected. Alpine and Nordic skiers, mountaineers and climbers, snowshoers, snowboarders, cold-weather distance runners and cyclists, speed skaters, snowmobilers, and players on almost any outdoor (cold weather) team sport (e.g., football, soccer) may suffer from frostbite.

**Pathophysiology:** Frostbite occurs with tissue freezing, subsequent tissue ischemia and release of inflammatory mediators, and eventual healing or tissue necrosis. The net result of frostbite involves varying degrees of direct cell damage and progressive tissue ischemia. Inflammatory mediators such as prostaglandins, thromboxanes, bradykinin, and histamine likely play critical roles in endothelial injury, edema formation, and arrest of dermal blood flow during all phases.

**Pathophysiologic classification: prefreeze, freeze–thaw, vascular stasis,** and **late ischemic phases**
- *Prefreeze:* tissue chilling, alternating vasoconstriction and vasodilation, capillary membrane instability, plasma leakage, and early edema
- *Freeze–thaw:* cyclic vasodilation and vasoconstriction at cold temperatures lead to tissue freezing and partial thawing (causes significant damage); extracellular ice crystal formation is followed by fluid shifts that cause intracellular dehydration and intracellular ice crystal formation; vascular endothelium may encounter microemboli; and arteriovenous shunting bypasses obstruction (leads to severe hypoxia of the affected tissue)
- *Vascular stasis phase:* continued plasma leakage, formation of ice crystals, vasospasm and vasodilation, and shunting; stasis coagulation may be more pronounced than that observed during the earlier stages
- *Late ischemic phase:* characterized by continued tissue ischemia, vascular thrombosis, arteriovenous shunting, autonomic dysfunction, denaturation of tissue proteins, and tissue necrosis and gangrene

**Clinical classification:** Historically, frostbite is classified into four categories: first-, second-, third-, and fourth-degree frostbite. A more clinically practical classification of **superficial** (previously first- and second-degree) and **deep** (previously third- and fourth-degree) frostbites is now more frequently used by clinicians.
- *Superficial frostbites* involve only the skin, and permanent tissue loss almost never occurs.
- *Deep frostbites* involve the skin and underlying tissues, which may include muscles, nerves, vessels, bone, and cartilage, and it is almost always associated with permanent tissue loss.
- It is important to realize that most frostbite injuries appear the same at initial evaluation (unless thawing is initiated) and that the *classification of frostbite is applied when tissue changes become evident after rewarming*.

**Presentation:**
- Frostnip is a mild form of frostbite before tissue freezing. Appropriate management of frostnip decreases the risk of a more severe injury.
- **Initial freezing:** coldness, numbness, clumsiness, and pain in affected extremity
- As freezing progresses, pain disappears, and the affected body part turns stiff and hard.
- Typically, all degrees of frostbite initially present with paleness, coldness, and firmness of affected tissue.
- The affected body part may also appear yellowish, mottled blue, and waxy.
- Initial tissue pliability may indicate superficial frostbite, whereas frozen solid tissue without pliability usually indicates deep frostbite.
- As tissue thaws with **rewarming,** signs and symptoms follow a relatively predictable pattern that illustrates the severity of tissue injury.
- Numbness and throbbing pain occur with tissue thawing.
- *Superficial frostbite:* hyperemia, increased sensation, and subsequent edema and blisters filled with clear or yellow fluid (Fig. 22.4A)
- *Less severe deep frostbite:* hemorrhagic blisters form and eschars form after blisters desquamate (Fig. 22.4B)
- *Severe deep frostbite:* no blebs or blisters form, edema is minimal (particularly proximal to injury), the affected body part lacks sensation, skin initially turns dark reddish and purple and eventually black, and mummification and autoamputation may occur weeks after injury (Fig. 22.4C)
- Mummification is the formation of a line of demarcation between viable and necrotic tissue.
- Progression of events: edema usually develops a few hours after thawing, blisters form within 24 hours, eschars may form over affected tissue after 1–2 weeks, and mummification may occur in 3–6 weeks.

**Treatment: General Considerations:** In-field treatment of frostbite should be balanced against time for evacuation and available treatment facilities. Frostbite is best rewarmed in an appropriate medical facility under strictly controlled conditions. The longer the tissue stays frozen, the greater the tissue damage. Therefore, certain experts recommend rapid in-field rewarming (provided appropriate equipment is available) if evacuation is not imminent (within a few hours) and if the affected body part can be absolutely protected from refreezing (more significant tissue damage may occur with refreezing after thawing). Spontaneous thawing may be allowed under the aforementioned circumstances if equipment is not available for rapid rewarming, but morbidity is higher in such situations rather than with rapid

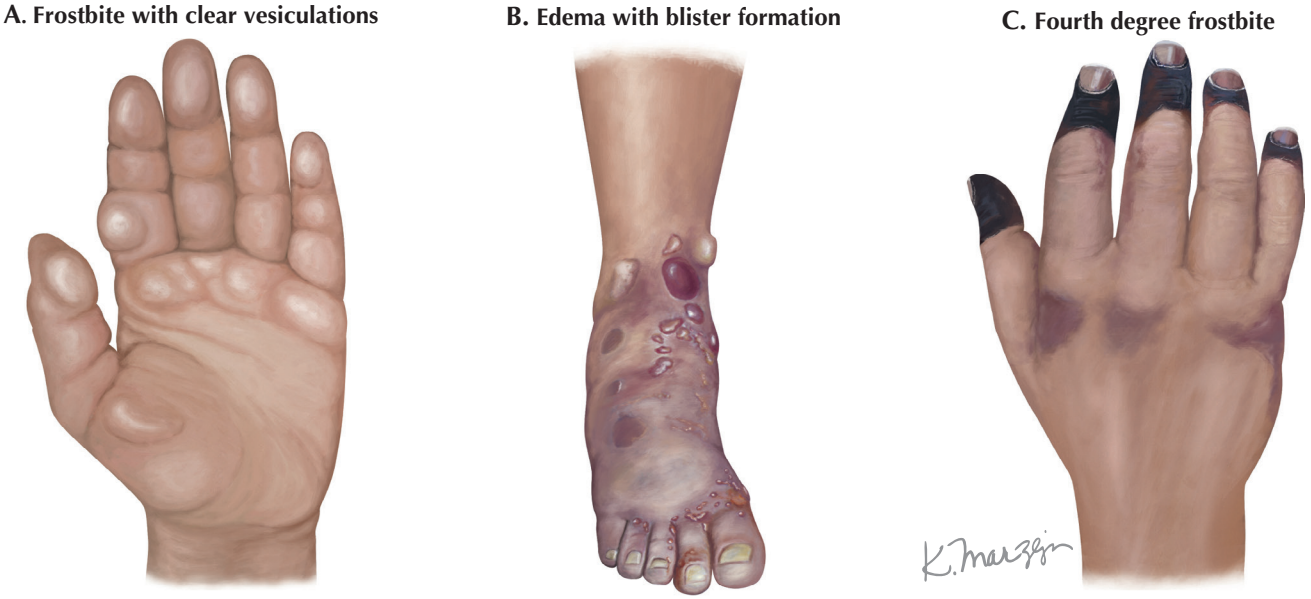


**Figure 22.4.** Frostbite.

rewarming, and this recommendation is highly controversial. As last resort, individuals with frostbitten feet may walk to safety, but they must realize that there is an increased risk of fracture and that their feet will likely thaw, which may leave them incapacitated.

**In-field Treatment**

- The *best treatment is prevention*. Adequate protection and appropriate planning minimize exposure of skin to cold environmental conditions.
- Continuously monitor tissue status in cold conditions, particularly if numbness is present, and seek safe conditions with any warning signs of frostbite.
- Individuals exposed to cold should:
  - Wear mittens instead of gloves
  - Ensure adequate trunk insulation (prevents peripheral vasoconstriction)
  - Keep head, face, and neck covered
  - Avoid wetting (via exposure or perspiration)
  - Avoid tight footwear and clothing
  - Avoid prolonged exposure to cold
  - Avoid using tobacco, alcohol, and vasoconstrictive drugs
  - Maintain adequate hydration and caloric intake
  - Avoid direct skin contact with cold conductive substances such as metal or gasoline
- Avoid cold-protecting ointments or emollients
  - Application of ointments or emollients, particularly to the face and ears, is a traditional technique believed by many to protect against frostbite. Use of ointments is particularly common in women and children of Finland, where the incidence of frostbite is high.
  - However, the use of ointments may actually be a considerable risk factor for developing frostbite, particularly of the face and ears. Ointments at best provide a negligible thermal insulation effect and may even have negative effects; these create a subjective skin-warming perception during acute cold exposure that may contribute to a false sensation of safety. The net result is perhaps an increased incidence of frostbite, probably resulting from neglect of effective protective measures.
- All frostbitten individuals should be evacuated to an appropriate medical setting as soon as possible.
- Ibuprofen or other anti-inflammatory medications should be administered (inhibits inflammatory mediators involved in tissue injury).
- Tight and/or wet garments should be removed and replaced with dry insulating garments that protect the affected body part from further exposure.
- Affected extremities should be adequately padded.
- Oral hydration should be encouraged.
- If equipment is available, certain experts feel that rapid rewarming may be performed during transport if transport time to a definitive treatment facility is minimal.
- Avoid tissue massage and use of dry or radiant heat.

**Definitive Treatment: Rapid Rewarming of Frozen Tissue**

- Rapid rewarming protocols are based on the work of McCauley and colleagues.
- Frostbite patients should be admitted to a specialized unit, if available. Affected body parts should be rapidly rewarmed in warm water (40°C–42°C [104°F–108°F]) for 15–30 minutes or until thawing is complete.
- Active motion during rewarming is helpful, but massage should be avoided.
- White blisters may be selectively debrided at the discretion of the treatment provider, hemorrhagic blisters should be left intact, and aloe vera should be applied every 6 hours.
- Affected body parts should be elevated, and splinting may be used as indicated.
- Antitetanus prophylaxis, ibuprofen or other anti-inflammatory medicines, and narcotic analgesics are administered.
- Certain experts advocate the use of prophylactic benzyl penicillin every 6 hours for 2–3 days.
- Perform daily hydrotherapy for 30–45 minutes with water at 40°C (104°F).
- Adequate intravascular hydration should be maintained. Certain experts use dextran (plasma expander) and vasodilators.
- Photographs of frostbitten tissue may be taken at admission, 24 hours, and every few days until discharge.
- Close serial follow-up should be arranged after discharge until all wounds are stable.
- The saying "Frostbite in January, amputate in July" illustrates that surgical debridement of severely frostbitten tissue is normally reserved as late treatment of frostbite, after the demarcation line between viable and necrotic or gangrenous tissue is clear.
- Traditionally, the decision of whether to debride (and occasionally reconstruct) or allow autoamputation of affected tissue must be taken months after initial injury, and early surgical intervention is reserved for escharotomy and fasciotomy, as indicated for circulatory compromise and compartment syndrome.
- Early surgical intervention involving free tissue transfer to improve vascularization of potentially viable tissue is currently being studied. However, early identification of viable tissue is clinically impossible. The use of technetium bone scanning (scintigraphy) within the first few days after injury may help predict eventual tissue demarcation by identifying deep tissue and bone infarction but fails to identify the condition of surrounding, potentially viable tissues. Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) show promise because they allow direct visualization of surrounding tissue and may thus facilitate a more accurate early assessment of the line of demarcation between viable and ischemic tissue.

**Long-term sequelae:** Significant tissue and limb loss may occur with deep frostbites. Other morbidities may include permanent cold sensitivity and susceptibility to cold injury, sensory loss, tingling and paresthesias, hyperhydrosis, chronic pain, growth plate disturbances (most common, phalanges in children), osteoarthritis, and heterotopic calcification.

### Chilblains

**Description:** Also referred to as *pernio*, chilblains ("cold sores") are characterized by tissue injury that may involve localized erythema, cyanosis, plaques, nodules, and occasionally in more severe cases, vesicles, bullae, and ulceration after *pronounced nonfreezing cold-induced vasoconstriction*.

**Pathophysiology:** May be related to vascular hypersensitivity, sympathetic instability, and subsequent lymphangiocytic vasculitis

**Symptoms:** Symptoms of intense pruritis, burning paresthesias, and skin changes may occur 12–14 hours after cold exposure.

**Treatment:** Includes anti-inflammatory medication, gentle rewarming, dry bandaging, and elevation to minimize swelling

### Trench Foot

**Description:** Otherwise known as *immersion foot*, trench foot involves tissue (e.g., muscles, nerves, and/or vessels) injury secondary to prolonged vasoconstriction and subsequent ischemia from *prolonged exposure to nonfreezing cold* and *wet conditions*.

**Pathophysiology:** May share a common pathophysiology with chilblains; progresses through prehyperemic, hyperemic, and posthyperemic phases

**Symptoms:** Occur after days and include painful paresthesias, numbness, and initial erythema, followed by pallor, mottling, and swelling
**Complications:** Vesiculation, ulceration, and gangrene may occur.
**Treatment:** Includes anti-inflammatory medications and keeping affected extremity dry, warm, protected, and elevated
**Prognosis:** Lifelong cold sensitivity, and occasionally pain, is common after injury.

### Miscellaneous Cold Injuries

#### COLD-INDUCED BRONCHOSPASM

**Description:** Cold may induce *bronchospasm*, particularly in asthmatics.
**Risk factors:** Most frequently affects athletes who exercise or compete in cold, dry conditions such as ice hockey and winter outdoor aerobic activities (e.g., running, cycling, and cross-country skiing)
**Treatment:** May include pre-exercise warm-up in a warm environment and/or pretreatment with beta-agonists, mast cell stabilizers, or leukotriene modifiers

#### COLD-INDUCED URTICARIA AND/OR ANAPHYLAXIS

**Description:** Most frequently affects athletes during pre-exercise warm-up in cold conditions
**Presentation:** Athletes present with wheals, hives, or pruritis; angioedema may be present, but true anaphylaxis involving laryngeal edema and hypotension is rare.
**Prevention:** Achieved with appropriate clothing and avoidance of cold-water activities
**Treatment:** Acute treatment may include antihistamines, and epinephrine administration may be required if anaphylaxis occurs. Prophylaxis using antihistamines (both H1 and H2 blockers may be used), the tricyclic antidepressant doxepin, and/or leukotriene modifiers is occasionally effective.

#### RAYNAUD'S PHENOMENON

**Description:** Vasospastic disorder characterized by initial pallor (ischemia secondary to vasoconstriction) followed by hyperemia (rebound vasodilation) of the digits (fingers most common) after cold exposure and/or emotional stress
**Pathophysiology:** May be primary idiopathic (Raynaud's disease) or secondary (Raynaud's phenomenon or syndrome) to various disorders
**Presentation:** The initial "white" ischemic phase may be followed by a "blue" cyanotic phase before the "red" hyperemic phase begins.
**Treatment:** Acute treatment includes warming the affected extremity and addressing the underlying disorders.
**Prevention:** May be achieved by avoiding direct cold exposure, ensuring adequate trunk insulation (minimizes peripheral vasoconstriction), wearing mittens instead of gloves, and using appropriate footwear; prophylaxis using vasodilating medications (e.g., calcium channel blockers and phosphodiesterase inhibitors) may be attempted if other primary preventive measures fail

#### COLD AGGLUTININ DISEASE

**Pathophysiology:** Usually develops after an infection and may follow mycoplasma infections or infectious mononucleosis in athletes; may also be idiopathic or associated with various other disorders
**Symptoms:** Symptoms of cyanosis, mottling, numbness, and pain in exposed body parts may occur when cold-activated IgM antibodies agglutinate red blood cells.
**Treatment:** Initially includes warming the affected body part and protecting from further exposure to cold

#### COLD-INDUCED RHINITIS

**Description:** Occurs in athletes exposed to cold air (e.g., skiers); it is a form of nonallergic, occasionally referred to as vasomotor, rhinitis that has minimal clinical significance and resolves after removal from the cold environment
**Treatment:** Ipatropium nasal spray may be administered if an athlete prefers treatment.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# HIGH-ALTITUDE TRAINING AND COMPETITION

# 23

Benjamin D. Levine • James Stray-Gundersen • Robert F. Chapman

## ALTITUDE ENVIRONMENT

- Barometric pressure is reduced at high altitudes, with a parallel decrease in inspired partial pressure of oxygen ($P_IO_2$); thus, **hypobaric hypoxia is the most prominent physiologic manifestation at high altitudes.** Fig. 23.1 shows the accepted terminology for the range of terrestrial altitudes as well as the magnitude of effects on selected outcome variables.
- Temperature decreases at a rate of approximately 6.5°C per 1000 m.
- Other features include dry air (increasing risk of dehydration), decrease in air density and therefore air resistance (marked effects in high-velocity sports such as cycling and onflight characteristics of objects—i.e., golf, baseball, or soccer), and increase in the amount of ultraviolet light (4% per 300 m), which increases the risk of sunburn.
  - Therefore, athletes must cope with hypoxia, cold, and dehydration and yet maintain maximal performance.
  - Timing of altitude exposure and degree of acclimatization are critical to successful outcomes.
  - Physiologic adaptation to high altitude may be beneficial. Altitude training is frequently used by elite athletes in an attempt to improve sea-level performance, but the manner in which it is completed will considerably affect the outcomes.

## EFFECT OF ALTITUDE ON EXERCISE

- *Oxygen cascade* is the term used to describe the physiologic effects of altitude on exercise: Oxygen moves from the **environment** (determined by the altitude achieved) to the **alveoli** (function of ventilation and hypoxic ventilatory response) across the **pulmonary capillary bed** (limited by diffusion) to be transported by the **cardiovascular system** (function of cardiac output and hemoglobin concentration) and diffused into **skeletal muscles** (depends on capillarity and biochemical state of muscles) to be used by muscle **mitochondria** (influenced by oxidative enzyme activity) for aerobic respiration and ATP production.
- **Altitude-induced hypoxia reduces the amount of oxygen available to perform physical activity.**
  - Maximal aerobic power ($\dot{V}O_2$max) is reduced by approximately 1% for every 100 m above 1500 m in nonathletic but healthy individuals.
  - For endurance-trained athletes, this effect is even greater—reductions in $\dot{V}O_2$max and performance can be identified at altitudes as low as 500 m and are linear (decrease of approximately 0.5%–1.5% for every 100-m increase in altitude) at altitudes ranging from 300 m to 3000 m.
    - Occurs because of diffusion limitation in both lung and skeletal muscles exacerbated by high pulmonary and systemic blood flow (cardiac output) in endurance athletes; severe hypoxemia can develop even during submaximal exercise (e.g., oxyhemoglobin saturation [$SaO_2$] < 80% in an elite male runner at a pace of 6 min/mile at 2700 m)
    - This magnitude of $\dot{V}O_2$max and performance decline at altitude, particularly in endurance-trained athletes, shows substantial interindividual variability. Data from several studies and other reports indicate the ability to maintain $SaO_2$ as a primary factor.
  - **During submaximal exercise at altitude, ventilation, lactate, and heart rate are greater for the same absolute work rate, which increases the sensations of dyspnea and fatigue.**
    - As a result of this increased rate of perceived exertion and dyspnea, training velocity (runners), training power output (e.g., cyclists), and $\dot{V}O_2$ are lower during training at altitude.
    - Heart rate and lactate responses to training at altitude are the same as training at sea level at the same relative effort, which complicates the determination of appropriate training zones/paces at altitude.
  - **Peak** blood lactate concentration is **lower** in individuals acclimatized to high altitude (termed *lactate paradox*), although this outcome is controversial and depends on nuances of workload and training altitude.
- Competitive performance outcomes at altitude, compared with that at sea level, is strongly influenced by the amount of aerodynamic drag on the body and the primary energy system utilized.
  - **Lower-velocity events** (e.g., distance running): in event distances requiring high levels of aerobic power (>2 minutes), performance is impaired at altitude because of a reduction in skeletal muscle oxygen delivery. In event distances requiring higher sustained power outputs (30 seconds to 2 minutes), performance may or may not be impaired at altitude, depending on the interplay of oxidative and glycolytic energy pathways.
  - **Higher-velocity events** (i.e., sprint running, cycling, or speed skating): the reduced air resistance at altitude actually results in an improvement in performance, despite systemic hypoxemia. In sprint events requiring short bursts of high-intensity activity (≤30 seconds), ATP production is not primarily dependent on oxygen transport. In high-velocity events lasting >2 minutes, the decline in aerobic power with reduced skeletal muscle oxygen delivery is effectively smaller than the influence of reduced air resistance. For example, as of January 2016, every world record in speed skating events from 500 m to 10,000 m in length was set at altitudes >1200 m, despite an expected reduction in $\dot{V}O_2$max at these altitudes.

## ACCLIMATIZATION PROCESS

- Chronic exposure to altitude stimulates acclimatization, which includes adaptations that improve **submaximal** work performance at altitude. Individual physiologic components of acclimatization have unique time frames of response, ranging from minutes to hours, days, months, or even generations. In addition, the rate and completeness of acclimatization is dependent on the altitude of residence—i.e., the hypoxic dose. For example, at high and extreme altitudes (≥4000 m), $\dot{V}O_2$max never returns to sea-level values despite prolonged acclimatization. At low altitudes (<2000 m), the maximal oxygen uptake may approach sea-level values after 1–2 weeks in nonathletic individuals.

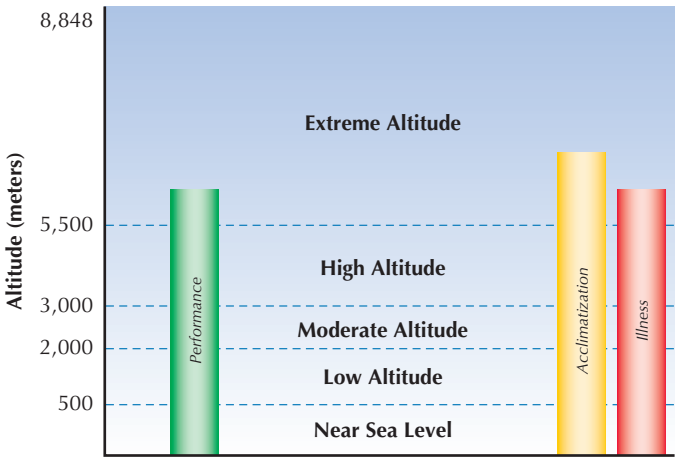


**Figure 23.1.** Accepted altitude definitions and ranges, from 2007 FIFA Consensus Conference. The amount of shading in the vertical bars represents the magnitude of variable (as influenced by altitude), with darker shading indicating a high level. (Data from *Scand J Med Sci Sports.* 2008:18[S1]:96-99.)

- Increases in alveolar ventilation and reductions in mixed venous oxygen content minimize the decline in exercise capacity at altitude—**this begins immediately on ascent**.
- Hyperventilation causes respiratory alkalosis, which stimulates renal excretion of bicarbonate and loss of plasma volume **over the first week** to normalize acid-base balance.
- Ventilation at rest (and to some extent during exercise) at altitude is influenced by the sensitivity of peripheral chemoreceptors; this hypoxic ventilatory response (HVR) is highly individualistic, with elite endurance athletes commonly showing blunted HVRs in comparison with untrained individuals. At low and moderate altitudes, a high HVR may affect the magnitude of dyspnea; at high and extreme altitudes, a high HVR may be critical for maintenance of even basic levels of physical activity and even survival.
- Sympathetic activation acutely (minutes to hours) increases heart rate and cardiac output so that oxygen delivery to tissues remains close to sea-level values at rest and during submaximal activity. By **2–3 weeks,** systemic and regional blood flow return to sea-level values as oxygenation improves. However, sympathetic activity continues to increase and may reach extraordinary levels, particularly at higher altitudes (>4000 m).
- The oxygen-carrying capacity of blood increases as a result of the increase in hemoglobin and hematocrit: **early** (1–2 days) increases result from plasma volume reduction; **later** (weeks to months) increases result from increases in red cell mass. **This critical adaptation offsets the reduction in atmospheric oxygen availability, thereby restoring oxygen transport to normal sea-level values**.
- Peripheral uptake of oxygen by skeletal muscles is facilitated by increased capillary density, mitochondrial number, myoglobin concentration, and 2,3-diphosphoglycerate (2,3-DPG), although these local changes may take weeks or months to manifest and are not universally observed.
- The buffering capacity of skeletal muscles may be increased as well.

## FAILURE OF ACCLIMATIZATION: HIGH-ALTITUDE ILLNESS AND OVERTRAINING

### Acute Mountain Sickness

- With moderate or higher altitudes (>2000 m) and rapid ascent rates (>300 m sleeping altitude per day above 3000 m), a maladaptive state called *acute mountain sickness* (AMS) may develop.
- Symptoms include headache, nausea, anorexia, fatigue, and difficulty in sleeping.
- Symptoms are usually mild and self-limited; rest and analgesics are sufficiently effective treatment.
- There is no evidence that competitive athletes are at any greater risk of developing AMS than nonathletes, although exercise may exacerbate the development of AMS, and physical activity should be appropriately reduced in symptomatic individuals.
- For patients who do not improve with rest, supplemental oxygen or descent to lower altitude virtually always results in prompt symptom relief.
- Other effective treatments include acetazolamide, dexamethasone, and simulated descent with a portable hyperbaric bag.
- AMS is best prevented by limiting the rate of ascent, allowing for rest or acclimatization days, maintaining adequate hydration, avoiding alcohol or sedatives during early acclimatization phase, and limiting training volume and intensity during first few days at altitude.
- Use of drugs to prevent AMS is discouraged in endurance athletes who are going to moderate altitude (<3000 m) unless a clear history of recurrent AMS is reported.
- The most frequently used drug is acetazolamide, which may be effective at low doses (125 mg at night or twice daily). However, diuretics including acetazolamide are on the World Anti-Doping Agency banned list as masking agents. Dexamethasone is probably more potent but is also banned as a steroid.

### Severe High-Altitude Illness

- In some individuals, AMS may progress to or be associated with more severe and life-threatening forms, including high-altitude pulmonary edema (HAPE) or high-altitude cerebral edema (HACE).
- **HAPE** is characterized by **dyspnea at rest,** cyanosis, severe hypoxemia, and noncardiogenic pulmonary edema.
- **HACE** is characterized by vomiting, **ataxia,** reduction in level of consciousness, and, in some cases, frank coma.
- Both of these syndromes can quickly result in death. **Immediate descent is mandatory.** High-flow supplemental oxygen or a portable hyperbaric bag, if available, may be useful adjunctive therapy while descending or if descent is delayed.
- Both HAPE and HACE are rare at moderate altitudes to which most athletes are exposed (<0.1%) although occurrence in athletes at low–moderate-altitude (<2000 m) should initiate search for congenital abnormalities of the pulmonary circulation.
- Medications that lower pulmonary arterial pressure may be used for adjunctive treatment of HAPE (this is less effective than descent and oxygen). Nifedipine has been most extensively studied and is effective both for treatment and prophylaxis. Phosphodiesterase inhibitors (e.g., sildenafil or tadalafil) are being investigated, but they may exacerbate AMS. Very recent studies suggest that dexamethasone is effective at preventing both AMS and HAPE and is the most effective adjunctive therapy for HACE. Drug treatment should be considered in athletes only if oxygen is unavailable or descent/evacuation is delayed. A staged slow ascent is the most effective preventive strategy, and it sidesteps the need to use banned substances for prevention.

### Overtraining

- Another potentially serious problem with training at altitude is the increased risk of overtraining.
- A comparison between exercise training and administration of medication, as shown in Fig. 23.2, is helpful. Every medication

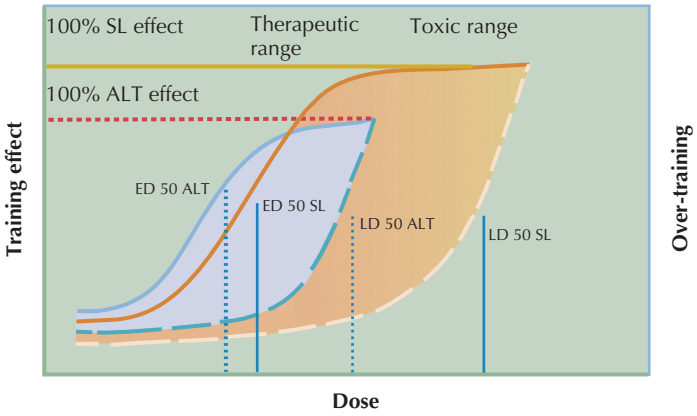


**Figure 23.2.** Toxic/therapeutic range of exercise training: the effect of high altitude. Although the mechanical stress of exercise is likely to be less at high altitudes because of the reduction in training speed, the metabolic stress may well be greater, at least with regard to the effect of hypoxia on the central nervous system. Thus, exercise training at high altitudes narrows the toxic/therapeutic range of exercise, possibly enhancing the training effect, but also increasing the risk of toxicity. SL = sea level, ALT = altitude, ED = effective dose at which 50% of the maximal response is achieved, LD = lethal dose at which 50% of the maximal toxicity is achieved.

has a specific dose–response relationship, accompanied by a toxic/therapeutic range. These parameters define the optimal dose and frequency of administration to maximize benefits but minimize side effects and toxicity. Exercise can be conceived as medication (so-called "exercise is medicine"): training response is proportional to volume and intensity (ED 50), but too much exercise results in clear toxic effects of musculoskeletal injury and systemic effects of overtraining (LD 50).

- **Overtraining may be precipitated by:**
  - **Inappropriately hard workouts:** Training at too high of a workload because of narrowed training zones and/or athlete inexperience; recovery training sessions/gentle workouts completed at too high of a workload
  - **Inadequate recovery:** Dehydration, sleep disturbances, and too short recovery times (either between workouts or between intervals within a hard workout)

## PRACTICAL STRATEGIES FOR IMPLEMENTING ALTITUDE TRAINING

From a practical perspective, critical issues for athletes and coaches are **"indications"** for altitude training, and if indicated, use of an appropriate training model and application of an appropriate "dose" of both altitude exposure and training

### Indications

#### Which Athletes Should Use Altitude Training?

- Athletes in events where $\dot{V}O_2$max is important for performance (i.e., endurance athletes in events lasting >2–3 minutes); sprint athletes may benefit from neuromuscular/kinesthetic training because of faster speeds allowed by reduced air resistance.
- Athletes who compete at altitude because appropriate acclimatization will mitigate performance declines at altitude.
- Because supervised training camp is a powerful stimulus for training, even in elite athletes, already well-trained athletes with general and competition-specific fitness are most likely to obtain benefits from altitude.
- **Altitude training is no substitute for a focused, well-designed training program with appropriate rest and nutrition.**
- Athletes who respond best to altitude training exhibit a robust and sustained increase in erythropoietin concentrations at altitude and a strong ability to maintain velocity and oxygen flux during training. These responses may be genetically determined.

#### Which Athletes Should Not Use Altitude Training?

- Team sports that depend more on strategy and technique for success (e.g., water polo, baseball, field hockey, or handball) are unlikely to derive much benefit from altitude training for sea-level performance.
- Sports that depend on normal air resistance for fine motor skills (e.g., basketball, archery, tennis, or soccer) are likely to be impaired by altitude training; such sports may require an adjustment period both at altitude and on return to sea level to compensate for differences in projectile movement through air at altitude.
- Swimming is most controversial. Recent data suggest a performance benefit with the live high–train low model of altitude training (see the Altitude Training Models section below), even during shorter events that are not traditionally thought to be limited by oxygen delivery. Because of extremely low mechanical efficiency, performance in swimming is more strongly tied to **biomechanics** than **physiology.**
- Iron-deficient athletes should not engage in altitude training (see the Nutritional Factors section below).
- Athletes with a small and brief increase in erythropoietin at altitude and those who cannot maintain training speeds even at low altitudes are least likely to respond well.

### Altitude Training Models/Best-Practice Recommendations

- For competitions at altitude
  - **Acclimatization** is critical and clearly improves performance **at altitude.**
  - Adequate time should be allowed for acclimatization to minimize altitude-mediated performance decrements. Evidence suggests that performance is worst on arrival, improves incrementally over a period of approximately 14–19 days, and does not significantly improve beyond that time point (out to 28 days).
  - If adequate time for acclimatization is not possible, anecdotal experience among athletes suggests that competing immediately (i.e., within 2–4 hours of arrival) at altitude may be best because it may minimize negative effects of plasma volume loss or poor sleep at altitude.
  - Recent studies have suggested that for low-altitude competitions (500–2000 m), living at altitudes higher than that of the competition may be worse for competitive performance than living at competition altitude, unless acclimatization for at least 19 days is possible.
- For competitions at sea level
  - Living and training at altitude has not been shown to improve performance at sea level; this is likely because of a reduced training effect at altitude (i.e., training slower at a lower oxygen flux).
  - **Living at altitude and training as close to sea level as possible (known as *living high–training low*) does improve sea-level performance.** Additional iterations and improvements of this model have shown that only completing high-intensity workouts at low altitude (i.e., remaining at moderate altitude for gentle/recovery workouts) produces the same performance outcomes as completing all workouts at low altitude.
  - A recent meta-analysis reported mean increases in aerobic power of approximately 4.0% following application of the live high–train low model of altitude training, while classical

altitude training (live high–train high) produced a nonsignificant change in aerobic power of 0.9% in subelite and 1.6% in elite athletes.

## Altitude Dose Recommendations (for Improving Performance at Sea Level)

- The variables that influence the "dose" at altitudes are critical in the amount of sea-level performance enhancement experienced with live high–train low altitude training.
  - **How high to live:** Locations that are 2200–2700 m above sea level will maximize acclimatization and minimize complications. Living lower may not provide an adequate erythropoietic stimulus, while living higher adds components of acclimatization that negatively affect performance (e.g., poor sleep, AMS, and greater ventilatory work during exercise). Simulated altitude techniques, such as sleeping in nitrogen-enriched environment to simulate hypoxia of high altitude, may promote similar benefits of terrestrial altitude living; **however, hypoxic exposure of >14 hours/day may be required** in most individuals to significantly increase hemoglobin mass, and excessive bedrest must be avoided.
  - **How long to reside at altitude:** Erythropoietin levels increase acutely, are clearly still elevated after 2 weeks, and return to sea-level values by 4 weeks. Thus, a minimum of 3–4 weeks appears necessary to develop sufficient acclimatization and augmented red cell mass, particularly for competition at sea level. "Intermittent" hypoxic exposures ranging from a few minutes, on and off, to up to 3 hours at extremely high altitudes have no benefits for sea-level performance.
  - **How high to train: High-intensity training:** For workouts that are faster than lactate threshold/maximal steady-state pace, best practice is to complete the workout at as low an altitude as possible, preferably <1500 m. **Low-intensity training:** Can be performed at an altitude that minimizes the logistical travel burden to low altitude; however, **low-intensity training must be performed at a relatively easy pace to prevent overtraining. Careful monitoring of heart rate and/or blood lactate during training may help ensure the correct training pace.**
  - **When to compete on return to sea level:** Emerging scientific and anecdotal data suggest competing either immediately on return (<72 hours after return to sea level) or after 2–3 weeks of sea-level training. Competing immediately allows the athlete to take advantage of the highest levels of hemoglobin mass but does include the potential negative effects of higher amounts of respiratory muscle activity from ventilatory acclimatization effects. On the other hand, competing after 2–3 weeks of sea-level training may facilitate enhanced chronic training effects, secondary to increased oxygen delivery to skeletal muscles at sea level. Athletes anecdotally report feeling "flat" or "off" when competing during a window of approximately 3–14 days after return, but there is no clear evidence to support this.

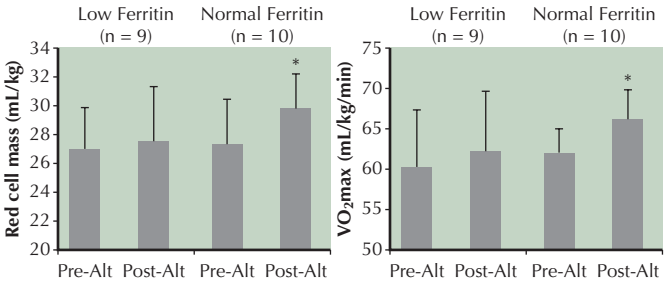


**Figure 23.3.** Red cell mass *(left)* and maximal oxygen uptake *(right)* measured before and after altitude training camp (4 weeks at 2500 m) in groups of athletes with low serum ferritin (women, <20 ng/mL; men, <30 ng/mL) and normal serum ferritin upon departure for altitude* significantly different from prealtitude measurement; $P < .05$ (Stray-Gundersen and Levine, unpublished data).

## Nutritional Factors

- Nutritional factors, particularly iron stores, play a critical role in the ability to respond to altitude training.
- Numerous (both male and female) athletes exhibit reduced iron stores based on low serum ferritin. Such athletes are unable to increase their $\dot{V}O_2$max and the volume of red cell mass (blood volume – plasma volume), despite an acute and sustained increase in EPO; thus, they are unable to obtain the primary potential benefits of altitude acclimatization (see Fig. 23.3).
- Simple measurement of hemoglobin or serum iron is inadequate because it does not reflect bone marrow iron stores. Moreover, because iron is a critical moiety in myoglobin as well as mitochondrial cytochromes, iron deficiency may not only compromise oxygen-carrying capacity but also inhibit oxygen extraction (arteriovenous $O_2$ difference) and reduce $O_2$ flux, thereby limiting $\dot{V}O_2$max and performance even in nonanemic athletes. Before initiation altitude training of any duration, iron stores (ferritin) must be normal (women, minimally >20 ng/mL; men, >30 ng/mL).
- High doses of oral iron (150–400 mg elemental iron daily in divided doses) are usually required to maintain ferritin levels during altitude training. Even athletes with normal iron stores at the start of an altitude training program experience a rapid decline in serum ferritin levels at altitude and must be supplemented and closely monitored.
- Oral iron doses are best tolerated and absorbed in liquid pediatric preparations—Feosol, 15 mL dissolved in orange juice and mixed with 500 mg vitamin C, 1–3 times/day, 0.5 hours before or 1–2 hours after a meal; more frequent administration of smaller doses may be preferred if gastrointestinal (GI) distress develops. Considering the GI distress caused by oral formulations, severely iron-deficient athletes should consider intravenous administration of iron, which is safe and efficient.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# TRAVEL CONSIDERATIONS FOR THE ATHLETE AND SPORTS MEDICAL TEAM

# 24

Craig C. Young • Nicolai Esala

## GENERAL PRINCIPLES

Modern transportation systems facilitate easy access to most regions of the world. While this has allowed for the rapid growth of international competition, it also creates unique physiologic and psychological challenges for athletes as well as the sports medicine team.

## JET LAG AND CHRONOBIOLOGY

- The American Academy of Sleep Medicine defines jet lag as a syndrome involving insomnia or excessive daytime sleepiness following travel across at least two time zones. Jet lag is a syndrome of symptoms manifested by physiologic adaptations that occur when the body is shifted to a new time zone.
- Travel fatigue is a more complex combination of physiologic, psychological, and environmental factors that develop during travel; it may accumulate over the course of a season and reduce an athlete's capacity to recover and perform.
- Chronobiology is the field of biology that examines cyclic phenomena in living organisms and their adaptation to solar- and lunar-related rhythms; these cycles are known as *circadian rhythms*.

### Physiology of Jet Lag

- An average human experiences endogenous cycles of energy, mood, and activity that last approximately 25 hours.
- The primary pacemaker is the suprachiasmatic nuclei of the hypothalamus. When a traveler changes time zones, this pacemaker must undergo entrainment, which is the process of resynchronization with the new environmental light–dark cycle. Physiologic mechanisms involved in this process include:
  - Melatonin, which is a hormone that is typically secreted at dusk by the pineal gland and helps the body anticipate the daily onset of darkness.
  - Adenosine accumulates when a person is awake and causes progressive sleepiness.
    - Adenosine accumulation is blocked by caffeine.
  - Direct neural pathway from the retina
    - Blue wavelength light, in particular, can interfere with the sleep cycle.
  - Arginine vasopressin
- Zeitgebers are environmental cues that help reset the pacemaker; these include: light, temperature, exercise, social interactions, and eating and drinking patterns.
- Disorders of circadian rhythm are most commonly experienced in the setting of a jet lag when a new sleep–wake cycle is required on entering a new time zone.
  - Signs and symptoms of jet lag include changes in mood, headaches, digestive difficulties, and increased susceptibility to illness. Typically, athletes also suffer decreases in cognition, concentration, visual acuity, and memory. These changes often have adverse effects on physical and athletic performance.
- The rate of adjustment to a new time zone is typically a day for each time zone crossed.

### Prevention of Jet Leg and Travel Fatigue

- A structured athlete travel program that encompasses preflight, inflight, and postflight periods is the first step in establishing an effective approach to travel fatigue and jet lag.
- Because there is no physiologic adaptation with repetitive time zone transitions, each long-distance journey is unique and requires its own specific travel strategy based on the direction of travel, duration, and times of arrival/departure.
- By adopting a structured program and fatigue monitoring system, athletes and medical staff can help minimize travel-related physiologic and psychological issues, limit symptoms, and improve overall performance.
- **Preflight component**
  - Although it may be difficult because of schedule restrictions, introducing a schedule within 7 days of travel is generally optimal.
  - Consider gradually changing the sleep–wake cycle and meals to the new time zone by shifting an hour a day.
    - Consider adjusting training to the destination time zone
    - Avoid bright light for 2–3 hours before bedtime
  - An emphasis should be placed on getting enough sleep before travel to reduce sleep debt.
- Inflight component
  - If they have not already done so, advise athletes to adjust their schedule to be in sync with the destination time zone as soon as they board the plane to assist them in preparing for the destination (e.g., watches, meals, and sleep schedules).
  - Maintaining appropriate hydration should be a priority.
  - Avoid light-projecting devices, such as computers, tablets, and movies.
- Postflight component
  - The postflight period stretches 2–4 days or more beyond arrival. During this time, the activities of an athlete (including meals, sleep, rest, and recovery) should be strategically planned by the staff to accommodate rapid circadian adjustment.
  - The most effective intervention in such situations is a combination of scheduled light therapy, light avoidance, and melatonin.
  - Additional fatigue countermeasures include the judicious use of napping and caffeine, both of which can synergistically improve alertness and reduce symptoms of fatigue.

#### Pharmacologic Measures

**Melatonin**: Melatonin supplements can aid in managing jet lag symptoms, both preflight and postflight.

- Preflight low doses (0.5–1.5 mg) of melatonin are most effective, whereas higher doses (3–5 mg) are recommended after flight.
- Pretravel melatonin may be used to gradually shift the feeling of dusk and bedtime to the anticipated time zone.
- Doses should be taken 30 minutes before bedtime on the night of travel and the initial 2–3 nights after arriving at the destination. This will mitigate sleep disturbances associated with jet lag while enhancing circadian adaptation.

**Sedatives:** Athletes who do not suffer from jet lag or who do not respond to melatonin and experience 1–2 days of insomnia on arrival will likely benefit from the use of a traditional medium-acting (20–30 minutes) or medium half-life (6 hours) sedative (e.g., eszopiclone or temazepam). Very-short-acting (<15 minutes) and short half-life (4 hours) sedatives (e.g., zaleplon or zolpidem) can be useful for sleep during the flight. However, caution must be exercised with inflight use of sedatives because this may increase the risk of deep vein thrombosis (DVT) and decrease responsiveness in the event of an inflight emergency. Use of sedatives may be considered illegal in certain sports without a therapeutic use exemption waiver.

**Stimulants:** Caffeine and other stimulants (e.g., modafinil) may be useful in combating fatigue. However, use of stimulants may be illegal for some athletes without a therapeutic use exemption waiver.

- Caffeine: The strategic use of caffeine (e.g., a 50–200-mg pill or beverage) in combination with a 15- to 30-minute nap has been shown to be effective in improving cognitive function in sleep-deprived states and at the lowest point of the circadian cycle. Athletes should be cautioned that caffeine above certain levels is often considered illegal by doping codes and may result in suspension, loss of medals, and vacation of victories.

### Nonpharmacologic Measures

**Preadaptation and light therapy:** Light exposure is the primary cue for circadian rhythms. Exposure to bright light of adequate intensity and duration can advance or delay circadian rhythms based on the timing of exposure. Bright light exposure in the morning will help advance the body clock, while exposure in the late evening will help delay it. Attempts to shift circadian rhythms with preflight exposure to bright light before departure have been successful during both eastward and westward travel. Exposure to natural bright light promotes circadian shifts. Avoidance of bright light, particularly short wavelength, blue light in the evening may help with shift for eastward travel. Use of blue blocking glasses, such as amber safety glasses, may diminish this exposure. Athletes should be cautioned to avoid evening use of tablets, computers, televisions, and mobile phones in order to minimize blue light exposure.

**Sleep:** Sleep can be used to acclimate or adjust an athlete's circadian rhythm before travel or decrease symptoms of sleepiness upon arrival. Shifting the sleep schedule 1–2 hours toward the destination time zone in the days preceding departure may shorten the duration of jet lag. In addition, strategic napping has been discussed as a potential method to mitigate the symptoms of jet lag. The best time to nap (inflight or postflight) is nighttime in the destination time zone. "Power naps" (20 minutes) do not result in sleep inertia and may decrease daytime sleepiness in individuals experiencing jet lag. However, naps of >20 minutes may delay sleep adaptation and slow resynchronization.

## DEEP VEIN THROMBOSIS (DVT) AND TRAVEL

- DVT is a condition wherein a blood clot develops in the deep veins, most commonly in lower extremities. A part of the clot has the potential to break off and travel to the lungs, causing a potentially life-threatening pulmonary embolism (PE).
  - Prolonged periods of inactivity caused by space limitations may diminish circulation and produce lower extremity edema. Prolonged sitting with bent knees compresses the popliteal veins, which is another potential risk factor for clot formation.
- "Economy class syndrome": Long-distance air travel has been associated with a 2–4-fold increased risk of venous thromboembolism (VTE), including DVT.
  - Environmental factors in flight, such as low oxygen, low humidity, and low cabin pressure, contribute to dehydration, which concentrates the blood; this effect is worsened when passengers consume alcohol or do not adequately replenish fluids lost due to dehydration. However, there is no evidence that dehydration is directly associated with VTE.
  - Certain reports have suggested that flights of ≥8 hours increase the risk of VTE in the presence of additional risk factors in a patient.
  - Other risk factors include age over 40 years, obesity, and estrogen use.
  - Athletes and individuals in general good health are at a lower risk of VTE.
- A DVT most often originates in the calf, with persistent cramping or "charley horse" that intensifies over several days. This pain may be accompanied by leg swelling and discoloration.
- In most cases, travel-related VTE occurs in the first 1–2 weeks after travel. The risk returns to baseline after 8 weeks.
- **Treatment:** early detection and anticoagulant drugs (e.g., heparin, low-molecular-weight heparin, warfarin: see Chapter 31, "Hematologic Problems in Athletes" for additional details)
- **Prevention:** maintaining hydration, exercise, and wearing support stockings may help decrease the overall risk. Periodic activity, approximately every 2 hours, can include isometric exercises, walks along the aisles, or stretching exercises. Below-the-knee graduated compression stockings that provide 15–30 mmHg of pressure have been advocated as a preventive measure. Drugs such as aspirin have antithrombotic properties but are not recommended as a prophylactic measure in otherwise healthy individuals.

## INFECTIOUS DISEASES

- Infectious diseases are common among travelers. Travel to developing countries results in approximately 8% of travelers requiring medical treatment. While preventing athletes from acquiring infections may be challenging, use of common sense approaches can minimize the risks. Team physicians should investigate which infectious agents are common at the destination, educate the athlete on appropriate preventative measures, and have treatment available.
- **Traveler's diarrhea (TD):** Approximately 50% of travelers, even in low-risk industrialized countries, will develop at least one diarrheal episode per short-term trip (Fig. 24.1). Besides the discomfort and time lost to bowel movements, fluid and electrolyte imbalances have the potential to adversely affect athletic performance.
  - Preventive dietary measures include avoidance of tap water, ice, unpasteurized dairy products, raw vegetables, salads, undercooked meat, and seafood.
  - In 2012, the British Olympic Medical team was successful in reducing illness by educating athletes regarding hygiene, including the importance of lowering the toilet seat lid before flushing in order to minimize risk of aerosolization of fecal bacteria.
  - Bismuth subsalicylate (2 tablets four times/day) may be used for prophylaxis and decreases the risk of TD by 50%.
  - Prophylactic antibiotics were considered an effective measure in the past but are no longer recommended owing to the developing antimicrobial resistance; nevertheless, short-term use for critical trips or in high-risk patients may be considered.
    - Parasites, not bacteria, are the most common cause of diarrhea in developing countries.
  - Antimotility agents, such as loperamide and diphenoxylate–atropine, may be used in cases of nonfebrile, nonbloody diarrhea.

| Etiologic organisms | *E. coli (enterotoxigenic)* | *Shigella* | *Salmonella* | *C. jejuni* | *V. parahemolyticus* | *Rotavirus Norwalk virus* | *E. histolytica* | *G. lamblia* |
|---|---|---|---|---|---|---|---|---|
| Approximate relative incidence of common infectious agents in travelers' diarrhea | 50% | 15% | 5% | 10% | 2% | 14% | 2% | 2% |
| Typical clinical features | Diarrhea, nausea, vomiting, malaise, fever | Diarrhea, tenesmus, cramps, fever | Diarrhea, cramps, nausea, vomiting, fever | Diarrhea, cramps, anorexia, fever, malaise | Diarrhea, cramps, vomiting, headache | Diarrhea, cramps, vomiting, fever, myalgia | Diarrhea, alternating with constipation, nausea, gas, cramps, fatigue | Foul diarrhea, cramps, foul gas, distention, rumbling, fatigue, weight loss |
| Incubation period | 4–24 hours | 1–3 days | 6–48 hours | 1–7 days | 12–24 hours | 18–48 hours | 1–7 days | 12–15 days |
| Duration of illness | 3–4 days | 2–7+ days | 3–4 days | 1–7+ days | 1–3 days | 1–7 days | 3–14+ days | 7+ days |
| Blood in stool | – | + | ± | + | ± | – | ± | – |
| Proctoscopy: ulcers, friable and hemorrhagic mucosa | – | + | ± | + | ± | – | ± | – |
| Diagnostic method | Clinical features and exclusion of other agents | Stool culture: EMB agar | | Stool culture: selective blood agar | Stool culture: TCBS agar | Clinical features and exclusion of other agents | Stool examination for parasites: Smear; Concentration | |
| Fecal leukocytes (seen with invasive bowel pathogens) | – | + | + | + | ± | – | – | – |

**Figure 24.1.** Traveler's diarrhea: incidence and differential features.

- Mosquito-borne illnesses including malaria, dengue fever, and chikungunya are common causes of febrile illness in travelers and should be considered even if symptoms do not occur until after travelers return home.
  - Prevention measures include wearing loose-fitting long-sleeve shirts and long pants treated with permethrin, use of bed netting, and limiting evening outdoor activities.
  - Sprays to prevent bites include 20%–30% N,N-diethyl-meta-toluamide (DEET), 20% picaridin, or 30% oil of lemon eucalyptus.
- Parasites are a potential concern particularly in extreme sport athletes in tropical and subtropical climates, who are at a particularly high risk of contracting nematode and protozoan infections (Fig. 24.2).
- **Vaccinations:** The Center for Disease Control's online Yellow Book contains recommendations of vaccinations for most destinations; these should be offered to all athletes well in advance of departure.

## INFLIGHT MEDICAL EMERGENCIES

- Occasionally, physicians will find themselves called upon by a flight crew to evaluate a passenger who has developed a medical issue. A physician may determine that he or she can treat and stabilize that patient or that an emergency landing will be needed.
- Typical airline first-aid supplies will include stethoscope, blood pressure cuff, oral airways, bag masks, CPR masks, IV supplies, gloves, needles, syringes, antihistamines (oral and injectable), atropine injection, albuterol inhaler, aspirin, 50% dextrose injection, epinephrine injection, lidocaine injection, nitroglycerin tablets, and AED.

## PREPARING A MEDICAL PLAN FOR TRAVEL

- Determination of contents of a medical kit (Table 24.1) will depend on numerous factors, including quality and availability of health care at the destination, the sport being covered, and potential injuries. In more remote locations, as well as in developing countries, inclusion of sterile equipment should also be considered because these may not be readily available. For international travel, physicians should carry following documents:
  - Letter from the team requesting medical coverage
  - List of medical supplies, including medications being transported
  - Medical license and passport
  - List of local medical contacts
- **Travel medical insurance coverage:** consideration should be given to the following issues:
  - 24-hour physician-backed support center
  - Network of referral providers
  - Guarantee of medical payments abroad
  - Direct pay to foreign hospitals and physicians
  - Preauthorizations or second opinion requirements before emergency treatment or surgery
  - Coverage of higher-risk activities (e.g., parasailing, mountain climbing, or scuba diving)
  - Coverage for pre-existing conditions
- **Medical evacuation:** Medical evacuation is very expensive, particularly if not contracted in advance. For international

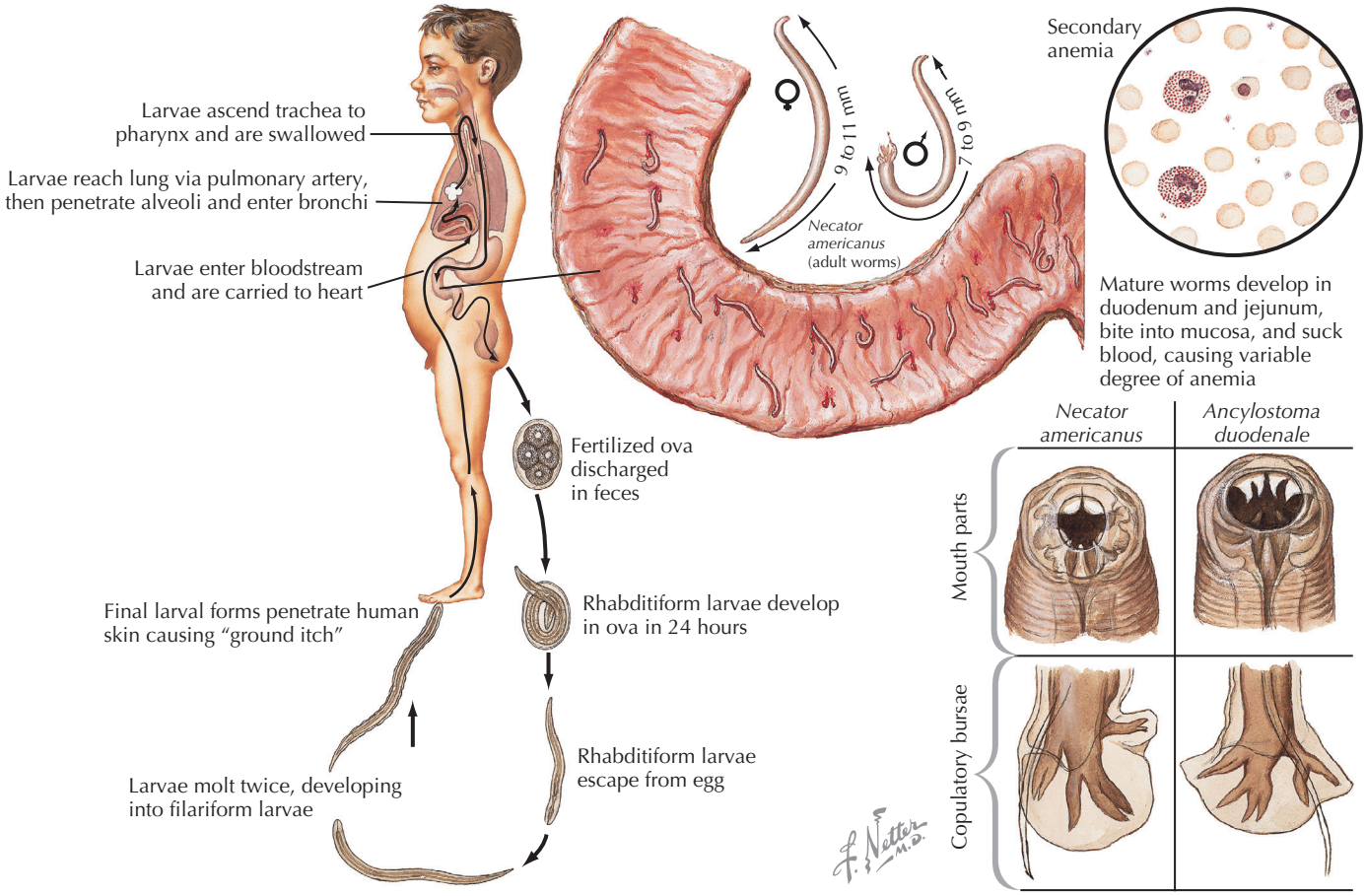


**Figure 24.2.** Parasitic diseases: necatoriasis and ancylostomiasis.

## TABLE 24.1 SAMPLE INVENTORY LIST FOR TRAVEL MEDICAL SUPPLIES

| | |
|---|---|
| **Documents** | • Inventory list<br>• Medical license<br>• Hospital ID<br>• Business cards<br>• Passport*<br>• Copy of prescriptions for personal medications*<br>• Letter describing team responsibilities*<br>• Medical evacuation contact numbers*<br>• World Anti-Doping Agency (WADA) prohibited substance list or online access or sports equivalent† |
| **Medications** | • Acetaminophen<br>• Anti-inflammatory agents<br>• Antibiotics<br>• Gastrointestinal medications (for nausea, diarrhea, and heartburn)<br>• Corticosteroid (topical)<br>• Antihistamines (e.g., diphenhydramine; plus a nonsedating variety)<br>• Anesthetic (injectable)<br>• Albuterol inhaler‡<br>• Glucose tablets |
| **Supplies** | • Gloves (examination and sterile)<br>• CPR mask<br>• Thermometer (and covers)<br>• Stethoscope<br>• BP cuff<br>• Bandage scissors<br>• Alcohol prep pads<br>• Povidone iodine<br>• Bandages<br>• Triangular bandage<br>• Elastic bandages<br>• Sterile contact solution<br>• Penlight<br>• Fluorescein strips<br>• Tampons<br>• Cotton applicators<br>• Laceration kit<br>• Butterfly bandages/adhesive skin closures<br>• Scalpel<br>• Forceps<br>• Hemostats<br>• Needles (18G and 25G)<br>• Syringes<br>• Sharps container<br>• Splints<br>• Mylar blanket<br>• Plastic zipper storage bags<br>• Rubber bands<br>• Pen<br>• Electric plug converters*<br>• Voltage converter*<br>• International (GSM-capable) cell phone* |
| **May Be Considered if Athletes Are Not Drug Tested** | • Corticosteroids (injectable)‡<br>• Decongestants‡<br>• Jet lag medications‡ |

*For international trips
†For coverage of athletes subject to drug testing
‡Carry and use with caution—may be restricted by WADA or other sports governing bodies
Note: Do not carry any pain medication. All pain medications should be carried only by the individual for whom it was prescribed: transportation by any other individual or as "team supplies" is against US Drug Enforcement Agency (DEA) regulations and may result in loss of DEA certificate, medical license, or prosecution.

travels, planners should strongly consider having both medical and medical-evacuation (back to the home country) insurance coverage for each member of the traveling party.

## MEDICOLEGAL CONCERNS

As of early 2016, in order to legally practice medicine, physicians traveling away from their home state are required to have a valid medical license (or reciprocity) in all locations that they intend to treat patients. Although legislation is currently pending to allow team physicians to care for their team for limited periods of time in other states, these situations are currently covered by individual state regulations. The Good Samaritan Act should not be counted on to cover team physicians because this varies from state to state. In addition, physicians should check with their malpractice insurers to ensure that their activities are covered while traveling.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE ROLE OF SPORT PSYCHOLOGY AND PSYCHIATRY

25

David B. Coppel

## WHAT IS SPORT PSYCHOLOGY?

### Definitions

- The American Psychological Association (APA) defined sport psychology (SP) as
  - Helping athletes apply psychological principles to achieve improved or optimal sport performance and mental health (MH)
  - Increasing knowledge regarding the impact of sport/exercise and physical activity on psychological development, health, and well-being over the lifespan
- The APA is expanding the definition of SP, as a result of its SP Proficiency certification program, to include psychological skills, optimizing the well-being of athletes, dealing with organizational and systemic issues in sport settings, and understanding social and developmental issues related to sports participation.
- **In a multidisciplinary context, SP is increasingly seen as a strong component of the sports medicine team.**
- Psychology has played a well-established and vital role in physical medicine and rehabilitation (PMR) programs; hence, its application in sports medicine is only natural.
- Increasing references to psychological factors or influence of emotional/MH factors on physical health and sports injury recovery and performance.
  - Team Physician Consensus Statements (TPCS) discussing sports injuries and concussion have commonly included psychological factors.
  - The National Collegiate Athletics Association (NCAA) publication "Mind, Body and Sport: Understanding and Supporting Student-Athlete Mental Wellness" depicts a bidirectional arrow between sports medicine and sport psychology.
- Although it is clear that physical injuries, illness, or disease can directly affect athletic performance and/or participation, psychological factors (broadly defined to include behavioral, emotional, or cognitive patterns; personality variables; developmental or adjustment issues; or diagnosable clinical issues) are also critically important to optimal athletic performance.
- Within the sports medicine team, a sport psychologist is involved in psychological care and consultation with athletes at all levels of competition.
- SPs use their training and experience to provide a unique contribution to the sports medicine team in dealing with injuries. They can address factors (e.g., stress) that may influence the risk of injuries as well as the psychological factors related to recovering from injury (see Appendix A). SPs can help in the management of concussion patients with prolonged/persistent symptoms, many of which are often caused by emotional or psychological or psychiatric factors.

## QUALIFICATIONS OF A SPORT PSYCHOLOGIST

**Influence of history:** Sport psychology has emerged from its roots in motor learning and kinesiology research and applications of clinical and counseling psychology principles to athletic settings. As a result, there are a range of sport psychology practitioners: academicians and researchers in exercise and sport science to applied/clinical psychologists.

**U.S. Olympic Committee (USOC):** In the early 1980s, the USOC created a Registry of SPs that was divided into three categories: educational, clinical, and research. This led to current and ongoing efforts by USOC SPs to include only those practitioners who meet the standards and certain criteria in their educational training and supervised practice in sport psychology. **USOC Registry** members are often involved in consulting with elite/Olympic athletes or teams or serve as regional referrals to athletes or teams.

**Association for Applied Sport Psychology (AASP)—Certified Consultant in Sport Psychology status:** The AASP reviews and approves individual credentials, coursework, and experience, certifying that these consultants meet the standards in their educational training in sport science and psychology; it supports the AASP Ethical Code and provides information about the role of psychological factors in sports and physical activity to individuals and organizations. Consultants can teach athletes specific mental, behavioral, psychosocial, and emotional control skills for sports and physical activity. Consultants with clinical or counseling training are qualified to work with athletes having clinical disorders or issues emerging out of injury.

**APA Division 47 (Exercise and Sport Psychology):** Involved in ongoing efforts at creating a standard for proficiency in sport psychology that "encompasses training in the development and use of psychological skills for optimal performance of athletes, in the well-being of athletes, in the systemic issues associated with sports setting and organizations, and in developmental and social aspects of sports participation" (see www.apa47.org).

**Importance of licensure: For clinical issues and/or emotional disorders in athletes, a referral to a licensed mental healthcare provider is essential; a referral to a provider who has experience in working with athletes is optimal. Other providers/practitioners may offer "sport psychology consultation," focusing on mental skills training or performance enhancement. Qualified clinical or counseling psychologists may also provide performance enhancement consultation.** The title of "psychologist" is typically one that requires licensure by states; therefore, practitioners identifying themselves as "SPs" should be licensed **and** have competency (education and training) in the field of sport and performance psychology.

## SPORT PSYCHOLOGIST ROLES

- **A psychological skills training consultant** typically educates athletes or teams about mental skills that enhance performance. These skills include goal setting, relaxation, imagery or visualization, positive self-talk, arousal regulation, increased concentration/focus, precompetitive routine/mental preparation, adaptability/handling pressure, time management, and general communication skills. Team consultation can enhance communication and cohesion among athletes.
- **A licensed clinical sport psychologist** can provide the same services that a psychological skills training consultant can, but he/she can also provide assessment and interventions to

athletes dealing with MH issues. Assessments can include the following:

- Classification or diagnosis of a presenting issue as well as identifying etiology or contributing factors
- Acquiring a specific and detailed understanding of personal and sport-specific issues or problems
- Interview, behavioral observation, psychological testing, neuropsychological testing, and completion of inventories or questionnaires
- Integration of an athlete's predisposing factors, precipitating factors, and current maintenance factors
- **A specialized role for a clinical sport psychologist** with training and experience as a neuropsychologist involves evaluation and consultation regarding sport concussion. This role involves evaluation of neurocognitive, emotional, and reported physical symptoms following a concussion. Sport neuropsychologists will use neuropsychological testing to assess cognitive status and provide inputs to team physicians and/or the sports medicine team (see Chapter 39: Head Injuries). In addition, sport neuropsychologists can evaluate, and in certain cases, treat the often overlooked emotional symptoms of concussion.

## PERFORMANCE ENHANCEMENT

- Athletes can be referred to a sport psychologist for guidance in adding, developing, and/or improving mental skills to improve performance.
- **Areas for consultation (7 Cs):**
  - **Commitment** issues dealing with motivational concerns (e.g., burnout or mental fatigue) and may involve developing goal-setting skills
  - **Control** skills are usually important for athletes who must have control over their body and movements as well as their minds; as athletes progress upward at various competitive levels, the importance of control over thoughts, emotions, and reactions becomes increasingly important. Athletes must learn to handle pressure (arousal control) and develop relaxation skills in order to find the level of arousal associated with optimal performance. Visualization or imagery skills are useful in improving control and focus.
  - **Concentration** is crucial for successful performance. This skill involves not only the initial focus but also the ability to refocus and deal with inevitable distractions. Focus on task-relevant skills is crucial for optimal performance.
  - **Confidence** is a factor that athletes (and coaches) describe as primary in successful athletic performance. Confidence is an expectation of success; it is linked to optimism, belief in self, and positive self-talk. Certain athletes focus on having confidence regarding outcomes, whereas others focus on having confidence about being able to put forth effort and concentrate on what they need to do. Slumps are examples of a loss of confidence.
  - **Communication** skills can be described as both interpersonal and intrapersonal. It is helpful to improve communication among teammates or between athletes and coaches (interpersonal) as well as between individual athletes in terms of how they communicate within themselves (intrapersonal). Moreover, development of positive and adaptive self-talk is associated with successful performance.
  - **Consistency** skills are associated with developing consistent efforts in practice and games and developing precompetitive routines and mental preparation skills. Precompetitive routines help build confidence in athletes by producing a reliable and predictable response to the behavioral sequence.
  - **Competence** skills are related to helping athletes see themselves as competent and capable competitors and people; they can identify with qualities of resilience, flexibility, and mental toughness. Self-statements such as "I can cope. I can handle it" are part of possessing competency skills. **Competency overlaps with confidence but is more reflective of self-efficacy (knowing what you need to do and when to do it and believing you can do it)**.
  - Athletes also need **courage and coping** skills, particularly when dealing with injuries.

## SPORT PSYCHIATRISTS

- Provide support to the sports medicine team and may provide medication evaluation and treatment as an adjunct to sport psychology service
- Can be helpful in managing various MH conditions (e.g., depression, anxiety, eating disorders [EDs], substance abuse, attention deficit disorder [ADHD], personality disorders, impulse control disorders, and psychosomatic illnesses).
- **The International Society for Sport Psychiatry (ISSP)** is an organization that applies the practice of psychiatry to the world of sports at all levels.

## PSYCHOLOGICAL ISSUES IN ATHLETES

### Clinical Concerns

- Clinical concerns include anxiety, depression, stress reactions, adjustment reactions, phobias, substance abuse, eating disorders, and burnout; all of these can be associated with performance decrements. NCAA student-athletes may face stressors/pressures that are unique in comparison with their nonathlete peers; scheduling demands, physical stress and fatigue, stereotyping effects, and the dual role of student and athlete.
- Athletes are not immune to MH issues. Survey data reflects that student-athletes struggle with anxiety and depression but may be less likely to report such issues than their nonathletic peers. Given this, athletic trainers, team physicians, and coaches can play important screening and supportive referral roles. General stress, interpersonal relationships, and sleep difficulties are associated with depression and anxiety.
- Psychoeducational efforts around MH or behavioral needs by the NCAA and athletic departments provide important options for student-athletes, and such efforts can decrease the stigma associated with seeking MH treatment.
- Treatment from a licensed MH professional is indicated for clinical issues; for athletes, finding a professional with sport psychology experience is preferable. If prescription medication is considered, a sport psychiatrist along with a clinical psychologist may work together and provide diagnosis, treatment goals, psychotherapy (individual or family), and/or pharmacotherapy. Treatment outcomes are optimized if there is an established referral pathway for MH issues and an interdisciplinary team approach.

### Mood Disorders

Mood disorders involve significant deviation from normal mood states with significant cognitive, behavioral, and physical symptoms that usually affect daily functioning and performance.

#### Major Depression

- **Major depression** has diagnostic criteria (*Diagnostic and Statistical Manual of Mental Disorders*, 5th ed.—DSM-5) that require 2 weeks of psychological or psychosocial symptoms that affect a person's capacity to function (Box 25.1, Fig. 25.1).
- For athletes, depressive feelings and clinical depression can emerge following injury or when recovery/rehabilitation is slower than expected or when stress and pressure are overwhelming and greater than available support resources.

**BOX 25.1 SYMPTOMS OF DEPRESSIVE DISORDER***

- Decreased mood/dysphoria
- Feelings of guilt
- Thoughts of death or suicidal ideation
- Social withdrawal
- Loss of pleasure or interest
- Feelings of worthlessness
- Low self-esteem
- Hopelessness
- Helplessness
- Indecisiveness
- Sleep disturbance (insomnia or hypersomnia)
- Change in appetite or weight
- Change in energy level or fatigue
- Attention or concentration issues
- Restlessness or psychomotor slowing

*Diagnostically, these symptoms do not emerge within the context of bereavement or grief, diagnosed medical illness, medication reaction, or substance abuse, although depression can accompany these issues.

**BOX 25.2 EXAMPLES OF MANIC SYMPTOMS**

- Abnormal or excessive elation
- Unusual irritability
- Decreased need for sleep
- Grandiose notions
- Increased talking
- Racing thoughts
- Increased sexual desire
- Greatly increased energy
- Poor judgment
- Inappropriate social behavior

### Persistent Depressive Disorder (Dysthymia)

- Diagnostic criteria:
  - At least 2 years of symptoms, with more days of depressed mood than not depressed
  - At least two of the following symptoms: hopelessness, helplessness, problems in concentration, indecisiveness, appetite loss or overeating, low energy or fatigue, low self-esteem, and sleep disturbances
- Individuals are often seen as chronically unhappy, irritable, and pessimistic.
- **Athletes with dysthymia may not self-report depression but may be ruminative, pessimistic, and not seem to be enjoying their athletic involvement (see Fig. 25.1).**

### Bipolar Disorder

- Bipolar disorder can involve both depressive episodes and manic or hypomanic episodes, and often extreme affective dysregulation. **Manic symptoms** are enlisted in Box 25.2 and **hypomanic episodes** are milder versions of manic episodes.
- Bipolar disorder may present in athletes as an extreme behavioral change (in a previously well-functioning person) or as self-destructive or violent behavior (and may involve substance use/abuse). Psychological treatment from a licensed MH professional, such as a clinical psychologist, and/or medication evaluation and management by a psychiatrist are recommended.

## Anxiety Disorders

- Anxiety disorders involve intense apprehension, general fearfulness, fear of losing control, and associated physical symptoms such as palpitations, tremors, and mental and physical avoidance efforts (worry and avoidance of people, places, or things).
- Efforts at avoidance are initiated to reduce the anxiety but often create significant life disruption.

### Panic Disorder

- Panic disorder involves periods of severe fear or discomfort with significant concomitant physical symptoms such as palpitations, dizziness, nausea, lightheadedness, fear of losing control or going crazy, numbness, or tingling. These experiences often result in emergency room visits for suspected cardiac issues; subsequently, individuals focus on panic sensations or the possibility of having a panic attack and become anxious about being anxious ("what if …") (see Fig. 25.1).
- Athletes with panic disorder often find it difficult to maintain focus or concentration because of the greater focus on anxiety/panic symptoms and sensations; in addition, they may display reduced energy and motivation. Sensations and fear of panic attacks are associated with certain conditions that generalize to an expanding set of conditions.
- Behavioral avoidance may occur as a person tries to prevent further attacks, which, in the extreme, creates only a small "safe zone" (agoraphobia).
- A vast majority of individuals with panic disorder report a life stress event as being causative (e.g., death of a significant family member, relationship breakup, job loss, illness, or injury).

### Generalized Anxiety Disorder (GAD)

- GAD usually involves long-standing anxiety symptoms, such as excessive worry, that can result in sleep disturbances, restlessness, and general muscle tension.
- Typically, individuals with GAD ruminate and worry excessively in multiple (if not all) spheres of their life (sports, school, work, health, family, or relationships).
- Vigilance to threats in the environment is thought to reduce threat or bad things from happening to the individual; the perceived success (no occurrence of bad things) reinforces the approach.
- For athletes, subclinical worry may also interfere with athletic performance, but it is less severe and interfering and does not extend to other areas.

### Social Anxiety Disorder

- Social anxiety disorder is often called "social phobia" and involves fear of evaluation in social situation.
- Individuals are concerned about and fear poor or inadequate performance and being negatively scrutinized, which can include athletic competitive situations, social interactions, public speaking, or other performance/outcome situations. This fear of negative outcome/evaluation can lead to avoidance behavior, which, in turn, reduces general functioning and reinforces negative self-perceptions.
- Several athletes may describe competitive anxiety, most often before a competition, but this does not rise to the intensity of symptoms found in social anxiety disorder; these athletes may not be able to refocus after distraction and have a greater negative response to real or perceived performance failures.
- Athletes with significant anxiety complaints and/or symptoms should undergo a thorough assessment regarding the degree and pervasiveness of their negative evaluation focus (usually a clinical interview and/or anxiety questionnaires or inventories).

### Obsessive-Compulsive Disorder (OCD)

- OCD involves the intrusion of recurrent obsessions (thoughts, ideas, images, or impulses) or compulsions into daily functioning, creating significant difficulties and anxiety; individuals engage in compulsive behaviors (often repetitive) that serve to reduce anxiety and distress.

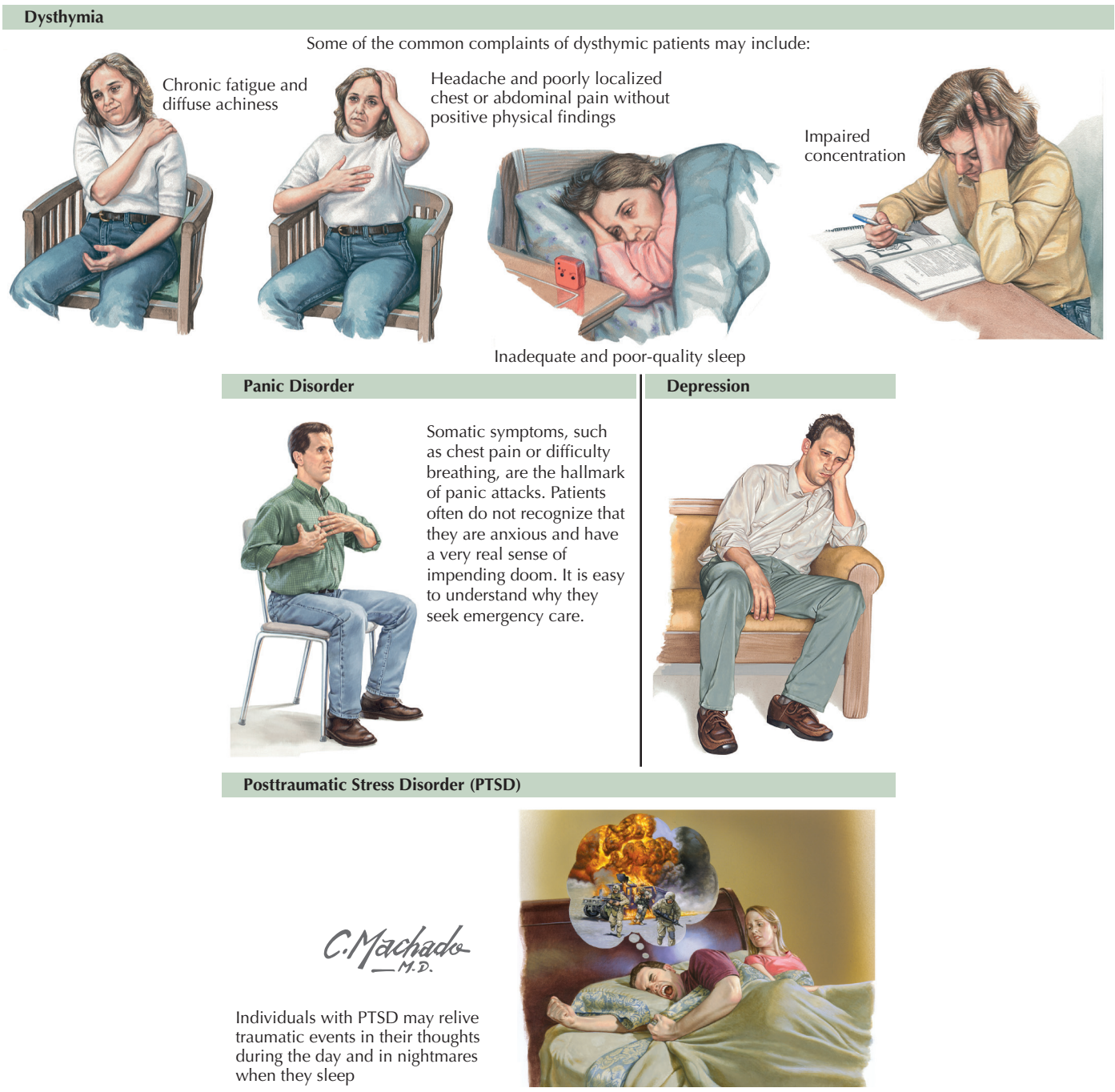


**Figure 25.1.** Common psychological disorders.

- Blockage of the compulsive behavior results in increased anxiety and decreased functioning.
- Athletes will often have precompetitive routines or rituals, which generally do not rise to the level of OCD; however, if athletes associate successful performance with only a narrow set of behaviors, it could create a negative impact on general functioning.

## Posttraumatic Stress Disorder (PTSD)

- PTSD involves a group of symptoms emerging as an intense emotional response to an event that involved actual or possible death or injury (or involved witnessing an event or learning about it occurring to a close friend or family member or being exposed to aversive details on a repeated basis) (see Fig. 25.1).
- Symptoms include re-experiencing symptoms from the event in the form of flashbacks or trigger cues, avoidance of all cues associated with the trauma and emotional detachment, physiological hyperarousal (vigilance, extreme startle response, and decreased attention and concentration). These symptom clusters often impair interpersonal relationships.

## Acute Stress Disorder (ASD)

- In certain athletes, a severe or career-ending injury may generate PTSD-like symptoms, but the symptoms may be a better

diagnostic fit for acute stress disorder (ASD), which involves exposure to a traumatic event that involves a serious injury, actual or threatened death, or threat to physical integrity of self or others.
- Diagnosis also requires three or more of the symptoms of numbness or detachment or lack of emotion, being dazed or somewhat unaware of current circumstances, derealization, depersonalization, and poor recall of the trauma.
- ASD involves recurrent thoughts/re-experiencing of the trauma and some avoidance behaviors.
- Symptoms typically emerge immediately (must emerge within 4 weeks of trauma and must last at least 2 days) and can last for up to a month.

## Eating Disorders (EDs)

- There are three diagnostic categories for EDs: avoidant/restrictive food intake disorders such as anorexia nervosa, bulimia nervosa, and binge-eating disorder (see Chapter 22: ED).
- Clinical sport psychologists with training and experience in ED may be helpful in assessment, diagnosis, and treatment of athletes with ED or disordered eating.
- Educating athletes regarding EDs can be helpful, but it is important to understand and acknowledge that athlete culture may help camouflage eating issues and/or be offering mixed messages.
- In several, if not most, cases, symptoms of depression or anxiety coexist with ED behavior (e.g., through issues of control and guilt or negative perfectionism).
- It is desirable to identify practitioners with expertise in ED so that referrals can be easily made, if indicated. A sport medical physician can be a crucial member of the multidisciplinary team that includes athletic trainer, psychiatrist, and therapist.
- The NCAA provides useful resources for team physicians, athletes, and coaches *(Managing Student Athletes' Mental Health Issues and Mind, Body, and Sport)*.

## Attention-Deficit Hyperactivity Disorder (ADHD)

- ADHD involves symptoms related to two clusters: inattention/disorganization and hyperactivity and impulsivity.
- A certain degree of impairment must occur before the age of 12 years in multiple settings (e.g., school, work, and home).
- ADHD is comorbid with other adolescent disorders (conduct or oppositional-defiant disorders), or incipient mood, anxiety, and substance abuse disorder; therefore, diagnosis of ADHD should be made after a thorough assessment.
- Common symptoms in childhood include difficulty in concentrating, difficulty in sitting still, excessive talking, impulsive behavior, and general restlessness.
- Although most individuals experience a decline in ADHD symptoms through adulthood, a significant number of adults describe ongoing problems with focus, concentration, organization, and impulsivity and/or experience problems in the workplace or within interpersonal relationships.
- **Assessment** should include a clinical interview and detailed history regarding medical, academic, and family history. Certain interview protocols include ratings of behavior from parents and teachers. Psychological and/or neuropsychological testing may provide additional data regarding the cognitive status of an individual and help in differential diagnosis.
- Attentional difficulties or problems with impulsivity, focus, and follow-through usually create problems within the competitive sports environment. However, a diagnosis of ADHD should not be made on inference alone (poor performance or mistakes that are attributed to inattention or poor concentration) or self-reported attention problems or impulsive behavior; as mentioned earlier, thorough and careful diagnostic evaluation of history and other factors should be completed before a diagnosis of ADHD is made.
- Athletes with ADHD may show inattentiveness, distractibility, or poor focus; may be mistakenly felt to have problems in motivation or attitude because of their inattentive behavior or errors in practice or games or their impulsivity or difficulty faced in following rules. Interpersonal issues may arise because frustration tolerance may be low. Such athletes may respond positively to a structured sports environment, which helps to reduce distraction. Ideally, referral to a clinical sport psychologist should be considered.
- Primary treatment options for ADHD can include behavioral interventions and medications. Psychiatrists can provide medication evaluation for athletes and monitor medication effectiveness over time along with potential negative side effects.
- Various medications used to treat ADHD, such as stimulants (e.g., Adderall), are banned by the NCAA except as medically indicated, and their use can result in a positive drug test result. NCAA rules mandate that athletes using stimulant medications for the treatment of ADHD provide annual documentation (including tests performed to make the diagnosis), medication dose, and treatment regimen by the treating physician as part of their records stating that nonstimulant medications were tried and/or were not effective. Similarly, the USOC requires a therapeutic use exemption for the use of stimulant medications.

## Anger Control and Impulse Control Issues

- Anger and impulse control issues are usually described as aspects of other disorders, such as mood or anxiety disorders or general impulse control problems (e.g., pathological gambling).
- In general, anger control issues emerge when the aggressive response (verbal or behavioral) is thought to be out of proportion relative to the nature of the event; certain individuals experience guilt or remorse following their behavior and its consequences.
- Anger dyscontrol may be associated with aggressive behavior, history of family violence, substance abuse, and certain medical conditions.
- Athletes may display issues with frustration and anger control, with a range of frequency observed. If such behavioral issues are occurring at a significant frequency, it is imperative to evaluate whether this is part of a larger dyscontrol pattern. It is crucial that the sport psychologist or other care provider take time to distinguish between competitive intensity/anger and anger control issues.
- In certain instances, the athletic environment rewards aggressiveness (usually seen as controlled aggression) because it may be associated with athletic success. Certain athletes, who may be predisposed to anger, may respond well to such rewards but have difficulty with controlling or compartmentalizing their impulses to appropriate levels or venues (i.e., they exhibit such behaviors in nonathletic circumstances).

## Personality Disorders

- Personality disorders involve personality or behavioral traits that endure over time and are maladaptive, causing significant subjective distress and/or observable functional impairment in major areas of life (school, work, or family) (see Fig. 25.1).
- Individuals are not usually diagnosed with personality disorder until the age of 18 years.
- Disorders are grouped in three clusters:
  - **Cluster A** includes schizoid, paranoid, and schizotypal personality disorders, which are characterized by atypical

interpersonal behavior, odd interaction styles, and perceptions of the world.
  - **Cluster B** includes histrionic, borderline, narcissistic, and antisocial personality disorders, which are characterized by affective and behavioral deregulation.
  - **Cluster C** includes avoidant, obsessive-compulsive, and dependent personality disorders, which are characterized by symptoms of anxiety and avoidant behavior.
- These disorders or their subclinical variants (traits or features that are present but do not meet diagnostic criteria) may be seen in athletes who have repeated interpersonal difficulties with teammates or staff, problems with authority or complying with rules, disciplinary issues, aggression or emotional dyscontrol, or issues with accepting responsibilities. Comorbid issues include substance abuse, anger, aggression, and mood issues.
- Clinical sport psychologists can be helpful in assessment of such disorders or general traits involved. Cognitive behavioral approaches have been helpful in reducing symptoms and enhancing functional levels; such chronic disorders may fluctuate, but their complexity usually suggests long-term therapeutic interventions.

## PSYCHOLOGICAL ASPECTS OF INJURY

- Injury can trigger psychological responses, and the sports medicine team should be aware of the cognitive, emotional, and behavioral responses to being injured as well as the recovery/rehabilitation process.
- **There may be certain stages of physical recovery**; however, there are also psychological stages of coping with injury, which are individualized and influenced by other factors (e.g., social support, impact on team or career, or general personality traits).
- **The sports medicine team should consider referral for psychological consultation because it can enhance physical recovery**.
- Psychological components of injury and recovery can include shock and emotional disorganization immediately after injury; general anxiety and uncertainty regarding the future during evaluation and treatment decision-making; and impatience, anger, frustration, lack of control, and depression during recovery.
- Psychological issues can develop during the return-to-play phase and may involve the fear of reinjury or of not being able to perform at the preinjury level. In addition, self-doubt and rumination can emerge, producing a larger issue of decreased confidence.
- Moreover, psychological factors such as stressful life events may contribute to the risk of athletic injuries. Personality factors have not been found to reliably relate to the risk of athletic injury, but athletes with high levels of stress, low or ineffective coping skills, and low social support may be at a particular risk of injury; such athletes may also be at a risk of a challenging injury response and recovery course.
- Athletes make efforts to deal with being injured in individualized ways and time frames. Emotional responses can include sadness and feelings of isolation, irritation, frustration, and anger as well as changes in sleep, appetite, and energy (fatigue). These symptoms can worsen and emerge as issues during the recovery/rehabilitation process. Depression is the most common response: it can progress from sadness to a more clinical level of depression.
- Of note, athletes often use their training/exercise and general physical activity as a coping mechanism and outlet for dealing with stress; thus, an injury can reduce access to this coping strategy, which, in turn, adds to the emotional stress.
- Signs of poor adjustment to injury include exaggerated or generalized fear of reinjury, general impatience or irritability, mood swings, withdrawal from significant others or social network, rumination about guilt for being injured and letting others down, increased somatic focus, obsession with return to play, general pessimism about the future in sports and other areas (demoralization), or apathy.
- Watch for athletes sliding (or falling) down the "D Slide:" this involves athletes feeling disappointment about the injury or being injured; this is a normal-range experience and involves the thought that "I can't do it and I should be able to." If disappointment is not appropriately dealt with, it can progress to feeling devastated, which involves the generalized thought "I can't do anything." If devastation is not addressed, the slide continues to feeling defeated ("I'm helpless to do anything [to change it]"). The low point on the slide is when an athlete feels defective and thinks, "There's something wrong with me."
- Injured athletes who are strongly identified with their sport may experience a greater sense of identity loss. Separation and loneliness (from the team) are experiences that can increase the probability of problematic emotional responses to injury. Maintaining contact with teammates reduces the sense of isolation and gives the athlete opportunities for support.
- Return-to-play issues include physical as well as psychological readiness and clearance. Assessment of psychological readiness is crucial to successful return to play and should be considered by the athletic trainer, team physician, team psychologist (if available), and other members of the athlete's support network.
- For further discussion of the importance of psychological issues related to injury in athletes, please refer to the ACSM TPCS (see Recommended Readings).

## SUMMARY

- Sport psychologists provide a range of educative and consultative services to individual athletes and teams for various psychological health issues; in addition, they can provide psychological skill training related to performance enhancement and team building.
- Inclusion of a clinical sport psychologist on the sports medicine team is thought to provide an excellent opportunity to address psychological aspects of performance, adjustment, and injuries.
- If a clinical sport psychologist is not a part of the sports medicine team, physicians should use sport psychology (or sport psychiatry) referrals to appropriately trained (and licensed) providers in the community.
- Sport psychology can make a particularly useful contribution to the team physician and sports medicine team in dealing with injured athletes and providing support related to mental health or psychological concerns.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# DRUGS AND DOPING IN ATHLETES

# 26

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## DEFINITION

- According to the World Anti-Doping Agency (WADA) Code, doping is defined as the occurrence of one or more of the following antidoping rule violations:
  - Presence of a prohibited substance or its metabolites or markers in an athlete's bodily specimen
  - Use or attempted use of a prohibited substance or prohibited method
  - Refusing, or failing without compelling justification, to submit to sample collection after notification, as authorized in applicable antidoping rules, or otherwise evading sample collection
  - Violation of applicable requirements regarding athlete availability for out-of-competition testing, including failure to provide required information on whereabouts and missing tests that are declared based on reasonable rules
  - Tampering, or attempting to tamper, with any part of doping control
  - Possession or use of prohibited substances and methods
  - Trafficking of any prohibited substance or method
  - Administration or attempted administration of a prohibited substance or method to any athlete or assisting, encouraging, aiding, abetting, covering up, or any other type of complicity involving an antidoping rule violation or any attempted violation

## SCOPE OF THE PROBLEM

- 1964: Drug testing began at the Olympics
  - Substance or method shall be considered for inclusion on the prohibited list if the WADA determines that a substance meets any two of the following three criteria (Box 26.1):
    - Medical or other scientific evidence, pharmacologic effect, or experience that a substance or method has potential to enhance or enhances sports performance
    - Medical or other scientific evidence, pharmacologic effect, or experience that use of a substance or method represents actual or potential health risk to the athlete
    - The WADA's determination that use of a substance or method violates the spirit of the sport
  - A substance or method shall also be included on the prohibited list if the WADA determines that there is medical or other scientific evidence, pharmacologic effect, or experience that the substance or method has the potential to mask use of other prohibited substances and prohibited methods.
- 1968: Formal drug testing adopted for the Summer and Winter Olympic Games; drug testing has been used at every Olympiad thereafter.
- 1985: The NCAA began a series of quadrennial surveys that documented substance abuse, use, and abuse patterns by intercollegiate athletes. The methodology changed significantly in 1997, and the number of participating schools and subjects increased. The eighth iteration in 2012 measured substance-use patterns in 20,474 male and female athletes (Table 26.1). Periodic surveys are necessary to assess patterns of use of recreational and ergogenic substances.
- Following a series of doping scandals, the International Olympic Committee convened the World Conference on Doping and Sport in February 1999 that led to creation of the WADA. The WADA was charged with developing standards and a consistent, worldwide doping-control program. In addition, each country founded its own antidoping agency (e.g., United States Anti-Doping Agency [USADA]) to ensure compliance. The WADA is composed of and equally funded by sports movement and governments of the world. Fig. 26.1 demonstrates the numerous worldwide tests conducted by WADA-accredited laboratories.
- 2003: Investigation into Bay Area Lab Co-Operative (BALCO) revealed a sophisticated conspiracy involving professional athletes as well as US and international Olympic athletes. In addition to developing designer anabolic steroids, BALCO records were used to discipline athletes based on "nonanalytical" positives, i.e., athletes were suspended based on doping records rather than an adverse analytical finding during drug tests. Use of nonanalytical positives and forensic investigations has increased since 2003 as another antidoping tool.
- Sports Pharmacology
  - Classifies drugs according to their reason for use rather than chemical structure, mechanism of action, or pharmacologic effects—a drug is classified as ergogenic, recreational, or therapeutic depending on the primary reason for use, which can influence prevention, identification, and treatment
    - Ergogenic drugs are substances consumed specifically to increase performance.
    - Nonperformance ("recreational") use of drugs by athletes for same reasons as nonathletes and carries the risk of addiction
    - Therapeutic drugs are taken to treat an underlying condition.
- The Physician's Role
  - The relationship between physicians and drugs in sports is often complicated and has a long historical legacy.
    - Since the ancient Greek Olympics, athletes have sought out physicians to provide performance aids.
    - 2012 NCAA Study: 20.9% of anabolic steroid users named an athletic staff member as a source of drugs.
    - Physicians play a central role in use and abuse of drugs by athletes.
      - Physicians are asked to provide education, act as medical directors or medical review officers for drug testing, and provide therapeutic use exemptions (TUEs). It is imperative that physicians who serve as team physicians are familiar with performance-enhancing drugs (PEDs) used by athletes and also with procedures used to determine positive test results.
      - Important to remember that a majority of high-profile doping scandals could not have been possible without active participation of physicians
- Role of National Olympic Committees (NOCs) and National Governing Bodies (NGBs)
  - Historical evidence of systematic doping of athletes sanctioned by either NOCs or NGBs exists.
    - East German swimmers admitted to systematic doping in the 1970s that was sanctioned by their NOC.
    - Discovery of widespread use of blood doping by US cyclists participating in the 1984 Summer Olympic Games

**BOX 26.1 WADA BANNED SUBSTANCE LIST**

**Anabolic Agents**

- Anabolic Androgenic Steroids (AAS)
  - Exogenous
  - Endogenous
- Other Anabolic Agents (e.g., clenbuterol, zeranol, and zilpaterol)

**Hormones and Related Substances**

- Erythropoietin (EPO)
- Growth Hormone (hGH), Insulin-like Growth Factors (e.g., IGF-1), Mechano Growth Factors (MGFs)
- Gonadotrophins (LH, hCG) (prohibited in males only)
- Insulin
- Corticotrophins

**Beta$_2$ Agonists**

**Agents With Antiestrogenic Activity**

- Aromatase inhibitors
- Estrogen receptor modulators
- Other antiestrogenic substances

**Diuretics and Other Masking Agents**

**Stimulants**
**Narcotics**
**Cannabinoids**
**Glucocorticosteroids**

*WADA*, World Anti-Doping Agency.

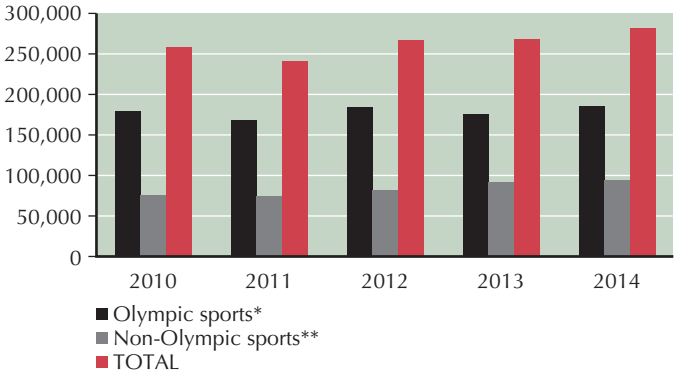


**Figure 26.1.** Number of worldwide tests conducted by WADA-accredited laboratories. (Data from Laboratory Samples Analyzed and Reported by Accredited Laboratories in Olympic and Non-Olympic Sports. World Anti-Doping Agency. 2014 Anti-Doping Testing Figures Report.)

- Recent evidence that systematic doping took place under aegis of the Russian Sports Federation uncovered in a recent WADA investigation

## ANABOLIC-ANDROGENIC STEROIDS (AAS)

**Definition:** Testosterone or testosterone-like synthetic drugs that result in both anabolic and androgenic effects, e.g., increase protein synthesis (anabolism) and enhance the development of male secondary sexual characteristics (androgenic)

**Prevalence:**

- Since 1988, surveys have sought to assess usage rates in athletes and nonathletes with conflicting results; however, it is clear that use of AAS is no longer confined to athletes participating in organized sports but is also prevalent in the general population for nonathletic enhancement.

**TABLE 26.1 DRUG USE BY INTERCOLLEGIATE ATHLETES IN PAST 12 MONTHS**

| | *Percentage (%) of Athletes Using* | |
|---|---|---|
| **Drug** | **2005** | **2012** |
| Anabolic steroids | 1.2 | 0.4 |
| Spit tobacco | 16.3 | 17.4 |
| Alcohol | 76.9 | 83.2 |
| Ephedrine | 2.5 | 0.9 |
| Amphetamines | 4.1 | 3.7 |
| Marijuana | 20.3 | 22.6 |
| Cocaine | 2.1 | 1.8 |

*Data from the NCAA National Study of Substance Use Habits of College Student-Athletes Final Report. July 2014.* http://www.ncaa.org/sites/default/files/Substance%20Use%20Final%20Report_FINAL.pdf. Accessed November 16, 2015.

**TABLE 26.2 COLLEGE ATHLETES' AGE AT THE FIRST TIME OF USE OF ANABOLIC STEROIDS**

| **Response Category** | **Percentage (%) 2012** |
|---|---|
| Before age 14 | 17 |
| Ages 14–15 | 8 |
| Ages 16–17 | 25 |
| Ages 18–20 | 42 |
| Ages 21+ | 8 |

Data from 2012 National Study of Substance Use Trends Among NCAA College Student-Athletes.

**TABLE 26.3 PRIMARY REASONS FOR COLLEGE STUDENT-ATHLETES FOR USE OF ANABOLIC STEROIDS**

| **Reason** | **Percentage (%)** |
|---|---|
| To improve athletic performance | 47 |
| For sports-related injury | 28 |
| For non-sports-related injury or illness | 23 |
| To prevent injury | 2 |

- 2014: Monitoring the Future study reported that 1.5% of 12$^{th}$ graders used AAS in the previous 12 months. Since 1989, this rate has ranged between 1% and 2.5%.
- 2012: An NCAA survey revealed that 0.4% (declined from 1.2% in 2005) of male and female athletes had used AAS in the past 12 months. Additional data from NCAA surveys are presented in Tables 26.2 and 26.3.
- The androgenization program developed by the former German Democratic Republic provided anecdotal information supporting the efficacy of AAS as well as its adverse effects.
- Current estimates indicate there are as many as 3 million AAS users in the US and that 2.7%–2.9% of young American adults have taken AAS at least once in their lives. A 2014 global epidemiologic study highlighted the widespread AAS

public health issue when it reported a global lifetime prevalence rate of 3.3% (6.4% in males and 1.6% in females).
- Recent estimates are that 1.5 million teenagers have tried AAS and that teenage girls may be the fastest-growing demographic for use of AAS.

**Mechanism of action:**
- AAS are bound by cytoplasmic proteins and transported to the nucleus. Activation of DNA-dependent RNA polymerase results in the production of messenger RNA for protein synthesis.
- Muscle size increases in users of AAS through hypertrophy and formation of new muscle fibers. According to a study conducted by Kadi et al., muscle biopsies suggested that use of AAS enhances activation of satellite cells and contributes to muscle fiber growth.
- In addition, AAS may have anticatabolic effects by attenuating effects of cortisol. Haupt proposed that AAS displace cortisol from receptors, allowing an athlete to train at a higher level; he also suggested that AAS increase motivation through heightened aggressiveness.

**In vivo studies of athletes and anabolic steroids:**
- Numerous studies of anabolic steroid use by male athletes have produced conflicting results. Certain studies have supported improvement in strength, whereas others have found no significant improvement in strength. The American College of Sports Medicine's official position statement as well as many other systematic reviews support the following position on AAS:
  - Use of steroids by athletes is contrary to rules and ethical principles of athletic competition.
  - With adequate diet, AAS can contribute to increase in body weight and lean mass.
  - Gains in muscular strength achieved through steroid use at doses beyond those utilized in clinical medicine improve performance and seem to increase aerobic power or capacity for muscular exercise, giving an unfair advantage to those who are willing to risk potential side effects to achieve gains in athletic performance.
  - Steroids have been associated with adverse side effects in therapeutic trials and in limited studies on athletes.
- A study conducted by Bhasin et al. in 1996 demonstrated an increase in fat-free mass and muscle size and strength in weight lifters who used weekly injections of 600 mg testosterone enanthate for 10 weeks. This was the first study to demonstrate that supraphysiologic doses of testosterone in trained weight lifters, combined with resistance training, increases strength.

**Designer steroids:** Chemical alternations are made to existing synthetic AAS in order to avoid detection by drug testing.
- Norbolethone
  - Originally documented 30 years ago; isolated in two urine samples from a female athlete in 2002 who later received a lifetime ban
  - Never commercially marketed because of possible toxic effects in animal studies and/or reports of menstrual irregularities
  - Anabolic activity 20 times higher than its androgenic activity, thus very attractive to athletes looking to gain a competitive edge
- Tetrahydrogestrinone (THG)
  - Unlike norbolethone, THG is a new chemical entity and would have remained undetectable if a syringe containing it had not been anonymously sent to the USADA in 2003.
  - Closely related to gestrinone, a progestin, and to trenbolone, a veterinary androgen
  - Limited information regarding the safety and anabolic effects of THG; has been found to be a potent androgen
  - Several Olympics laboratories have reported positive results for THG.
- Several other designer AAS have appeared as increasing sophistication of drug testing continues.

**Over-the-counter steroids:**
- The 1994 Dietary Supplement and Health Act made supplements available over the counter, including testosterone (androstenedione, androstenediol, dehydroepiandrosterone [DHEA] and nandrolone precursors [19-norandrostenedione, 19-norandrostenediol]).
- Athletes use these short-acting compounds to increase muscle mass despite lack of definitive studies on the benefits and adverse effects.
- Some of these have been found to increase testosterone and nandrolone metabolites; assumed to have similar profile to AAS

**Governmental regulations:**
- 1990 Anabolic Steroid Control Act: AAS were added to Schedule III of the Controlled Substances Act.
- 2004 Anabolic Steroid Control Act: added steroid precursors such as androstenedione to the list of controlled substances; DHEA was not included in the Act and is still legally available as a dietary supplement despite being banned by several sports organizations; also increased penalties for trafficking
- Designer Anabolic Steroid Control Act of 2014: further defined designer AAS and increased penalties

**Potential therapeutic uses**: True medical indications for AAS probably account for <3 million prescriptions/year. **Indications** include refractory anemias, hereditary angioedema, palliation therapy in advanced breast carcinoma, replacement therapy in hypogonadal males, and muscle-wasting states associated with HIV infection. Moreover, AAS may be useful in patients with constitutional delay of growth (as adjunct to growth hormone [GH] therapy) and osteoporosis and has potential use as a male contraceptive. A major marketing effort by pharmaceutical companies and "antiaging" clinics has increased the number of prescriptions for testosterone amid liberalization of diagnosis of hypogonadism. The Endocrine Society published guidelines for legitimate prescription of testosterone in men with androgen deficiency. Recent position statement published by American Association of Clinical Endocrinologists and American College of Endocrinology reinforces these principles.

**Dosage: Doses taken by athletes may be 10–40-times higher than the therapeutic dose**. Athletes frequently use combinations of anabolic steroids (**stacking**) or cycling in a pyramidal fashion to achieve maximum effect.

**Adverse reactions:**
- **Gastrointestinal:** hepatocellular dysfunction, peliosis hepatis, and case reports of hepatocellular carcinoma. Hepatic effects are more severe when 17-alpha-alkylated compounds are consumed orally. Serum liver function tests should be performed in athletes with suspected history of AAS use.
- **Cardiovascular:** increase in total cholesterol and low-density lipoprotein (LDL) cholesterol; decrease in high-density lipoprotein (HDL) cholesterol; hypertension; thrombotic risks; compartment syndromes; reported cases of myocardial infarction, atrial fibrillation and cerebrovascular accident

  The Food and Drug Administration (FDA) has released a warning to reflect potentially increased risks of myocardial infarction and stroke with testosterone use. However, while retrospective reports have suggested that testosterone therapy increases cardiovascular risk, randomized controlled trials have been insufficiently powered to evaluate the risk of cardiovascular events in men who have undergone testosterone replacement therapy.
- **Psychological effects**: changes in libido, mood swings, aggressive behavior, exacerbation of underlying mental

illness, addiction to the appearance when on AAS, and suicide attempts; dependence pattern with opioid medications as well as conduct disorder has been reported. Pope and Katz interviewed 41 bodybuilders and football players who had used AAS and found that according to DSM III-R criteria, nine (22%) athletes displayed full affective syndrome and five (12%) exhibited psychotic symptoms in association with AAS use. However, a well-designed study by Bahrke et al. of current AAS users, previous users, and nonusers demonstrated that although perceived or actual psychological changes may occur, they were not demonstrated on several standardized inventories.

- **Male reproductive effects**: oligospermia, azoospermia, decreased testicular size, and gynecomastia; case of adenocarcinoma of prostate has also been reported. In addition, a case of a young male using high doses of oral turinabol suggested a potential causal relationship to intratesticular leiomyosarcoma.
- **Female reproductive effects:** reduced luteinizing hormone (LH), follicle-stimulating hormone (FSH), estrogen, and progesterone; menstrual irregularities; male-pattern alopecia; hirsutism; clitoromegaly; and deepening of voice: the latter three are probably irreversible
- **Youths:** irreversible and premature closure of the epiphyses; several highly publicized cases of suicide in high school athletes related to steroid use (e.g., Taylor Hooton)
- **Additional drug use:** AAS users are likely to use other drugs. A 2007 study conducted by Elliot evaluating high school female students revealed that AAS users were more likely to use alcohol, cigarettes, marijuana, and cocaine. In 2009, Ip conducted the Anabolic 500 survey demonstrating that AAS-dependent users were significantly more likely to report heroin use within past 12 months and endorse a history of physical and sexual abuse.

**Miscellaneous:**

- Spontaneous tendon rupture
- Increase in sebaceous glands and acne
- Infectious complications, including HIV (due to sharing of contaminated needles), hepatitis B and C, and intramuscular abscess
- A case report described bilateral deltoid myositis ossificans in a patient who repeatedly injected anabolic steroids in his bilateral deltoids in the past.
- Worsening of tic symptoms in patients with Tourette's syndrome
- Suppression of humoral immunity and immunoglobulin levels
- Anecdotal reports of an association with compartment syndromes following trauma and/or surgical procedures

**Side effects:** documented in a secret AAS program for elite athletes sponsored by German Democratic Republic: liver damage, gynecomastia, polycystic ovarian syndrome, arrested body growth, and three deaths

**Prevention:** programs designed to reduce AAS use have been developed (e.g., by Goldberg and the ATLAS program for use at the high school level)

**Detection:** The presence of aforementioned adverse effects should arouse clinical suspicion. Drug testing (discussed later) can detect AAS with a high degree of accuracy.

## GROWTH FACTORS

### Human Growth Hormone (hGH)

**Definition:** polypeptide hormone composed of 191 amino acids with a molecular weight of 21,500 and contains several different isoforms, the predominant ones being a 22-kD isomer and approximately 10% 20 kD; typically, 5–10 mg stored in the anterior pituitary

Men have a production rate of 0.4–1.0 mg/day. Production of recombinant human growth hormone (rhGH) in 1980s dramatically increased potential supply. As opposed to natural GH, rhGH contains only the 22-kD isomer.

**Prevalence:** With growing effectiveness of gas chromatography and mass spectrometry in detecting AAS and testosterone, numerous athletes have turned to hGH; estimating prevalence is difficult but thought to be widely used by various professional athletes.

**Mechanism:** hGH stimulates the production of various markers, most prominent being insulin-like growth factor (IGF-1) or somatomedin-C. Although there is some debate regarding whether substances such as liver-produced IGF-1 are markers or mediators, hGH exerts most of its effects through receptors at target cells. There is limited evidence that commercially available IGF-1 products have any ability to increase IGF-1 levels or strength; it is also clear that while certain controlled, albeit limited studies, on hGH have revealed increases in IGF-1 and changes in lean body mass, none have definitively demonstrated increases in strength or athletic performance with hGH alone.

- **Function:** Administration of hGH to GH-deficient children results in positive nitrogen balance and stimulation of skeletal and soft tissue growth.
- **Metabolic effects:** GH reduces glucose and protein metabolism and has a net anti-insulin effect by inhibiting cellular uptake of glucose. In addition, it stimulates mobilization of lipids from adipose tissue and greatly increases protein synthesis in hypophysectomized animals.
- **Effects on muscle:** Several studies have reported conflicting data regarding effects of hGH on muscle. Goldberg conducted animal experiments and concluded that hGH increased the basal metabolic rate of protein synthesis; however, the effect was determined by the amount of muscular work. It is difficult to predict the ability of hGH in increasing contractile elements and improving performance of normal muscle in normal humans. hGH studies conducted by Deyssig revealed that while there may be increases in IGF-1 and changes in lean body mass, there is no definitive demonstration of increases in strength or athletic performance. Studies have demonstrated increases in muscle mass in settings of current or past use of AAS.
- According to a previous study, hGH treatment in adults with acquired GH deficiency increased lean body mass, decreased fat mass, and increased basal metabolic rate, inferring that hGH can regulate body composition through anabolic and lipolytic actions.

**Therapeutic uses: The FDA, under USC 33(e), has established guidelines for the legitimate use of hGH; it cannot be used "off-label" (Table 26.4).** The FDA has established the use of the GH stimulation test to diagnose adult GH deficiency. **Low serum IGF-1 levels alone are not considered evidence of GH deficiency.**

**Dosage:** FDA-approved dosage is 0.003–0.004 mg/kg/day via subcutaneous injection, although other governing bodies recommend 0.15–0.3 mg/day regardless of body weight.

**Adverse reactions:**

- **Acromegaly** is a potentially serious side effect of megadoses of hGH. It is estimated that acromegalic patients with hGH concentrations of 5–30 ng/mL have production rates of 1.5–9 mg/day. **As little as a two-fold increase in recommended dose may result in acromegaly, which leaves a narrow therapeutic window**. With athletes consuming hGH up to 20 mg/day, the risk of acromegaly is significant. Complications of acromegaly include diabetes, arthritis, myopathies, and characteristic coarsening of the bones of face, hands, and feet.
- In general, the incidence of side effects with the use of physiologic replacement doses in GH-deficient patients is

**TABLE 26.4 FDA-APPROVED INDICATIONS OF HUMAN GROWTH HORMONE**

| | |
|---|---|
| **Children, Poor Growth Due to** | Turner's syndrome<br>Prader–Willi syndrome<br>Chronic renal insufficiency<br>hGH Insufficiency/deficiency<br>Children born small for gestational age who fail to manifest/catch-up growth by 2 years of age<br>Idiopathic Short Stature or growth failure associated with short stature homeobox gene (SHOX) deficiency<br>Noonan syndrome |
| **Adults** | Wasting syndrome of HIV/AIDS<br>hGH deficiency<br>Short bowel syndrome |

Data from: Department of Justice Drug Enforcement Administration Office of Diversion Control: "Human Growth Hormone."

low. Reported adverse reactions include intracranial hypertension, hyperglycemia, and glycosuria. However, in 2012, the FDA reported that a long-term epidemiologic French study, SAGhE, demonstrated a 30% increased risk of death when using rhGH. Data suggested mortality was associated with bone tumors and cardiovascular events (primarily subarachnoid or intracerebral hemorrhage). The risk was higher when higher than normal doses were prescribed. The FDA maintained that when appropriately used, benefits outweigh risks.

- Adult patients using hGH have reported fluid retention, arthralgia, myalgia, gynecomastia, hypoesthesia, and paresthesia (e.g., carpal tunnel syndrome via median nerve edema).
- **Use of hGH by "antiaging" clinics without establishing GH deficiency constitutes illegal prescribing**.
- Use of hGH derived from cadaveric pituitary glands can result in Creutzfeldt–Jakob disease. Although use of synthetic hGH obviates this issue, athletes often obtain substances from black-market sources, which thus increases the risk of this catastrophic neurologic disorder.

**Detection**: Banned by the WADA with two established serum methods of detection

- **Isoform Test**: Immunoassays used to estimate the amounts of various GH isomers in serum. Although a majority of circulating GH is 20 and 22 kD, several other isomers naturally exist. When rhGH (22 kD) is administered, natural production of all GH isomers is suppressed; thus, the ratio of 22- to non-22-kD isomers will increase, indicating use of synthetic GH. The isoform test can detect the use of hGH for up to 72 hours, and there have been several positive tests worldwide. Recent research has established decision limits for this test, which has been upheld by the Court of Arbitration for Sport (CAS). The current detection threshold is 1.45 for males and 1.8 for females.
- **Biomarker Test**: It measures the serum markers IGF-1 (from liver) and procollagen type III (P-III-NP from bone) that can discriminate hGH users from nonusers up to 2 weeks after use. The test was first used at the 2012 London Olympic Games and revealed positive tests in two Russian Paralympian weight lifters who admitted use. This test should be available worldwide in 2016.

### IGF-1

- hGH stimulates the production of IGF-1 by the liver.
- Limited evidence that commercially available IGF-1 products have any ability to increase IGF-1 levels or strength
- Limited controlled studies on rhGH have revealed increases in IGF-1 and changes in lean body mass, but none has definitively demonstrated increases in strength or athletic performance.
- Supplements containing deer antler velvet extract have been advertised to contain IGF-1. Although these products contain IGF-1, research has demonstrated that human IGF-1 was added after production, and thus, it is illegal.
- Adverse effects related to IGF-1 use include hypoglycemia, decreased hGH secretion, carbohydrate oxidation preferentially over lipids, interference in insulin–glucagon axis, and an association with carcinoma of prostate, colon, and lung.
- Mechano growth factors are a new form of IGF-1 produced in response to mechanical loads and have been described by Goldspink.

### Growth Hormone-Releasing Peptides (GHRPs)

- Synthetic peptides with a small fraction of GH that are potent stimuli for GH secretion
- GH peaks in 15–30 minutes and returns to baseline in 2–3 hours.
- Ghrelin analogs GHRP 1,2,4,5,6, and Ipamorelin
- Oral, intravenous, and nasal routes with metabolites detectable for 20 hours by liquid chromatography–mass spectrometry (LC-MS).
- Multiple positives in professional and Olympic sports
- Has been detected in nutritional supplements; not legally available but can be purchased on the Internet for "research purposes"

### Insulin

- Physicians are trained to view insulin as a solely therapeutic drug, but increasing anecdotal information associates AAS use with insulin; a study by Parkinson and Evans revealed that 25% of AAS users also used insulin.
- Speculated that insulin might enhance strength through its inhibitory functions (deters lipolysis, glycolysis, gluconeogenesis, proteolysis, and ketogenesis); when combined with an anabolic agent such as hGH or AAS, the protein-sparing effects of insulin produce larger anabolic results and significantly increase lean body mass
- Risks of insulin use in normoglycemic athletes include lethal hypoglycemia, lipodystrophy, lipoatrophy, insulin allergy/resistance, and production of insulin autoantibodies.
- Insulin prohibited by the WADA, but no approved test for detection

## AMPHETAMINES

**Definition:** classified as indirectly acting sympathomimetic amines with central and peripheral effects

Amphetamine use in athletes is complicated by the fact that they can be used for ergogenic, recreational, or therapeutic uses.

**Prevalence:**

- A 2012 NCAA study reported 3.7% use of amphetamines in past 12 months, a decrease of 0.5% since 2005, and first such decrease in use of amphetamines as ergogenic aids since 1993. In addition, use of methylene-n-methylamphetamine (Ecstasy or Molly) as a recreational stimulant has caused concern.
- Commonly found on the black market ("greenies") and often found to contain clobenzorex
- **The 2012 NCAA study revealed that 16% of NCAA athletes had used ADHD medications, but 9% reported using those medications without a prescription**.

**Mechanism of action:** Several theories have been proposed to explain central and peripheral effects of amphetamines: increased liberation of endogenous catecholamines, displacement of bound catecholamines, inhibition of monoamine oxidase, interference with catecholamine reuptake, and production of false neurotransmitters; all of these probably contribute to the observed physiologic responses, including increase in blood pressure and heart rate, bronchodilation, increased metabolic rate, and increased free fatty acid production.

**Relationship with athletic performance:** Conflicting data in literature

- Smith and Beecher reported 75% of trained swimmers, weight throwers, and runners experienced improved performance after taking amphetamines.
- Chandler and Blair demonstrated no substantial improvement in athletic performance.
- Explanation may be that amphetamine enhances performance of simple repetitive tasks but not of more complicated maneuvers.

**Therapeutic uses:** used legitimately to treat several conditions, including refractory obesity, narcolepsy, ADHD, and severe depression. However, high abuse potential has limited its utility in such conditions. Incidence of ADHD in young adult male population is difficult to accurately determine but has been estimated at 4%–5%.

**Dosage:** Taken orally, amphetamines exert effects within 30 minutes of ingestion, and actions can last for 12–24 hours. Dosages vary depending on individual athlete and type of preparation.

**Adverse reactions:**

- **Central nervous system**: restlessness, insomnia, psychological addiction, psychosis, tremor, anxiety, dizziness, headache, and cerebral hemorrhage
- **Cardiovascular**: lowered threshold for arrhythmias and provocation of angina
- **Miscellaneous:** disruptions in thermoregulation and predisposition to heat illness

**Detection:** readily detected by urine tests because both unchanged amphetamines and metabolites appear in urine

## COCAINE

See eAppendix 26-1 on **www.ExpertConsult.com**.

## CAFFEINE

**Definition:** naturally occurring plant alkaloid derived from aqueous extracts of *Coffea arabica* and *Cola acuminata*; classified as central nervous system stimulant and found in coffee, tea, and cola drinks; is a methylxanthine and chemically related to theobromine and theophylline; used by athletes for its stimulant properties and potential to increase work and power

**Prevalence:**

- 60% of Americans aged over 18 years drink coffee daily and average three cups per day.
- Energy drinks for the US market have grown by 56% from 2009 to 2014. Alcohol intake appears significantly higher when energy drinks are mixed with alcohol.

**Mechanism of action:** Rapidly absorbed, and peak levels in 30–60 minutes with a half-life of 3.5 hours; competitive antagonist of adenosine and causes vasoconstriction (except in renal afferent artery), increased diuresis and natriuresis, central nervous system stimulation, increased lipolysis in adipocytes, and increased gastric secretion

**Dietary supplements may contain high levels of caffeine. Anhydrous caffeine, a highly concentrated form of caffeine, has been associated with several deaths. Moreover, companies have marketed oral pouches containing caffeine to use as substitutes for spit tobacco.**

**Performance:** Increased work and power probably result from increased mobilization of free fatty acids, increased rate of lipid metabolism, and direct effects on muscle contraction. Overall, the ability of caffeine to enhance or prolong work output has been controversial. Any benefits in athletic performance are likely limited to endurance activities alone.

**Therapeutic uses:** Caffeine has been used as a stimulant in fatigue states, in combination with analgesic compounds, and in diet pills.

**Adverse reactions:**

- **Central nervous system:** anxiety, hypochondriasis, insomnia, headache, tremors, depression, scotomata, and addiction with withdrawal states
- **Cardiovascular:** tachyarrhythmias, particularly paroxysmal atrial tachycardia
- **Renal:** diuretic effect, which is of significance in athletes who are at a risk of dehydration

**Detection and dosage:** The WADA no longer bans caffeine but maintains monitoring. The NCAA has set a maximum urinary concentration of caffeine at 15 μg/mL. A study found that to exceed that threshold, an athlete needs to consume almost 1000 mg of caffeine within 3 hours of testing. This is above the average daily consumption of 200 mg of caffeine. Athletes concerned about testing should be aware that caffeine excretion is variable and can be affected by several factors, including exercise and the concomitant use of SSRIs (e.g., fluoxetine) and cimetidine.

## SYMPATHOMIMETIC AMINES

**Definition:** Synthetic congeners of naturally occurring catecholamines

In addition to amphetamines, several other weaker sympathomimetic amines have potential to be abused by athletes, including phenylpropanolamine, phenylephrine, ephedrine, and pseudoephedrine.

**Prevalence:** Sympathomimetic amines appear in various cold remedies, common nasal and ophthalmological decongestants, and most asthma preparations. After passage of Dietary Supplement Health and Education Act in 1994, ephedrine appeared in various over-the-counter dietary supplements for weight loss and energy and was often listed as ephedra and ma huang. However, following at least 150 deaths, including several high-profile athletes purportedly linked to ephedrine use (Steve Bechler of the Baltimore Orioles, Korey Stringer of the Minnesota Vikings, and Rashidi Wheeler of Northwestern University), the FDA banned dietary supplements containing ephedrine in 2004. Despite the ban, ephedrine-containing products are still available on the Internet, and 0.9% of NCAA athletes in 2012 still reported using ephedrine in the past 12 months.

**Mechanism:** Response to sympathomimetic amines depends on relative selectivity of the specific drug. Although earlier scientific studies demonstrated that ephedrine improved athletic performance, three studies have shown no significant increases in performance.

- Alpha effects: smooth muscle contraction, primarily vasoconstriction
- $Beta_1$ effects: production of intracellular cAMP and increased heart rate and strength of contraction
- $Beta_2$ effects: smooth muscle relaxation, bronchodilation, and stimulation of skeletal muscles

**Therapeutic uses:** nonemergent treatment of allergic reactions, asthma, hypotension during spinal anesthesia, atrioventricular block, and nasal congestion

**Dosage:** Many types of drugs in this category have varying potencies and durations of action.

**Adverse reactions with increasing doses:** anxiety, epigastric distress, palpitations, tremulousness, insomnia, drowsiness, hypertension, stroke, and seizures

## eBOOK SUPPLEMENTS

### eAppendix 26-1, Cocaine

**Definition:** naturally occurring alkaloid derived from the leaves of *Erythroxylon coca* plant. Although it is a topical anesthetic, it also acts as central nervous system stimulant. This effect has led to its use by athletes as an ergogenic aid.

**Prevalence:**

- 2013 NSDUH showed that 1.5 million (1.0%) individuals had used cocaine within the past month. In addition, 855,000 had abused cocaine (0.3%), although the abuse rate in 2013 was lower than that reported in 2002–2009 (0.4% and 0.7%, respectively).
- The 2012 NCAA study found that 1.8% of athletes had used cocaine in the previous 12 months, which has not significantly changed since 2005.
- Cocaine dependence is typically associated with cyclical patterns of drug use and abstinence. During abstinence, periods of intense cocaine craving, and other withdrawal symptoms (anergia, anhedonia, and depression) often lead to relapse.

**Mechanism:** increases release and blocks reuptake of norepinephrine from neurons in the nervous system. Increased availability of epinephrine causes euphoria, increased blood pressure, tachycardia, lowered threshold for seizures, and ventricular arrhythmias. Cocaine also stabilizes axonal membranes and blocks initiation and conduction of nerve impulses. Combined with its properties as a vasoconstrictor, it is an excellent topical anesthetic. Cocaine may cause hyperglycemia, hyperthermia, and increased peripheral reflex speed.

**Therapeutic uses:** Although scientists of the 19$^{th}$ century hailed cocaine as a cure for various ailments from hemorrhoids to broken bones, its legitimate use is now limited to topical anesthesia alone.

**Dosage:** is readily absorbed by intravenous, intranasal, and pulmonary routes

Recreational users of intranasal cocaine may use 1–3 gm/week. To mimic intense high associated with IV use without complications of needles, users have turned to smoking. This began with smoking of its free alkaloid form, known as "free base." Availability of ready-to-smoke, low-priced free-base ("crack") cocaine has led to epidemic smoking in urban areas. Effects of crack are rapid and lasts only 5–10 minutes. Half-life of cocaine is 2–6 hours; however, it can be detected in the urine for 3–5 days.

**Adverse reactions:**

- **Cardiovascular:** Increased levels of catecholamines associated with cocaine use can directly induce ventricular dysrhythmia, coronary vasospasm with thrombosis, and myocardial infarction, all of which can lead to **sudden death, even in patients without underlying heart disease**. Aortic rupture and cerebrovascular accidents also have been reported.
- **Central nervous system:** Chronic use can result in agitation, insomnia, and tremulousness. Toxic psychosis, severe depression, paranoia, and dysphorias have been reported as well as rapid addiction.
- **Respiratory system:** When consumed intranasally, cocaine can cause swelling of nasal mucosa, rhinitis, sinusitis, epistaxis, and nasal septal necrosis. Bronchitis and bronchiolitis obliterans with organizing pneumonia have been reported.
- **Infectious diseases:** Several animal studies have explained why cocaine use has been linked to HIV progression. The underlying mechanism is thought to be linked to enhanced viral DNA integration in primary CD4 T cells.
- **Considerations in athletes:** Cocaine has direct effects on central thermoregulation, and athletes exercising in heat are susceptible to hyperthermia. It has been proposed that elite sprint-trained athletes may be at a higher risk of severe lactic acidosis and cocaine-induced seizures because of greater proportion of glycolytic muscle fibers. Studies have demonstrated that chronic cocaine-conditioned animals have exaggerated catecholamine response to a combination of cocaine and exercise. A study by Welder and Melchert revealed increase in myocardial events when cocaine was combined with AAS.

**Detection:** Cocaine is readily detectable by most drug testing. According to data from Anderson and colleagues, most cocaine users do not participate in drug use with teammates. May be difficult for coaches, trainers, or team physicians to detect patterns of cocaine usage.

**Detection:** Banned by the WADA and can be detected by drug testing

The WADA allows the use of inhaled selective beta$_2$-agonists such as terbutaline, albuterol, bitolterol, orciprenaline, and rimiterol. Sympathomimetic amines such as ephedrine and synephrine are banned by the NCAA, but phenylephrine and pseudoephedrine are not.

### Clenbuterol

**Clenbuterol has garnered attention for its potential as anabolic substance**

**Definition:** beta$_2$ agonist, similar to albuterol, used as a bronchodilator; has recently been considered anabolic or "repartitioning" agent; anabolic effects have been purported to occur only with oral forms, not via inhalational route

**Prevalence:** Limited information about prevalence; is derived mostly from increasing number of athletes who are disqualified because of positive drug tests

Six Olympians were disqualified in 1992: two Americans, two Germans, and two British. Parkinson and Evans reported clenbuterol use for fat loss by AAS users. A 2007 report found that clenbuterol was illegally present in certain dietary supplements available on the internet.

**Mechanism:** Most data about anabolic effects derived from animal studies. To date, no study has examined effects of oral clenbuterol on nonasthmatic athletes. Livestock treated with clenbuterol demonstrated increased muscle mass and decreased fatty deposits. Denervated rat hindlimbs demonstrated reduced muscle wasting when treated with clenbuterol as well as a decreased net bone loss. Clenbuterol produced no changes in the cross-sectional area of muscles in patients undergoing medial meniscectomy in a randomized study conducted by Maltin et al. Although the findings were not statistically significant, the authors asserted that the clenbuterol group patients regained strength more rapidly. The exact mechanism of anabolic effects of clenbuterol have not been determined, but it has been previously shown that catecholamines attenuate amino acid release from muscles by beta-mediated depression of protein catabolism. Clenbuterol may also act by increasing contractile tension.

**Therapeutic uses:** Clenbuterol is used as a bronchodilator for treatment of asthma but is not legally available in the US.

**Dosage:** Recommended dosage for asthma is 0.02–0.03 mg twice daily. Anabolic dosage is not known, but extrapolation from animal data translates into 0.001–0.01 mg/kg or 0.07–0.7 mg in 70-kg person. Maltin et al. used a lower asthma dosage, 0.02 mg twice daily, in their study on orthopedic patients.

**Adverse reactions** (typical beta$_2$ effects): muscle tremor, palpitations, muscle cramps, tachycardia, tenseness, headaches, and peripheral vasodilation; case report of clenbuterol potentiating the effects of AAS that resulted in myocardial infarction as well as multiple other cases associating its use with MIs

**Detection:** Clenbuterol is prohibited by the WADA and NCAA. It is also banned by the Association of Official Racing Chemists for its ergogenic effects on thoroughbred racehorses. It can be detected in urine by gas chromatography/mass spectroscopy (GC/MS).

### Other Beta$_2$ Agonists

Debate about clenbuterol has raised questions about anabolic effects and possible banning of other beta$_2$ agonists. It is controversial because most studies of athletes reveal that 10% suffer from exercise-induced asthma and depend on beta$_2$ agonists to compete. While most studies about ergogenic effects of other inhaled beta$_2$ agonists have been inconclusive, a recent randomized, double-blind, crossover study demonstrated increased power output associated with increased rates of glycolysis and glycogenolysis in skeletal muscles of sprinters who had been given inhaled terbutaline; in addition, the counteraction of reduction of ATP in Type II fibers with use of terbutaline demonstrated in this study may explain the delay in development of fatigue in these fibers. While further research is warranted, it would be extremely difficult to deny asthmatics the use of inhaled beta$_2$ agonists.

### Designer Stimulants

Numerous new stimulants have been developed in recent years in order to circumvent regulations regarding dietary supplements and potentially avoid drug testing. These include heptaminol, dimethylamylamine (DMAA), dimethlybutylamine (DMBA), methylhexanamine, oxilofrine, and others. Many of these have never been studied in humans and have been associated with significant adverse effects.

## NONSTEROIDAL ANTI-INFLAMMATORY DRUGS

See eAppendix 26-2 on **www.ExpertConsult.com**.

## ALCOHOL

**Definition:** Ethanol, most abused recreational drug in the US, is classified as a depressant.

**Prevalence:** Approximately 70% of adult Americans drink alcohol, and the per capita consumption is estimated at 2.23 gallons/year. Perhaps 5%–10% of drinkers are, or will become, alcoholics. There are 75,000 annual alcohol-related deaths. The 2012 NCAA study revealed that 83% of student-athletes consume alcohol; moreover, 49% and 15% consumed more than five and 10 drinks per setting, respectively. It has been suggested that athletes are at a high risk of developing such behaviors.

**Mechanism:** Physiologic effects of alcohol are well known and not reviewed here.

**Therapeutic uses:** None

**Adverse reactions:** Physicians are familiar with several adverse consequences of alcohol abuse. With respect to athletes:

- **After literature review, the American College of Sports Medicine issued a current comment about use of alcohol in relation to athletic performance:** Acute ingestion of alcohol has deleterious effects on various psychomotor skills, including reaction times, hand–eye coordination, accuracy, balance, and complex coordination. However, alcohol consumption does not substantially influence physiologic functions that are crucial to physical performance ($VO_2$ max), respiratory dynamics, and cardiac function. It does not improve muscular work capacity and may decrease performance levels as well as impair temperature regulation during prolonged exercise in cold environment.
- A study by Urbano-Marquez et al. concluded that alcohol is toxic to striated muscle in a dose-dependent manner.
- Driving while impaired substantially increases the risk of eventual death in alcohol-related crashes.

**Detection:** The WADA prohibits use of alcohol in the following sports: air sports, archery, automobile, motorcycling, and powerboating. Neither the NCAA nor WADA specifically tests for presence of alcohol in other sports; however, breath or blood levels may be determined at the request of an International Federation.

**Identification:** American Medical Association defines **alcoholism** as an "illness characterized by significant impairment that is directly associated with persistent and excessive use of alcohol. Impairment may involve physiologic, psychological, or social dysfunction." Because effects of alcoholism may take 5–20 years to develop and usually occur after the age of 30 years, early identification of athletes with alcohol problems is imperative.

## eAppendix 26-2, Nonsteroidal Anti-Inflammatory Drugs

**Definition:** Nonsteroidal anti-inflammatory drugs (NSAIDs) have analgesic and anti-inflammatory properties because of their inhibition of prostaglandin synthesis.

**Prevalence:** NSAIDs are among the most widely used therapeutic drugs by athletes and nonathletes. At least 28 brands of NSAIDs are sold in the US, accounting for 70 billion prescriptions and 30 billion over-the-counter tablets annually. Given their use in soft-tissue injuries, it is not surprising that Anderson et al. found that 30% of their sample used NSAIDs.

**Mechanism:** Although the exact mechanism is uncertain, inhibition of prostaglandin synthesis has several effects: inhibition of superoxide generation, competition with prostaglandins for binding at receptor sites, inhibition of leukocyte migration, inhibition of release of lysosomal enzymes from leukocytes, and interactions with adenylate cyclase system. Effects of these actions is to ameliorate soft tissue inflammation. In addition, NSAIDs uncouple oxidative phosphorylation in skeletal muscles. The discovery of specific cyclooxygenase enzymes (COX-1 and -2) allowed for the development of NSAIDs with specific COX-2 inhibition.

**Therapeutic uses:** NSAIDs are useful as adjuncts to treatment of tendonitis, sprains, strains, and other soft tissue derangements. In addition, they are used in the treatment of rheumatoid arthritis, other rheumatologic conditions, degenerative joint disease, acute and chronic pain, and dysmenorrhea.

**Dosage:** The US offers a wider variety of NSAIDs than any other country. In addition to numerous prescription NSAIDS, over-the-counter medications include aspirin, ibuprofen, and naproxen. Variable half-lives allow once-daily dosing. Certain NSAIDs are available as topical gels (diclofenac) and parenterally (ketorolac).

**Adverse reactions:**

- **Gastrointestinal (GI):** upset that causes discontinuation of NSAIDs has been reported in 2%–10% of patients with rheumatoid arthritis; effects include nausea, dyspepsia, gastritis, ulceration, bleeding, and hepatotoxicity. NSAIDs may also cause small intestine enteropathy. Since the GI tract is rich in COX-1, theoretically, COX-2 NSAIDs should have fewer GI adverse effects. However, in reality, both COX-1 and COX-2 NSAIDs can cause significant gastric ulceration and GI bleeding. Interestingly, topical formulations can also cause GI ulceration, suggesting that the effects are not just locally mediated.
- **Renal:** Increased serum creatinine, sodium and water retention, hyperkalemia, papillary necrosis, interstitial nephritis, proteinuria, and acute renal failure

  Of note, renal pathology appears more frequently in patients who take NSAIDs while they are hypovolemic. A potentially troublesome study on NSAIDs and exercise conducted by Walker et al. demonstrated that indomethacin can compromise renal function and potentiate the risk of acute renal failure.
- **Hematological:** Bone marrow suppression, reversible inhibition of platelet aggregation, and possible decreased coagulation; the latter two are less frequent with COX-2 NSAIDs
- **Central nervous system:** Headache, tinnitus, dizziness, and sedation
- **Cardiovascular:** COX-2 inhibitors have been shown to increase the risk of major fatal and nonfatal cardiovascular events (cardiovascular death, heart attack, stroke, or heart failure). The FDA strengthened this risk warning in 2015 and stated that cardiovascular risks may increase with higher doses, may appear as early as a week within NSAID use, and may even occur in patients without any cardiovascular risk factors. There may not be equal risk among different NSAIDS, but more evidence is needed.

**Considerations in athletes:** Uncoupling oxidative phosphorylation can affect oxygen consumption and directly stimulate ventilation, promote sweating and dehydration, and lead to heat illness. There has also been speculation that NSAIDs interfere with fracture healing. Hence, there must be clear indications for the use of an NSAID, careful monitoring and appropriate individual selection of the appropriate agent. There have been reports of athletes using intramuscular ketorolac before competitions without a specific injury. There is no rational basis for this, and in fact, oral administration demonstrates earlier peak levels than intramuscular dosing.

**Detection:** All NSAIDs are allowed by both the NCAA and WADA.

Several measures are designed to help assess alcohol problems, including Perceived Benefit of Drinking Scale (PBDS), Children of Alcoholics Test (CAST), Michigan Alcoholism Screening Test (MAST), and **CAGE questionnaire**. Education and identification are keys to prevention in athletes.

## NICOTINE

**Definition:** Volatile alkaloid derived from tobacco and responsible for various effects of tobacco

It first stimulates (small doses) then depresses (large doses) autonomic ganglia and myoneural junctions. Methods of consumption include cigarettes, electronic cigarettes (e-cigs), and smokeless tobacco (loose leaf tobacco [chewing], moist or dry powdered tobacco [snuff or "dipping"], compressed tobacco ["plug"], and e-cigs).

**Prevalence:** According to the 2012 NCAA study, number of daily cigarette users has decreased dramatically. However, there was an increase in spit tobacco use within past 12 months from 2005 (15.7%) to 2012 (17.4%). A recent 2015 CDC study that evaluated tobacco use in high school athletes from 2001 to 2013 demonstrated that smokeless tobacco use had significantly increased (10%–11.1%) despite a decline in both total tobacco use (33.9%–22.4%) and use of combustible tobacco products (31.5%–19.5%). Of note, an athlete was more likely to use smokeless tobacco products, if he/she perceived that smokeless tobacco was less dangerous, and participated in multiple sports. While controversial, some experts believe that marketing of e-cigs may be directed to youths. A 2014 National Youth Tobacco Survey of 22,000 youths conducted by the CDC showed that e-cig use had tripled (4.5%–13.4%) from 2013 to 2014.

**Mechanism:** Nicotine has various actions, depending on receptor binding and dose, and readily crosses the blood–brain barrier. Binds to acetylcholine receptors on autonomic ganglia, adrenal medulla, and neuromuscular junction and in the central nervous system. Stimulation of central nicotinic receptors activates neurohumoral pathways of the central nervous system to release acetylcholine, norepinephrine, dopamine, serotonin, vasopressin, GH, and ACTH. Nicotine affects sympathetic nerves by releasing catecholamines. At low doses, ganglionic stimulation and sympathetic discharge leads to increase in heart rate and blood pressure that are mediated through the central nervous system. At moderate doses, effects of direct peripheral nervous system lead to ganglionic stimulation and adrenal catecholamine release. At high doses, ganglionic blockade leads to hypotension and bradycardia. Pharmacodynamic tolerance develops to subjective and hemodynamic effects.

**Therapeutic uses:** As aid to smoking cessation, nicotine gum and transdermal patches have been somewhat effective in reducing withdrawal symptoms of physical nicotine dependence in smokers who are trying to quit. Bupropion has been used to reduce cravings and varenicline may assist quitting by blocking alpha-4-beta-2 nicotinic acetylcholine receptors. There are conflicting reports about the use of e-cigs as adjuncts to smoking cessation, and at this point, these cannot be recommended as a safe alternative.

**Dosage:** Smokeless tobacco users have blood nicotine levels equivalent to nicotine-dependent smokers. Normal single dose of snuff contains 1.5–2.5 g of tobacco, and each gram has 14 mg of nicotine. Ten percent of nicotine is absorbed, leading to 2.0–3.5 mg nicotine in bloodstream (2–3-times higher than the dose of a standard 1-mg cigarette). Average single dose of chewing tobacco contains 7 gm, with a nicotine content of 7.8 mg/gm; 8% is available to be absorbed. On an average, 8–10 chews/day result in a nicotine dose equivalent to 30–40 cigarettes/day.

**Adverse reactions:** Long-term effects of cigarette smoking are well known and are not reviewed here. Studies on the effects of smokeless tobacco are enlisted below:

- **Oropharynx:** 50-fold increase in oral carcinomas, 2.4-times higher incidence of dental caries, and increase in the incidence of gingival disease and leukoplakia
- **Hemodynamics** (cigarette smoking compared with smokeless tobacco): similar levels of nicotine throughout the day, equivalent increases in heart rate and myocardial oxygen demand; increased sodium (added for flavoring) absorption from smokeless tobacco may lead to increase in blood pressure
- **Reaction time and concentration:** smokeless tobacco has long been touted for its ability to improve reaction time and concentration. A study on athletes and nonathletes conducted by Edwards et al. demonstrated no neuromuscular performance enhancement; no changes in reaction time, movement time, or total response time; and a significant elevation in heart rate. A survey by Connolly et al. of Major League Baseball players who used smokeless tobacco found that only 10% believe it improved concentration, and none felt that it sharpened reflexes.
- **Addiction:** Smokeless tobacco can be as addictive as cigarette smoking. National Cancer Institute, in conjunction with Major League Baseball, has prepared "Beat the Smokeless Habit: Game Plan for Success" to provide readers with facts about smokeless tobacco: includes a "9-inning game plan" to help athletes quit smokeless tobacco. An additional program, "Knock Tobacco Out of the Park," can be found at www.tobaccofreebaseball.org.

**Detection:** Nicotine is not currently tested for by international organizations or the NCAA, but the NCAA bans the use of tobacco products by players, coaches, and officials during competition. Major League Baseball bans the use of spit tobacco in Minor Leagues and open displays in Major Leagues. Several stadiums now ban the use of any tobacco products by spectators or participants.

## MARIJUANA

**Definition:** naturally occurring cannabinoid-containing active ingredient δ9-tetrahydrocannabinol (THC); is currently an illegal recreational drug in most states and is used as a euphoriant

Many states now allow the medicinal use of marijuana, and others have legalized its nonmedicinal use.

**Prevalence:** Estimated that >94 million Americans aged ≥12 years have tried marijuana, and at least 14.6 million are regular users

Of the 22.6% of athletes who had used marijuana in the past 12 months in the 2012 NCAA study, 97% stated that they had used marijuana for reasons not related to sports performance or injuries.

**Mechanism:** THC exerts its effects on various tissues and systems, most prominently the central nervous system and the cardiovascular system. Effects depend on route, dose, setting, and prior experience of the user.

- **Cardiovascular:** tachycardia is dose-related and can be blocked by propranolol. Systolic blood pressure increases in the supine position and decreases in the standing position.
- **Central nervous system:** impaired motor coordination, decreased short-term memory, difficulty in concentrating, and decline in work performance
- **Male reproductive system:** decreased plasma testosterone, gynecomastia, and oligospermia

**Therapeutic uses:** THC has been used as an antiemetic agent in conjunction with chemotherapy for cancer patients and for lowering intraocular pressure in glaucoma patients. It is also anecdotally used for various symptoms (e.g., anxiety or pain). It

is important for athletes to remember, particularly during interstate travel, that regardless of state law, marijuana use is still a violation of federal law and banned by most drug testing programs.

**Dosage:** THC content of marijuana in the US ranges from 0.5%–11%; serum concentration depends on the method of ingestion.

**Adverse reactions:** Renaud and Cormier found following effects of marijuana on exercise performance: reduction of maximal exercise performance with premature achievement of $VO_2$ max; no effects on tidal volume, arterial blood pressure, or carboxyhemoglobin compared to controls; causes inhibition of sweating that can lead to increase in core body temperature

**Detection:** Banned by the WADA in competitions at a level of >150 ng/mL, while it is considered a street drug by the NCAA and is prohibited at a level of 5 ng/mL; because of high lipid solubility, can be detected for as long as 2–4 weeks by drug testing

**Synthetic cannabinoids**: First developed for research purposes in the 1980s by John W. Huffman (JWH) and not intended for human consumption: 2011 Monitoring the Future study revealed that 11% of 12$^{th}$ graders had tried these; first compound was JWH-018, but currently, there are many others and are 10-times more potent than THC: known as Spice, K2, and so on

Usually is an herbal mixture sprayed with synthetic cannabinoids and has been recently classified as Schedule I by the DEA; can cause lethargy, hypotension, seizures, hallucinations, and paranoia; should be a high index of suspicion in an impaired individual with negative toxicology screen for THC; are banned by most sports organizations and can be detected in urine

## BLOOD DOPING

**Definition:** Blood is infused into a nonanemic athlete for increasing red blood cell mass. Blood doping can be autologous (from the same person) or heterologous (donated from another person).

**Prevalence:** The actual extent of blood doping is unknown; however, there have been widespread rumors of this practice for the past 20 years. In 1984, USOC admitted that seven US cyclists had engaged in blood doping at Summer Olympic Games. In 1990, a study by Scarpino et al. of 1018 Italian athletes revealed that 7% athletes had tried blood doping. Advent of better tests for EPO has led athletes back to blood doping. Cyclist Tyler Hamilton tested positive for blood transfusions after winning the gold medal at the Athens 2004 Olympics.

**Mechanism:** Transfusion increases oxygen delivery to exercising muscles, and studies have confirmed that red cell mass and $VO_2$ max are well correlated. Studies on blood doping in elite runners compared with controls have demonstrated improvement in maximal oxygen consumption, increased total exercise time, and increased hemoglobin concentration.

**Therapeutic uses:** Red cell transfusions are limited to patients with symptomatic anemia.

**Dosage:** Most studies have used 2000 mL of homologous blood or 900–1800 mL of cryopreserved autologous blood.

**Adverse reactions:** Incorrectly matched donor blood can result in transfusion reactions that may be fatal. Immune side effects are reported in 3% of all transfusions. Moreover, using donor blood has additional risks of infectious complications, which is substantially lower with use of autologous blood.

**Detection:** The WADA bans blood doping, but enforcement is limited by the lack of effective technique for its detection. A test for heterologous blood transfusions was implemented at the 2004 Summer Olympic Games in Athens. The test confirms doping by providing evidence of different cell populations. The WADA and the Partnership for Clean Competition are funding research projects aimed at developing a test for autologous transfusions through various methods, including microRNA and other potential markers. The WADA has also implemented the Athlete Hematological Passport to detect subtle changes in hematological parameters that may be indicative of hemoglobin manipulation.

## ERYTHROPOIETIN (EPO) AND RELATED COMPOUNDS

**Definition:** Sialic acid-containing hormone that enhances erythropoiesis by stimulating the formation of proerythroblasts and release of reticulocytes from bone marrow; is primarily secreted by kidneys, and level of erythropoietin (EPO) is inversely related to the number of circulating red cells. In 1986, a recombinant form of EPO (called r-HuEPO) was discovered, which was identical to natural EPO except for addition of sialic acid residues. In 2002, a long-acting form called darbepoetin was introduced. In 2008, a large (60 kDa) polyethylene glycol compound called Continuous Erythropoietin Receptor Activator (CERA, or trade name Micera) was approved with a half-life that was 20 times greater than that of h-HuEPO with very little urinary excretion.

**Prevalence:** With increased attention (and attendant risks) to blood doping, r-HuEPO has become a potential avenue for abuse among athletes.

- Unexplained deaths of 18 Dutch and Belgian cyclists between 1987 and 1990 raised specter of r-HuEPO abuse.
- The Festina cycling team was expelled in the first week of the 1998 Tour de France after a team car was found loaded with PEDs including r-HuEPO.
- Several cyclists, including Bernard Kohl and members of Lance Armstorng's cycling team, have admitted to the use of EPO products.

**Mechanism:** Subcutaneous injection of r-HuEPO stimulates red blood cell production within days, and effects can be seen for as long as 3–4 weeks. Based on data from red-cell reinfusion studies, r-HuEPO can theoretically increase $VO_2$ max by 10%. An uncontrolled study using r-HuEPO demonstrated increases in mean maximal oxygen uptake and run time to exhaustion.

**Therapeutic uses:** Studies in patients suffering from anemia secondary to end-stage renal disease have demonstrated that r-HuEPO eliminates the need for transfusions and restores hematocrit to normal in many patients; can partially correct renal anemia and significantly increases both exercise capacity and maximum work and can maintain normal hemoglobin concentration in uremic patients over time. r-HuEPO was found useful in treatment of anemias secondary to prematurity, multiple myeloma, and cancer and in patients with AIDS treated with zidovudine. r-HuEPO safely and effectively increases yield of autologous blood donors over a 21-day period and can reduce the need for transfusions in patients undergoing hip replacement.

**Dosage:** In a study of autologous blood donors with initially normal hemoglobin levels, 600 U/kg of r-HuEPO was administered intravenously six times over 21 days. Doses in chronic renal patients have ranged between 15 and 500 U/kg administered three times/week. r-HuEPO is usually administered subcutaneously, but improved detection methods have led athletes to begin intravenous microdosing to avoid detection.

**Adverse reactions:**

- Observed side effects in patients with renal anemia
  - Hypertensive patients required additional antihypertensive medications.
  - Serum levels of potassium and bilirubin were increased.
  - 20% of patients developed flu-like syndrome.

- 14% of patients developed thrombosis of arteriovenous fistulas and veins.
- 11 patients reported visual hallucinations.

- Potential risks of r-HuEPO in athletes with normal hemoglobin
  - Increases in hemoglobin and blood viscosity accentuated with dehydration may lead to cerebral or cardiovascular ischemia, vascular thrombosis, hypotension, hyperkalemia, and iron deficiency.
  - Attendant risks of intravenous medication (e.g., infection with hepatitis, human immunodeficiency virus [HIV], and endocarditis)

**Detection:** Direct test for the presence of r-HuEPO in the urine; test shows isoform patterns of r-HuEPO in urine by separation in an electrical field and detection with a very sensitive and selective method. Isoform pattern of rHuEPO is distinctively different from native EPO. Moreover, isoelectric focusing urine test yields a pattern for darbepoetin alfa that is distinct from both EPO and r-HuEPO. Testing for CERA with standard urinary isoelectric focusing can be challenging owing to low urinary excretion, but a related serum test using the SARCOSYL–PAGE method has proven adept at its detection.

## GAMMA-HYDROXYBUTYRATE AND DERIVATIVES

See eAppendix 26-3 on **www.ExpertConsult.com**.

## AGENTS WITH ANTIESTROGENIC ACTIVITY

**Definition:** Substances that oppose estrogens and include
- Aromatase inhibitors such as anastrazole and letrazole
- Selective estrogen receptor modifiers such as tamoxifen and raloxifene
- Other antiestrogen substances such as clomiphene

**Prevalence:** Unknown, but multiple anecdotal reports among users of anabolic steroids

**Mechanism:** High doses of androgens will overwhelm hepatic capacity, and some of the excess is aromatized to estrogenic compounds, creating unwanted adverse effects in males. These drugs are used to counteract the effects, although they are not technically performance-enhancing themselves in men. In addition, AAS users have used clomiphene to help testes resume testosterone production, but there is limited data on efficacy, although may improve fertility

**Potential therapeutic uses:** Used for various legitimate uses, primarily in women, including adjuvant therapy in breast cancer and ovulation induction

**Adverse reactions:** Rarely used therapeutically in males; as such, adverse reactions are limited to women; is likely that interference with the hypothalamic–pituitary–gonadal axis will have major adverse effects in males

**Detection:** Banned by the WADA and can be detected using LC-MS

## GENETIC DOPING

**Definition**: Nontherapeutic use of genes, genetic elements, and/or cells that have the capability to enhance athletic performance

**Mechanism:** May be achieved through several methods:
- Viral vector, such as modified adenovirus
- Gene transfer that produces a specific message or "switch" to turn on production of a desired substance

**Potential therapeutic uses:** Genetic manipulation has the potential to cure a wide spectrum of diseases such as muscular dystrophy and has been used in congenital immunodeficiency states.

**Adverse reactions:** Use of gene therapy has resulted in certain severe effects, including the development of cancer. Primate studies with the insertion of an EPO-producing gene resulted in animals requiring regular phlebotomies owing to uncontrolled red cell production. Immune reactions are a major concern.

**Specific drugs:** Has been speculated, but unconfirmed, that the patented drug Repoxygen may have been used by athletes. Drug alters EPO production in response to low oxygen tension.

**Detection:** Although practice is banned by the WADA, there are no tests for detection. In 2004, the WADA also created an Expert Group on Gene Doping to study latest advances in the field of gene therapy and methods to detect doping; research projects funded by the WADA in this area

Possible deterrents include:
- Detection of viral genome but may be difficult if a common virus is used
- Detection of identifying switch
- Testing serial biomarkers or pharmacodynamic tests
- Manufacturer "labeling" of genetic material to allow detection
- Control over production and supply of materials

**Miscellaneous:** In addition to genetic doping, practice of genetically identifying sport-specific genes in children has begun in order to promote athletic success. Some of these markers include creatine kinase, angiotensin-converting enzyme, and mitochondrial enzymes.

**Summary:** Genetic doping and genetic identification of athletic traits is on the horizon and likely very difficult to harness once unleashed. It raises ethical issues and threatens the very definition of "sports."

## DRUG TESTING

**Purpose:** A major means of attempting to enforce compliance with a list of banned substances; increased public awareness by positive tests in elite athletes. To date, most drug testing has been of the "announced" variety at championship or Olympic events, although there are attempts to increase unannounced testing.

**Cost:** USADA expenses were almost $16 million in 2014, of which, drug testing and results management accounted for almost $10 million to conduct 8900 blood and urine tests, both in and out of competition.

**Reliability:** Tests vary in specificity, sensitivity, and expense.
- **Radioimmunoassay and enzyme-multiplied immunoassay:** two most commonly used screening methods

  Although manufacturers claim a 97%–99% accuracy rate, such is not usually the case. To avoid false-positive tests when an athlete's career may be at stake, a second, highly sensitive and specific test is often required.
- **Gas chromatography/mass spectroscopy (GC/MS):** gold standard and only drug test legally admissible in court. All state-of-art laboratories must use GC/MS as confirmatory test because it provides a "fingerprint" of the detected substance. Unfortunately, high cost of both equipment and test prohibits smaller laboratories from using GC/MS.
- **Gas chromatography–combustion–isotope-ratio mass spectrometry (IRMS):** a new tool that the WADA accepts as a method to determine whether an elevated ratio of testosterone to its isomer epitestosterone (T:E) is the result of exogenous testosterone use

  IRMS is based on the finding that 98.9% of carbon atoms in nature are $^{12}C$, with 1.1% being $^{13}C$ (an isotope that contains an additional neutron). The ratio of $^{13}C/^{12}C$ can be measured with high accuracy and precision by using IRMS. Extremely small differences in the abundance of $^{13}C$ can be detected to allow differentiation of carbon sources. IRMS values for steroids are expressed as delta values: $\delta^{13}C\%$. The more negative a delta value, the less $^{13}C$ the compound contains. Pharmaceutical testosterone is manufactured from

## eAppendix 26-3, Gamma-Hydroxybutyrate and Derivatives

**Definition:** endogenous neurotransmitter produced through metabolism of gamma-aminobutyric acid (GABA). Gamma-hydroxybutyrate (GHB) increases cerebral dopamine levels and acts on the endogenous opioid system. It is structurally similar to GABA. Following its ban in 1991, manufacturers began producing GHB precursors, including gamma-butyrolactone (GBL), 1,4-butanediol, and gamma-valerolactone (GVL).

**Prevalence:** There are no surveys to date about the prevalence of its use; however, anecdotal reports seem to indicate widespread use.

**Mechanism:** GHB crosses the blood–brain barrier and has been shown to double dopamine and dynorphin levels in the brain. Hippocampal receptors for GHB have been isolated, and synaptic transmission and regulation have been demonstrated. GHB facilitates slow wave sleep, which is associated with GH release. It is postulated that this may increase muscle mass.

**Potential therapeutic uses:** GHB is a Schedule I drug and because of its ability to increase cerebral dopamine levels, GHB has been experimentally used to treat myoclonus and cataplexy associated with narcolepsy (only indication for use in the US). GHB has been used in other countries as an anesthetic agent (particularly in children) for ethanol withdrawal and as treatment for ischemic conditions. It has been illegally marketed in the US as a muscle-building drug and sleeping aid.

**Dosage:** Usual directions are one tablet or ½ to three teaspoons of powder dissolved in water. It is unclear how much athletes are consuming. Typically, 10 mg/kg oral consumption can cause amnesia and hypotonia; 20–30 mg/kg produces somnolence within 15 minutes; and >50 mg/kg can cause unconsciousness and coma.

**Adverse reactions:**

- Multiple cases of poisonings secondary to GHB have been reported with patients complaining of GI symptoms, central nervous system and respiratory depression, and uncontrolled movements.
- At least 11 patients have been hospitalized, and nine required ventilator support.
- In 1991, FDA issued a report that GHB was unsafe and illicit and that its use should be discontinued because of potentially dangerous adverse effects.
- In a 2005 National Swedish Epidemiology Study, there were 141 reported toxic events related to GHB and GBL. There have also been multiple deaths related to GHB's precursor, 1,4-butanediol.

**Detection:** These compounds are highly volatile with half-lives of approximately 1 hour, making detection difficult. Kankaapaa recently reported an experimental method for blood and urine detection.

a soy compound that contains relatively less $^{13}C$ content compared with endogenously produced testosterone, thus yielding significantly different results on IRMS analysis. However, analysis of seized illegal testosterone has revealed the presence of $^{13}C$-enriched testosterone products that could potentially reduce the effectiveness of the test.

- **Athlete Biological Passport (ABP):** Emerging field of longitudinal testing to help detect manipulation of red blood cell parameters and androgens. While not a direct test for a specific substance, can be an aid in early detection of suspicious samples

**Testing for anabolic steroids/testosterone:**

- To combat use of exogenous testosterone, T:E ratio has been quantified. The normal T:E ratio is 1:1. When exogenous testosterone is administered, serum testosterone is elevated out of proportion relative to epitestosterone. A ratio >6:1 is considered a positive test by the NCAA; however, the USADA and NFL consider a ratio >4:1 to be positive. The test is limited because of genetic variations in testosterone metabolism that can result in T:E ratios >4:1. Because of improved accuracy of high-resolution GC/MS in detecting synthetic AAS, use of exogenous testosterone has increased. Athletes have used epitestosterone in conjunction with testosterone ("the cream") to maintain "normal" T:E ratio. To combat this practice, the WADA and USADA have currently banned epitestosterone at concentrations >200 ng/mL.
- GC-IRMS (as above)
- **Athlete Steroid Profile:** Similar to the ABP, this longitudinal test can detect perturbations in an athlete's endogenous steroid profile that suggest androgen use.

**Circumvention by athletes:** Although GC/MS may approach 100% accuracy, athletes have attempted numerous methods to avoid detection.

- **Masking agents:** Diuretics and tubular blocking agents such as probenecid have been used to mask or dilute banned substances in the urine. Drug tests now use minimum urinary specific gravity to combat this practice and ban these drugs.
- **Determination of drug half-life:** With announced drug testing, athletes can determine for how long a drug can be detected in the urine; this can be addressed with random unannounced testing.
- **Substitution of urine:** Athletes have developed numerous methods to substitute "clean" urine, including self-catheterization and innovative "delivery systems." To eliminate this problem, collection is conducted under constant supervision and direct observation.

**Extent of testing:** Most athletic organizations, professional and amateur, have developed drug-testing programs. Policies are subjected to frequent change depending on law, collective bargaining, and contemporary issues. It is best to check with respective organizations when specific questions arise.

- **Olympic level:** The WADA and USADA conduct formal drug testing at sanctioned events, such as Olympics, Olympic Trials, and Pan Am Games. No announced notice (NAN) testing out of competition has been initiated as an increased deterrent. National pool players are required to provide whereabouts to the USADA and testing agencies. The WADA has a strict testing policy. Olympic athletes can be randomly tested 24 hours a day, 365 days a year, with no exceptions. Athletes are allowed two missed tests before it results in a suspension. Usual suspensions for PEDs are a minimum of 2 years from competition.
- **Collegiate level:** The NCAA began testing in 1986 at postseason football games and championship events at Division I, II, and III levels. The NCAA conducts year-round drug testing on the campuses of NCAA Division I and Division II member schools and on campuses of Division III schools that sponsor Division I or Division II sports. First positive test results in 1-year suspension and second positive test for PEDs leads to permanent loss of eligibility.
- **Major League Baseball**: Separate programs for Minor and Major Leagues with both programs testing in and out of season. First positive test for PEDs results in an 80-game suspension, second positive test in 162-game suspension, and third positive test in permanent suspension.
- **National Basketball Association:** Amphetamine, cocaine, LSD, opiates, PCP, marijuana, and steroids are prohibited. Individual players can be tested with "reasonable cause" for prohibited substances without prior notice, and rookies are tested three times on a random basis throughout the season.
- **National Football League:** currently tests both in and out of season in an unannounced fashion. First positive test for most PEDs is a 4-game suspension, although suspensions from 2–6 games can also be imposed; 10-game suspension for second offense, and a minimum of 2-year ban for third offense
- Beta blockers are prohibited in-competition in specified sports, particularly in target-shooting sports.

**Effectiveness:** Effectiveness of drug testing in preventing drug abuse by athletes is difficult to evaluate. In the 2012 NCAA study, 51.4% surveyed athletes agreed that drug testing by individual colleges has deterred college athletes from using drugs, and 55.2% agreed that NCAA drug testing has deterred college athletes from using drugs. It is difficult to reconcile disparity between positive drug tests (<1% at the Olympics and 2.5% by the NCAA), and a larger prevalence of drug use is presumed in athletes. Numerous moral and ethical questions remain to be answered about drug testing, particularly for college athletes. Courts have upheld legality of high school testing.

**Announced vs. NAN testing:** Limitations of testing only at competitions are obvious, and sports organization have moved to NAN testing to deter drug use. Fig. 26.2 demonstrates the distribution of in-competition to out-of-competition testing conducted by the WADA.

**Legal issues:** Evolving aspect of law varies according to state, and many issues have yet to be fully resolved; it is prudent to consult local legal experts before embarking on a testing program. Several landmark cases have involved drug testing.

- 1987: As a result of a suit filed by Stanford athletes, the court ruled that drug testing violates student-athletes' right to privacy. Overturned in 1994 by US Supreme Court.
- 1994: US Supreme Court rules that random drug tests violate privacy rights of University of Colorado athletes, trainers, and cheerleaders.
- 1995: US Supreme Court rules that urine drug screening of junior high and high school student-athletes is allowable. Court states that minors are not protected by Fourth Amendment rights to privacy like adults, and "individualized suspicion" is not necessary to conduct drug testing of athletes.
- 2002: US Supreme Court ruled in the Earls case that students possess limited privacy rights, the intrusion of drug testing was not significant, and the government had a legitimate interest in deterring drug use.

**Guidelines for drug testing:** The NCAA has suggested guidelines regarding drug-testing protocols for member institutions. Although not obligated to institute separate programs, universities must follow their own policies.

**Alternative matrices:** Drug testing has traditionally involved urine testing, but need for witnessed urination and the possibility of substitution and adulteration has led to exploration of other methods. Blood testing is now used for the ABP, hGH

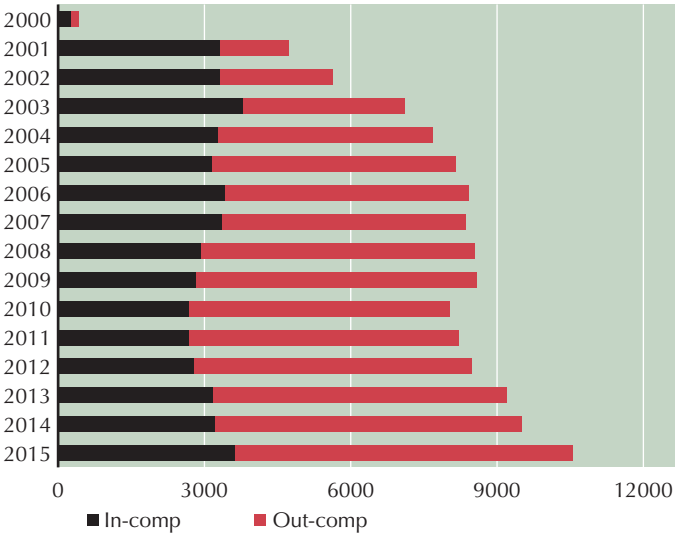


**Figure 26.2. Distribution of in-competition to out-of-competition testing conducted by WADA.** (Data from US Anti-Doping Agency.)

testing, and certain EPO tests; in fact, certain surveys have suggested that athletes prefer blood tests to observed urine collection. In addition, there is work being done with oral fluids, sweat testing, and hair analysis. All these have limitations and would have to withstand forensic scrutiny.

**Nonanalytical rule violations** have allowed antidoping organizations to apply sanctions in cases where there is no positive doping sample, but where there may still be evidence that a doping violation has occurred (e.g., through a combination of three missed tests, whereabouts failures, longitudinal testing, or evidence brought forward through an investigation).

## THERAPEUTIC USE EXEMPTIONS (TUES)

- Approval given to an athlete to use a medication that is normally prohibited in their sport by the restrictions of a doping control program
- Must be applied for before competition during which it will be needed
- Retroactive approval is considered only in case of emergency treatment of an acute condition or exceptional circumstances.
- An athlete must have a documented need for the prohibited substance or method and would experience serious adverse consequences if the use of the medication or method was not allowed.
- There must be no reasonable alternatives to the use of the prohibited substance or method.
- Approval of the TUE would not allow use of a prohibited substance or method to enhance performance above what would be found if athlete returned to normal health.

## DRUG EDUCATION

- **Education of athletes** can be used to deter drug abuse. However, to be effective, it must be started at a young age.
- Educational intervention is probably best suited for deterring use of **recreational drugs**, which usually have a negative impact on performance.
- Although education may alert athletes to the risks of **PEDs**, positive benefits of improved performance make drug testing a necessary component of deterrence.
- Use of PEDs violates the rules of most sports and is thus considered "cheating." Most athletes conform to the rules of sport, and reminding athletes about consequences of cheating (e.g., social, family, and public repercussions) can be an effective educational tool for deterrence.
- **Substance abuse patterns in athletes are constantly changing** based on current social practices and technological advances. Sports medicine professionals have obligation to keep abreast of such changes.

## EBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eAppendix 26-1, Cocaine
- eAppendix 26-2, Nonsteroidal anti-inflammatory drugs (NSAIDs)
- eAppendix 26-3, Gamma-hydroxybutyrate and derivatives

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# EATING DISORDERS IN ATHLETES

# 27

Lauren E. Costello

## ANOREXIA NERVOSA (AN)

### Diagnosis Criteria

- Essential features and diagnostic criteria (Fig. 27.1):
  - Restriction of energy intake relative to requirements, leading to a significantly low body weight
    The World Health Organization (WHO) and Centers for Disease Control (CDC) consider a body mass index (BMI) of ≤17 kg/m$^2$ to indicate significantly low weight. An adult with BMI ranging between 17 and 18.5 kg/m$^2$ or even >18.5 kg/m$^2$ (lower limit of normal weight in adults) might be considered to have a significantly low weight if supported by clinical history or other physiologic information. For children and adolescents, a BMI below the 5$^{th}$ percentile suggests that the individual is underweight.
  - Intense fear of gaining weight or becoming fat that is not alleviated by weight loss.
  - Disturbance in the way in which one's body weight or shape is experienced.
- Severity (based on BMI):
  - Mild: ≥ 17 kg/m$^2$
  - Moderate: 16–16.99 kg/m$^2$
  - Severe: 15–15.99 kg/m$^2$
  - Extreme: <15 kg/m$^2$
- **Characteristics**: Strong need for control, concrete thinking, limited social spontaneity, perfectionism, and preoccupation with and/or restriction of food (Fig. 27.2)
- **Comorbidity:** Depressive, obsessive-compulsive, personality, bipolar, and anxiety disorders
- For more information, see **American Psychiatric Association DSM-5 Guidelines.**

### Medical Complications of AN

**Cardiovascular:**

- **Hypotension and bradycardia:**
  - Systolic blood pressures as low as 70 mmHg
  - Sinus bradycardia: HR as low as 30–40 beats/minute
  - Attributed to decrease in basal metabolic rate
- **Arrhythmias, EKG:**
  - Sinus bradycardia
  - ST-segment elevation
  - T-wave flattening
  - Low voltage
  - Long QT interval increases the risk of sudden death
- Cardiomyopathy: from aggressive refeeding or ipecac use

**Endocrine and Metabolic:**

- **Amenorrhea:** Results from disorders in the hypothalamic–pituitary–ovarian axis (Fig. 27.3). Levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are low despite low levels of estrogen. Marginal energy intake coupled with high energy expenditure due to intense physical activity may result in neuroendocrine adaptations, leading to an overall metabolic deficit. Leptin and ghrelin levels may be important indicators of nutritional status; may contribute to functional hypothalamic amenorrhea
- **Serum chemistry:** Hypokalemia, hyponatremia, hypomagnesemia, hypophosphatemia (particularly with refeeding), hypercholesterolemia, and elevation of hepatic enzymes
- **Euthyroid sick syndrome:** Decrease in T3/T4 levels; no treatment is required
- **Osteopenia/osteoporosis:** Undernutrition and its metabolic consequences directly reduce bone turnover and bone formation. Estrogen deficiency is only one factor that contributes to bone loss by inhibiting osteoclastic bone resorption. In addition, low BMI is the consequence of decreased lean muscle mass, low insulin-like growth factor-1 (IGF-1), relative hypercortisolemia, and alterations in hormones impacted by energy availability—specifically higher leptin and lower ghrelin levels. Some improvement in bone mineral density (BMD) occurs with weight gain and resumption of menses, but the rate is lower than that in normal-weight, age-matched controls, and "catch-up" does not always occur.
- Hypothermia
- Hypoglycemia
- Decreased resting energy expenditure

**Gastrointestinal:** Constipation, delayed gastric emptying, and decreased intestinal motility
**Hematologic:** Anemia, leucopenia, and thrombocytopenia
**Integumentary:** Dry skin and hair and lanugo
**Neurologic:** Cerebral atrophy and ventricular enlargement
**Reproductive:** Infertility or low-birth-weight infant

## BULIMIA NERVOSA (BN)

### Diagnosis Criteria

- Essential features and diagnostic criteria:
  - Recurrent episodes of binge eating: characterized by both of the following:
    - Eating, in a discrete period of time (e.g., within a 2-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time
    - A sense of lack of control over eating during the episode
  - Recurrent, inappropriate compensatory behaviors to prevent weight gain: self-induced vomiting; laxative misuse, diuretics, or other medications; fasting; and excessive exercise
  - On an average, both binge eating and inappropriate compensatory behaviors occur at least once a week for 3 months.
  - Self-evaluation is unduly influenced by body shape and weight.
  - The disturbance does not exclusively occur during episodes of AN.
  - Severity:
    - Mild: An average of 1–3 episodes of inappropriate compensatory behaviors per week
    - Moderate: 4–7 episodes/week
    - Severe: 8–13 episodes/week
    - Extreme: ≥14 episodes/week
- **Characteristics**: In general, normal weight but may be slightly overweight or underweight; individual may conceal food or hoard food for binges, usually secretive, either planned or spontaneous; low self-esteem, anxiety/depressive symptoms, and substance abuse
- **Comorbidity:** Mood and bipolar disorders; personality disorders, specifically borderline personality disorder; substance

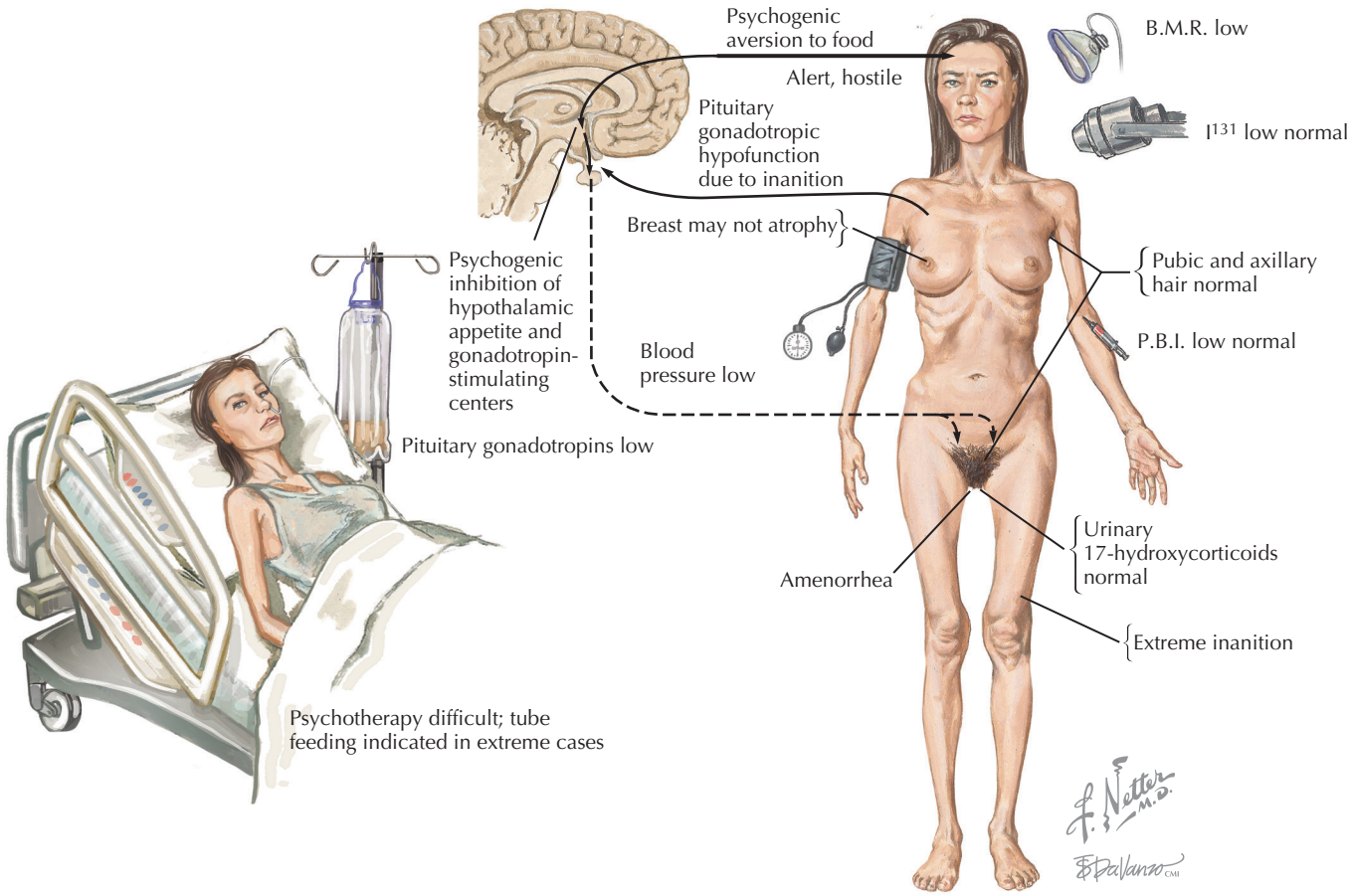


**Figure 27.1.** Anorexia nervosa: a psychoneurotic disorder.

abuse/dependence; and increased incidence of attempted suicide, stealing/kleptomania, and promiscuity
- For more information, see **American Psychiatric Association DSM-5 Guidelines.**

## Medical Complications of BN

**Cardiovascular:** Arrhythmias and hypertension (with diet pills)
**Endocrine and metabolic:** Menstrual irregularities, *less common than in patients with AN*; electrolyte imbalance—compulsive vomiting, hyponatremia, hypokalemia, hypomagnesemia, and metabolic alkalosis; excessive vomiting alone—hypochloremic metabolic alkalosis, which also indicates excessive laxative use
**Gastrointestinal:** Enlarged salivary glands leading to mildly elevated amylase, constipation, gastritis, esophageal dysmotility patterns (GERD and rarely, Mallory–Weiss or gastric tears), and postbinge pancreatitis
**Integumentary:** Russell's sign: scarring and calloused areas on the dorsum of the index and middle finger from self-induced vomiting
**Neurologic:** Cerebral hemorrhage (with diet pills)
**Orofacial:** Dental caries, dental erosion, and enlarged parotid glands
**Respiratory:** Pneumomediastinum

# BINGE EATING DISORDER

## Diagnosis Criteria

- Recurrent episodes of binge eating as described under BN.
- The binge eating episodes are associated with three or more of the following patterns:

**Figure 27.2.** Distorted body image.

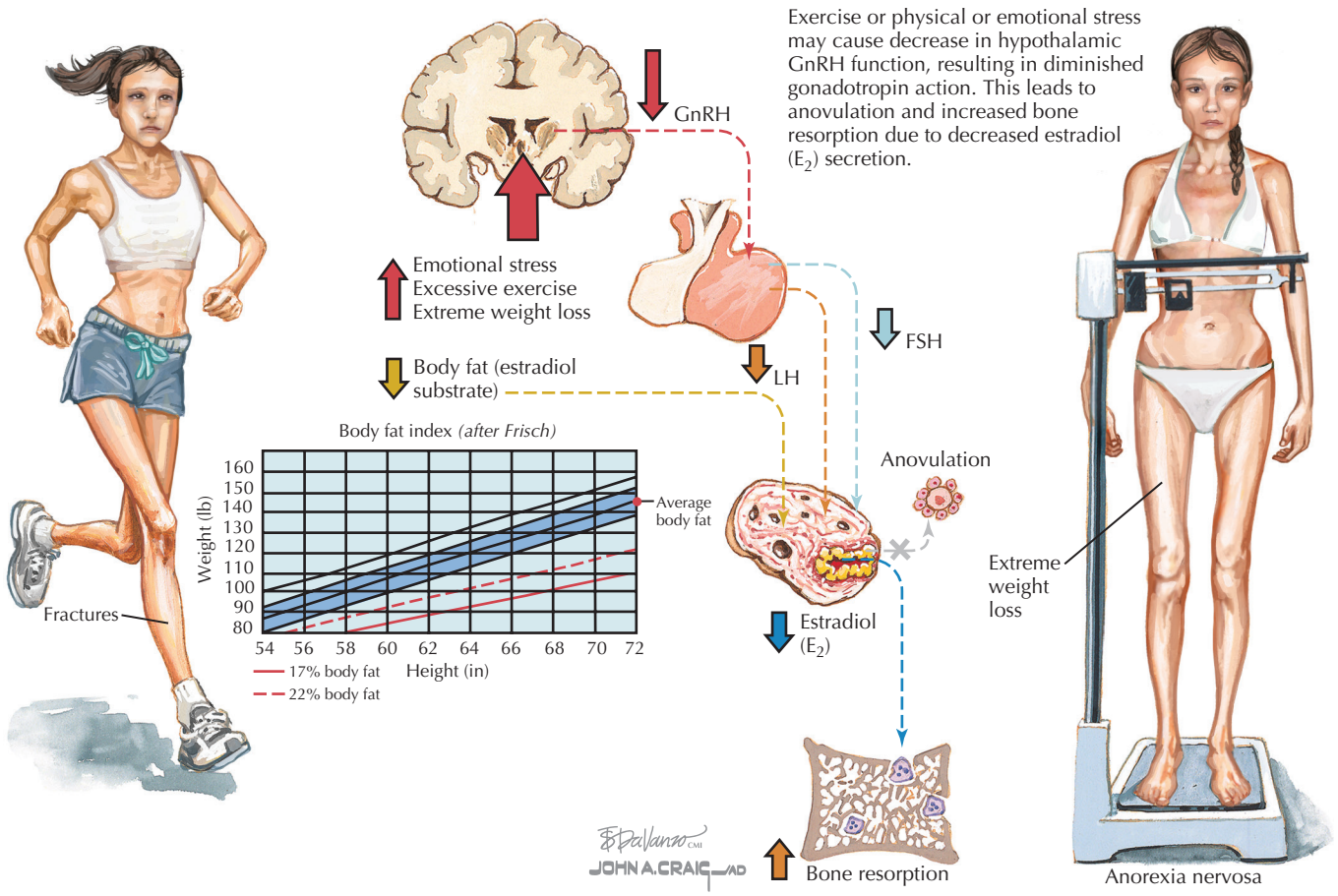


**Figure 27.3.** Osteoporosis associated with amenorrhea.

- Eating much more rapidly than normal
- Eating until feeling uncomfortably full
- Eating large amounts of food when not feeling physically hungry
- Eating alone because of feeling embarrassed by how much one is eating
- Feeling disgusted with oneself, depressed, or very guilty afterward

- Marked distress regarding the binge
- On an average, the binge occurs at least once/week for 3 months.
- The binge is not associated with recurrent, inappropriate compensatory behavior.
- For more information, see **American Psychiatric Association DSM-5 Guidelines.**

## OTHER SPECIFIED EATING OR FEEDING DISORDERS

### Examples

- **Atypical AN:** Criteria for AN met except that despite weight loss, the individual's weight is within or above the normal range
- **BN (of low frequency and/or limited duration):** Criteria for BN met except on an average, behaviors occur <1 time/week and/or for <3 months
- **Binge eating disorder** (of low frequency and/or limited duration): Criteria for BE met except on an average, the binge eating occurs <1 time/week and/or for <3 months
- **Purging disorder:** Recurrent purging behaviors in the absence of binge eating
- **Night eating syndrome:** Recurrent episodes of night eating, as manifested by eating after awakening from sleep or excessive food consumption after the evening meal
- **Repeatedly chewing and spitting out, but not swallowing, large amounts of food**
- **Anorexia athletica:** fear of weight gain although lean
  - Weight is ≤5% of the expected weight
  - Muscular development maintains weight above anorexic threshold
  - Restricted caloric intake—may be broken by binges
  - Excessive or compulsive exercise
  - Menstrual dysfunction—may include delayed puberty
  - GI complaints

## PREVALENCE

- AN: Lifetime prevalence, 0.5%–1%
  The crude mortality rate (CMR) is approximately 5% per decade.
- Most individuals experience remission within 5 years of presentation, but remission rates may be lower in individuals who are hospitalized.
- BN: Lifetime prevalence, 1%–5%; CMR is approximately 2% per decade
- Study of elite athletes: overall prevalence of eating disorders (EDs) of 13.5% in elite athletes and 4.6% in a control group of nonathletes
- Male versus female: ED among college men ranges 4%–10% and includes AN, BN, and binge eating disorder. Men represent

approximately 10% of AN cases. Men are more concerned about weight, body shape, and strength and believe that they need to be lean and muscular; being thin equals faster performance. Muscle dysmorphia is a subtype of body dysmorphia disorder characterized by preoccupation with appearance of muscularity; manifestations include excessive and compulsive weight lifting, abnormal eating patterns designed to enhance muscularity, and use/abuse of supplements including anabolic steroids.

- Increased incidence ED in "lean sport" athletes: e.g., gymnastics, ballet, figure skating, and distance running. Men competing in sports such as bodybuilding, wrestling, and light-weight crew are also at a greater risk.

## RISK AND TRIGGER FACTORS IN ATHLETIC POPULATION

- Early dieting, injuries, and participating in "lean sports" or weight-controlled sports
- The "athletic personality"—characteristics include goal orientation, perfectionism, and compulsiveness
- Extreme exercise appears to be a risk factor for developing AN, particularly when combined with dieting. Exercise is excessive when it interferes with important activities, occurs at inappropriate times or in inappropriate settings, or when an individual continues to exercise despite injury or other medical complications and has been advised to refrain (Fig. 27.2).

## TREATMENT

### Medical Monitoring/Treatment

- Monitor weight, blood pressure, pulse, electrolytes, and behaviors (Box 27.1).
- For underweight patients, realistic weight gain targets are 2–3 pounds/week for hospitalized patients and 0.5–2 pounds/week for outpatients.
- No approved indication for of bisphosphonates in patients with amenorrhea and low BMD. Consider daily supplementation with calcium (100–1500 mg) and vitamin D (600–800 IU).
- Hormone Replacement Therapy (HRT): Although oral HRT is frequently prescribed to improve bone density, no good supporting evidence exists either in adults or in adolescents to demonstrate its efficacy; may be related to decreases in IGF-1 and/or androgens with oral estrogen administration. A recent study revealed that using 100 ugm of transdermal estrogen (does not decrease IGF-1 production) with 10 days of oral progesterone every 1–2 months increases spine and hip BMD in mature adolescent girls/women (those with a bone age [BA] of ≥15 years). The same study utilized escalating doses of oral ethinyl estradiol (to mimic the early pubertal rise in estrogen) to treat immature (BA < 15 years) anorectic girls, which also resulted in increases in BMD over 18 months. These small doses of oral estrogen do not suppress IGF-1. Ongoing RCTs are evaluating whether a combination of estrogen and IGF-1 improves BMD in adolescent girls with AN. Before considering estrogen supplements, attempts must be made to increase weight and achieve resumption of normal menses naturally.
- Weight gain should not be excessive: rapid refeeding can lead to cardiovascular collapse, hypophosphatemia, and electrolyte abnormalities. Increase daily caloric intake by 200–300 kcal every 3–5 days until a sustained weight gain of 1–2 pounds/week is achieved.
- Patients with history of purging behaviors should be referred for a dental examination.
- Weight-bearing exercises increase BMD in women recovering from AN. However, recent research suggests a negative effect of moderate weight-bearing exercises on BMD during the time of active EDs. Activity should be adapted according to food intake and energy expenditure of the patient; occasionally, activity may need to be decreased or stopped.
- IGF-1: Administration of IGF-1 in the form of rhIGF-1 causes increased levels of bone formation. This treatment is experimental; there are ongoing studies to determine the impact of long-term IGF-1 administration on BMD in adolescents with AN.

### Additional Treatment

- Multidisciplinary team: Including a psychologist, psychiatrist, physician, and nutritionist trained in subspecialty of ED; ideally, include certified athletic trainer; consider others: e.g., coaches, teammates, and family members
- Medications:
  - AN: Selective serotonin reuptake inhibitors (SSRIs) are ineffective in accelerating weight gain but may be used to treat comorbidities (e.g., depression, anxiety, or OCD). Atypical antipsychotic agents at low doses (e.g., olanzapine at 2.5–10 mg/day) may improve weight gain, depression, and obsessive thoughts.
  - BN: Fluoxetine 60 mg daily is FDA approved. Patients show decrease in binge eating/purging frequency but rarely attain abstinence.
  - Binge eating disorder: SSRIs have proven useful in both decreasing binge frequency and improving symptoms of depression and anxiety.
- Psychotherapy:
  - Cognitive behavioral therapy (CBT): one of the more effective treatment options for acute episodes of BN
  - Psychodynamic/psychoanalytical approaches may be incorporated once bulimia symptoms improve. Family therapy should be considered whenever possible.
  - Treatment of patients with acute AN with low weight should be supportive and aimed at weight restoration. In addition, traditional family therapy may be useful. Once weight is reasonably restored, more structured psychotherapy is useful, particularly CBT and group therapy. Art therapy, yoga, and meditation are useful adjuncts. Family-based therapy or the Maudsley method emphasizes separating the patient from the illness and assisting the family in addressing the issues of AN, including supervision of meals, without attacking or judging their child. Studies support its use, particularly in adolescent patients with AN.

### BOX 27.1 LABORATORY ASSESSMENTS FOR PATIENTS WITH EATING DISORDERS

**Laboratory Studies**

- CBC with differential
- Complete metabolic panel
- Liver function tests
- Thyroid function tests
- HCG, FSH, LH, prolactin, and estradiol
- ESR
- Drug screen
- Urinalysis
- Amylase (if purging)

**Other Studies**

- ECG
- Bone density test (DEXA) (if indicated)
- Echocardiogram (if indicated)
- Chest or abdominal radiography (if indicated)

### BOX 27.2 INDICATIONS FOR HOSPITALIZATION

- Adults: heart rate <40 bpm
- Children: heart rate <50 bpm
- Adults: blood pressure <90/60 mmHg
- Children: blood pressure <80/50 mmHg
- Temperature <36.1°C (97.0°F)
- Potassium <3 mmol/L
- Symptomatic hypoglycemia
- Severe dehydration
- Weight <75% of expected weight
- Rapid weight loss of several kilograms within a week
- Lack of improvement or rapid worsening while in outpatient treatment

## Level of Care

- Treatment options include outpatient, partial hospitalization (full-day outpatient care), residential, or inpatient hospitalization (Box 27.2). See full criteria in Recommended Readings #1.
- Sports participation and symptomatic athletes:
  - An excellent resource to help guide return-to-play decision-making is the "2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad." The committee formulated a scoring system to evaluate "magnitude of risk" resulting in a cumulative risk score (Fig. 27.4), which would then be utilized to determine clearance-to-participate decisions (Fig. 27.5). The coalition recommends complete restriction from training and competition for all female athletes with BMI <16 kg/m$^2$ or moderate to severe BN (purging >4 times/week). They then go on to say that future participation depends on treatment of their EDs, including attaining a BMI of >18.5 kg/m$^2$.
  - The IOC also convened an expert panel and published their own consensus statement in 2014: "The IOC consensus statement: Beyond the Female Athlete Triad—Relative Energy Deficiency in Sport (RED-S)." These have been subdivided into 3 risk categories: high risk, moderate risk, and low risk. Utilizing these risk categories along with additional modifiers, a "return-to-play" strategy was developed. The IOC recommends that athletes in the "high-risk" category should not be cleared to participate in sports. The criteria in the high-risk category are broader and less defined and do not include any specific BMI or purging severity. The panel suggests that these criteria can also be used to evaluate male athletes for ED/disordered eating (DE).
  - These statements are guidelines to help healthcare providers evaluate and treat athletes with ED/DE. Ultimately, decisions regarding sport participation while a student-athlete remains symptomatic will need to be made on a case-by-case basis. It is reasonable to use a BMI of 18 as an indicator for further evaluation and screening for EDs. Additional important components of evaluation include, but are not limited to, DE behavior (Box 27.3), BMI, menstrual status, history of stress fractures, medical stability, and prior history of ED as well as other psychological diagnoses. Early identification and early intervention have been shown to shorten recovery from EDs.
  - A majority of athletes with DE behaviors and medical consequences of such behaviors will not be categorically restricted from all training and competition. For moderate- or high-risk athletes, it is important to involve all members of the multidisciplinary team not just in the treatment but also in level-of-play decisions. It is encouraged that such athletes sign a written contract outlining treatment requirements and level-of-activity parameters (eForm 27.1). As an

| Risk Factors | Magnitude of Risk<br>Low Risk<br>0 points each | Moderate Risk<br>1 point each | High Risk<br>2 points each |
|---|---|---|---|
| *Low EA with or without DE/ED* | ☐ No dietary restriction | ☐ Some dietary restriction‡; current/past history of DE | ☐ Meets DSM-V criteria for ED* |
| *Low BMI* | ☐ BMI ≥18.5 **or** ≥90% EW** or weight stable | ☐ BMI 17.5<18.5 **or** <90% EW **or** 5 to <10% weight loss/ month | ☐ BMI ≤17.5 **or** <85% EW **or** ≥10% weight loss/month |
| *Delayed Menarche* | ☐ Menarche <15 years | ☐ Menarche 15 to <16 years | ☐ Menarche ≥16 years |
| *Oligomenorrhea and/or Amenorrhea* | ☐ >9 menses in 12 months* | ☐ 6–9 menses in 12 months* | ☐ <6 menses in 12 months* |
| *Low BMD* | ☐ Z-score ≥–1.0 | ☐ Z-score –1.0*** <–2.0 | ☐ Z-score ≤–2.0 |
| *Stress Reaction/ Fracture* | ☐ None | ☐ 1 | ☐ ≥2; ≥1 high risk or of trabecular bone sites† |
| *Cumulative Risk (total each column, then add for total score)* | ___ points + | ___ points + | ___ points = ___ Total Score |

**Figure 27.4.** Female athlete triad: cumulative risk assessment. The cumulative risk assessment provides an objective method of determining an athlete's risk using risk stratification and evidence-based risk factors for the Female Athlete Triad. This assessment is then used to determine an athlete's clearance for sport participation (Figure 27.5). ‡Certain dietary restrictions, as evidenced by self-report or low/inadequate energy intake on diet logs; *current or past history **≥90% EW absolute BMI cut-offs should not be used for adolescents. ***Weight-bearing sport; †High-risk skeletal sites associated with low BMD and delay in return to play in athletes with one or more components of the Triad include stress reaction/fracture of trabecular sites (femoral neck, sacrum, or pelvis). *BMD,* Bone mineral density; *BMI,* body mass index; *DE,* disordered eating; *EA,* energy availability; *ED,* eating disorder; *EW,* expected weight. (From Joy E, De Souza MJ, Nattiv A, et al. 2014 female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad. *Curr Sports Med Rep.* 2014;13[4]:219-232.)

### BOX 27.3 RECOMMENDED NUTRITIONAL SCREENING QUESTIONS

Are you on a special diet or do you avoid certain foods or food groups?
Are you trying to or has anyone recommended that you gain or lose weight?
Do you make yourself sick (vomit) because you feel uncomfortably full?
Do you worry that you have lost control over how much you eat?
Have you recently lost more than 15 lbs in a 3-month period?
Do you believe yourself to be fat when others say you are thin?
Would you say that food dominates your life?
Have you ever been told you have had an eating disorder?
Do you worry about your present weight?

| | Cumulative Risk Score* | Low Risk | Moderate Risk | High Risk |
|---|---|---|---|---|
| *Full clearance* | 0–1 point | ☐ | | |
| *Provisional/ limited clearance* | 2–5 points | | ☐ Provisional clearance<br>☐ Limited clearance | |
| *Restricted from training and competition* | ≥6 points | | | ☐ Restricted from training/ competition-provisional<br>☐ Disqualified |

**Figure 27.5.** Female athlete triad: clearance and return-to-play (RTP) guidelines by medical risk stratification. *Cumulative Risk Score determined by summing the score of each risk factor (low, moderate, and high risk) from the Cumulative Risk Assessment (Figure 27.4). Clearance/RTP status for moderate- to high-risk athletes for the Triad: provisional clearance/RTP—clearance determined from risk stratification at the time of evaluation (with possibility for status to change over time depending on the athlete's clinical progress); limited clearance/RTP—clearance/RTP granted, but with modification in training as specified by the physician (with possibility for status to change depending on clinical progress and newly gathered information); restricted from training/competition (provisional)—athlete not cleared or able to RTP at the present time, with clearance status re-evaluated by the physician and the multidisciplinary team with clinical progress; disqualified—not safe to participate at the present time. Clearance status to be determined at a future date depending on clinical progress, if appropriate. The Consensus Panel recommends that athletes diagnosed with anorexia nervosa who have a body mass index (BMI) <16 kg/m$^2$ or with moderate-to-severe bulimia nervosa (purging >4 times/week) should be categorically restricted from training and competition. Future participation is dependent on treatment of their eating disorder, including ascertainment of BMI >18.5 kg/m$^2$, cessation of binging and purging, and close-interval follow-up with the multidisciplinary team. (Reprinted with permission from Joy E, De Souza MJ, Nattiv A, et al. 2014 female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad. *Curr Sports Med Rep.* 2014;13[4]:219-232.)

athlete's health status improves, his/her level of activity may be increased. If no progress is observed in the given time period, the level of play may have to be decreased or completely restricted. The desire of athletes to continue to compete in their sport can aid in their recovery. However, there are situations where the sport may only contribute to the ED, and the athlete will need to be removed from the sport. The ultimate goal is to aid the athlete in recovering back to health, even if it requires temporarily limiting or completely removing an athlete from the sport.

## PREVENTION

- Do not overly focus on effects of lower body weight on athletic performance.
- Be aware of behaviors associated with EDs—constant preoccupation with food/calories/dieting; purposeless, excessive physical activity not part of training; extreme weight fluctuations; secretive eating; avoidance of eating with others; and vomitus in restroom.
- Educate coaches regarding signs and symptoms of ED as well as the role they may play in helping or hindering development of EDs, particularly, tying performance to weight.
- Encourage coaches to *avoid weighing* their athletes.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eForm 27.1, 2014 Female Athlete Triad Coalition consensus Statement on Treatment and Return to Play of the Female Athlete Triad

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

- eForm 27.1, 2014 Female Athlete Triad Coalition consensus Statement on Treatment and Return to Play of the Female Athlete Triad

**2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad**

_____ (Athlete Name)

The following items are mandatory and must be completed as prescribed. Failure to do so will result in the consequences listed below the requirements. All benefits and consequences are subject to change at any time and at the discretion of the Multidisciplinary Team.

**Multidisciplinary Team:**

_____ (Physician), _____ (Psychotherapist), _____ (Dietitian)

**Requirements:**

☐ Meet with _____ (therapist) 1x per week, or as recommended by therapist.
☐ Meet with _____ (dietitian) 1x per week, or as recommended by dietitian.
☐ Meet with Dr. _____ 1–2x per month, or as recommended by Dr. _____ .
☐ Follow daily meal plan set forth by sports dietitian.
☐ Keep daily workout log updated with specific type, length, and effort.
☐ Weight gain of _____ lbs per week.
☐ Weekly weigh-in with _____ (name team member), or at time intervals of ____ weeks.
☐ Must achieve minimal acceptable body weight of ____ lbs by _____ (date).
☐ After this date, must maintain weight at or above minimal acceptable body weight.
☐ Limit of _____ workout sessions per week with no one session being more than _____ minutes in length.
All activity counts (e.g., biking, running, weight lifting, and swimming).

**Benefits:**

If ALL requirements are met then clearance to participate in team activities and use of athletic facilities will:
☐ be granted ☐ continue

**Consequences:**

If ANY requirement(s) are not met, then clearance to participate in team activities and use of athletic facilities will be revoked, and re-instatement will be at the discretion of the team physician and multidisciplinary team

I, _____, have read this contract and all of my questions were answered.

| _____ | _____ | _____ |
|---|---|---|
| Athlete Name | Athlete Signature | Date |
| _____ | _____ | _____ |
| Team Physician Name | Team Physician Signature | Date |

*Reprinted with permission from DeSouza, MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. Br J Sports Med 2014;48:289.*

## RECOMMENDED READINGS


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# OVERTRAINING

# 28

Thomas M. Howard • Francis G. O'Connor

## INTRODUCTION

- Overtraining syndrome (OTS) is a medical disorder occurring in athletes, and it is complicated by several diagnostic and therapeutic challenges. Current research in this area is limited by a small number of studies and inconsistent results. Several researchers have concluded the following: there are poorly established diagnostic criteria; there are significant confounding influencers, including illness, injury, menstruation, and unique training methods for different sports, and it is difficult, if not impossible, to establish controls and/or laboratory models to study the illness.
- This chapter will review the epidemiology and key terminology as well as describe the proposed pathophysiology of OTS. In particular, we will discuss the clinical presentation, diagnosis, management, and prevention of this disorder.

## EPIDEMIOLOGY

- OTS is an issue that has plagued athletes, trainers, coaches, and clinicians for generations. As evidenced by this quote from Dr. D. C. Parmenter, this ailment is not new to the medical literature:

*"Overtraining or staleness is the bug-a-boo of every experienced trainer… a condition difficult to detect and still more difficult to describe…evaluation should focus on training load, nutrition, sleep and rest, competition stress and psychological state."*

**Parmenter**

- The current literature on OTS has limited insight into the incidence and prevalence of this disorder. However, observational data and clinical experience suggest that OTS is not rare. An early study reported that 65% of elite competitive swimmers had experienced staleness at some time in their career. A subsequent survey study of endurance runners described a prevalence rate of 10% during one training cycle and a lifetime risk of 64% and 60% among male and female runners, respectively. Athletes who have experienced OTS appear more likely to relapse: a study of college swimmers reported that 91% of swimmers who experienced OTS in their freshman year suffered a repeat episode during the subsequent year as opposed to 34% of those who did not experience an initial episode.

## TERMINOLOGY

**Training:** A series of stimuli used to stress or displace homeostasis to provide stimulation for adaptation. Training involves progressive overload in an effort to improve performance in a sport or activity. Training for success involves a balance between achieving peak performance and avoiding negative consequences of overtraining.

**Adaptation:** Physiologic response to stress that results in an adjustment in function or dimension of an organism

**Recovery:** The period of time following a training stimulus when work capacity returns to prestimulus levels. If recovery time is optimal, supercompensation results, whereas excessive training, without rest, can result in a decline in performance (Fig. 28.1). Available literature describes four components of recovery: hydration and nutrition, sleep and rest, relaxation and emotional support, and stretching and active rest. Recovery that is inadequate results in fatigue. Fatigue may be classified as either pathologic or physiologic.
  - Physiologic fatigue includes categories such as insufficient sleep, nutritional, jet lag, pregnancy, and training-induced (from either excessive competition or overreaching).
  - Pathologic fatigue includes the following categories: medical, including infectious, neoplastic, hematologic, endocrine, toxic, iatrogenic, and psychiatric; chronic fatigue syndrome; and OTS.

**Periodization:** Planned sequencing of increased training loads and recovery periods within a training program

**Acute Fatigue (AF):** The immediate response to overload training; although fatigued, the athlete experiences no perceivable decline in performance

**Functional Overreaching (FOR):** Short-term decrement in performance after a period of overload training usually lasting <2 weeks after a period of overload training

**Non-functional Overreaching (NFOR):** A longer period of performance decline (2 weeks to 2 months) following a period of overload training

**Overtraining Syndrome (OTS):** Maladaptive response to training from an extended period of overload (usually 2 months or more); the result of too severe or a prolonged period of overreaching (see Fig. 28.1). Overtraining is manifested by decreased sport-specific performance and enhanced fatigability, pronounced vegetative complaints, sleep disorder, emotional instability, organ-related complaints without organic disease, overuse injuries, blood chemistry changes, and immune dysfunction.

## PATHOPHYSIOLOGY

### Overview

- The pathophysiology of OTS has not been fully elucidated. There are multiple models/hypotheses that attempt to explain the signs and symptoms of this disorder, including the autonomic imbalance hypothesis, the central fatigue hypothesis, the glycogen depletion hypotheses, and the cytokine hypothesis.
- Fig. 28.2 summarizes the various hypotheses to explain the complexity of this disorder.

### Autonomic Imbalance Hypothesis

The autonomic imbalance hypothesis proposes that underlying imbalances in the autonomic nervous system cause OTS. The initial stage of this proposed mechanism involves negative feedback caused by a surge of catecholamine release during periods of heavy training. This causes a decrease in baseline catecholamine secretion. Second, an increase in metabolism during exercise causes an imbalance among plasma amino acids and alterations in brain neurotransmitter metabolism leading to an increase in the concentrations of aromatic amino acids (phenylalanine, tryptophan, and tyrosine). The resulting increases in hypothalamic tryptophan and cerebral dopamine concentrations cause "metabolic error signals" with inhibitory effects on the sympathetic nervous system. In addition, the increase in core temperature associated with high-intensity training (HIT) may exert inhibitory effects on the sympathetic

centers of the hypothalamus. Third, a neuronal negative feedback system results in down-regulation of catecholamine receptors in the exercising muscles.

## Central Fatigue Theory

The central fatigue theory hypothesizes that OTS is caused by an increase in the synthesis of 5-hydroxytryptomine (5-HT) in the central nervous system (CNS). Extensive exercise results in glycogen depletion in muscles, leading to the use of secondary energy sources by these muscles. The branched-chain amino acids (BCAAs: leucine, isoleucine, and valine) are oxidized to glucose. Concurrently, levels of fatty acids increase as well. Fatty acids compete with tryptophan for albumin-binding sites, leading to an increase in plasma tryptophan. Since both BCAAs and tryptophan use the same transporter to pass through the blood–brain barrier, a decrease in plasma BCAAs and an increase in plasma tryptophan leads to an increase in tryptophan passing through into the CNS. In the brain, tryptophan is converted into the neurotransmitter 5-HT. 5-HT is known to play a role in various neuroendocrine and emotional functions, all of which can be seen with OTS. This connection between OTS and an increase in the free tryptophan/BCAA ratio forms the basis for this hypothesis.

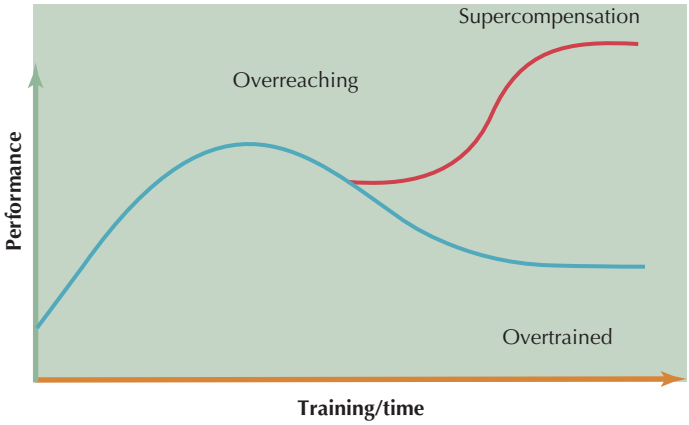


**Figure 28.1.** Hypothetical overtraining syndrome model.

## Glycogen Depletion Hypothesis

- Glycogen is the predominant energy source for moderate to intense exercise.
- According to the glycogen hypothesis, glycogen depletion may lead to overtraining through following three mechanisms.
- Direct effects of glycogen depletion: low levels of muscle glycogen stores may cause muscular fatigue and poor performance.
- Glycogen depletion may also cause increased oxidation of BCAAs, which eventually leads to central fatigue.
- Glycogen depletion may lead to a net negative caloric state, which induces a catabolic state and multiple neuroendocrine changes.

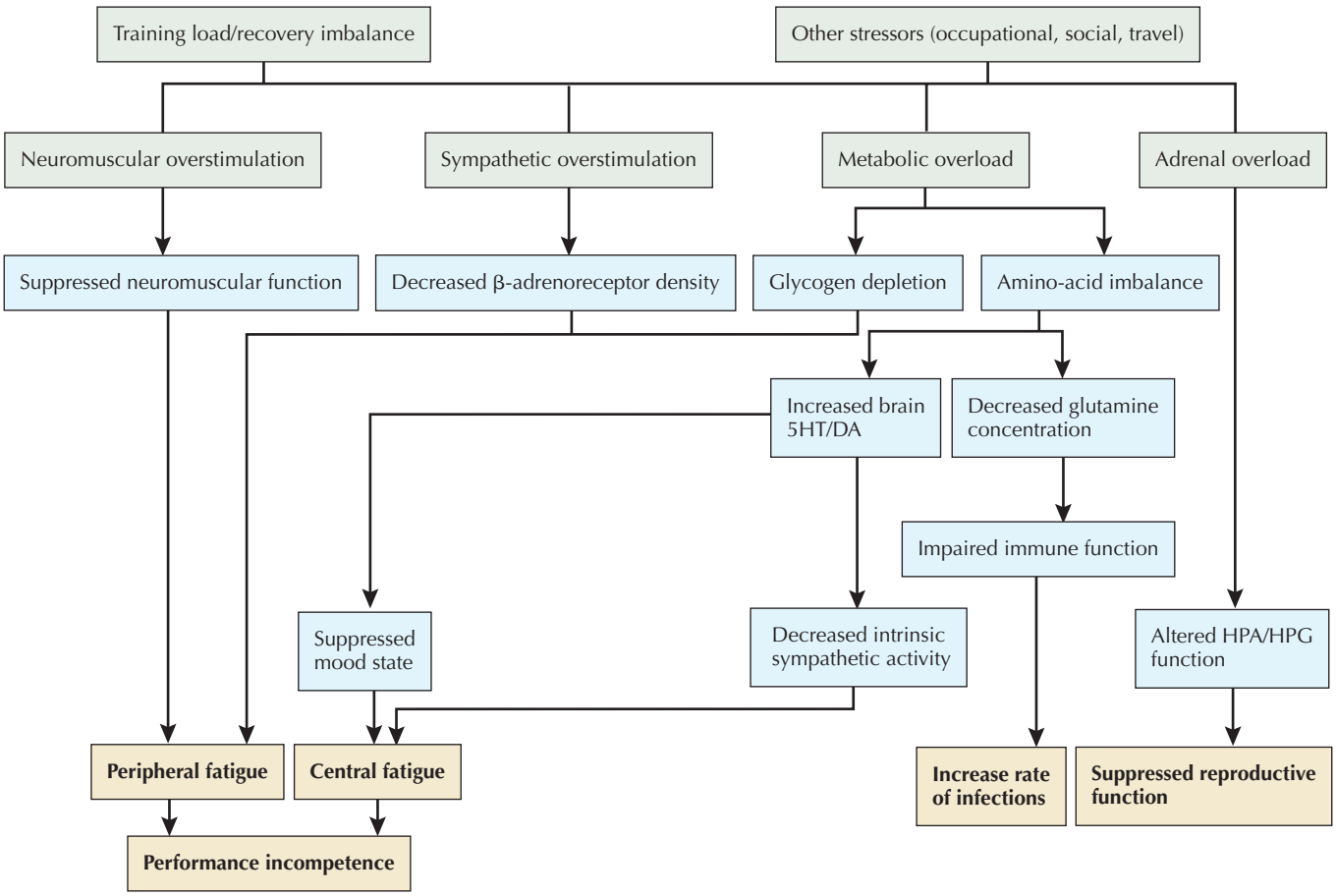


**Figure 28.2.** Genesis of overtraining syndrome. *5-HT*, Serotonin; *DA*, dopamine. (Modified from Foster C, Lehmann M. Overtraining syndrome. In Guten GN, ed: *Running Injuries*. Philadelphia: W.B. Saunders; 1997.)

**TABLE 28.1 CLINICAL PRESENTATION OF OVERTRAINING SYNDROME**

| | |
|---|---|
| **Sport-Specific Performance Complaints** | • Inability to meet prior performance standards<br>• Prolonged recovery time<br>• Decreased coordination<br>• Decreased muscular strength |
| **Physiologic Findings** | • Blood pressure changes<br>• Increased resting heart rate<br>• Weight loss<br>• Increased incidence of injuries<br>• Increased incidence of infections<br>• Amenorrhea |
| **Subjective Complaints** | • Fatigue<br>• Feeling of depression<br>• Anorexia<br>• Hypersomnia/disturbed sleep<br>• Myalgias<br>• Gastrointestinal disturbances<br>• Headaches<br>• Increased irritability<br>• Concentration difficulties<br>• Apathy |

### Cytokine Hypothesis

- The cytokine hypothesis theorizes that chronic tissue injury without regenerative healing leads to a systemic inflammatory and immune response. This systemic inflammatory response leads to increased concentration of interleukins, interferons, tumor necrosis factor, and other proinflammatory factors.
- These factors are thought to act centrally, promoting central fatigue, anorexia, depression, a catabolic state, and changes in the hypothalamic–pituitary–adrenal and hypothalamic–pituitary–gonadal axes.

## CLINICAL PRESENTATION

### Overview

- Although a universal feature of OTS is decreased performance, it may include a broad array of psychiatric, musculoskeletal, cardiovascular, and immunologic symptoms (Table 28.1). However, on account of individual variation, few (if any) athletes exhibit all features of overtraining.
- Training volume, training intensity, or lack of rest, alone, may not explain many or even most cases of OTS. Rather, OTS commonly develops when the cumulative stress imposed on an individual outstrips his/her ability to cope with that stress. Contributing factors can include the training regimen, rest status, nutritional state, concurrent infectious disease, allergic reactions, and financial and relationship stress.

### Psychological Features

- Fatigue
- Loss of appetite or libido
- Anxiety, irritability, or anger
- Insomnia or hypersomnolence
- Depression

### Cardiovascular Features

- Increased first A.M. resting heart rate (increase of 5–10 beats per minute over baseline is suggestive)
- Reduced maximum heart rate and $VO_2$ max
- Decreased stroke volume and altered contractility
- Possible relative decrease in plasma volume

**TABLE 28.2 DIFFERENTIAL DIAGNOSIS OF OVERTRAINING SYNDROME**

| | |
|---|---|
| **Infectious Etiologies** | • Postviral syndrome<br>• Infectious mononucleosis<br>• Lyme disease<br>• Viral hepatitis<br>• Myocarditis |
| **Collagen Vascular Disorders** | • Polymyalgia rheumatica<br>• Systemic lupus erythematosus<br>• Fibromyalgia<br>• Chronic fatigue syndrome |
| **Metabolic** | • Nutritional deficiency<br>• Hypothyroidism or hyperthyroidism<br>• Anemia<br>• Electrolyte disorders |
| **Pharmacologic** | • Alcohol<br>• Caffeine<br>• Illegal or performance-enhancing drugs |
| **Psychiatric** | • Depression<br>• Physical abuse<br>• Sexual abuse<br>• Emotional abuse<br>• Posttraumatic stress disorder<br>• Malingering |
| **Other** | • Cancer<br>• Myopathy<br>• Pregnancy<br>• Sleep deprivation |

### Musculoskeletal Features

- Muscular fatigue at previously tolerated exercise levels
- Decreased performance
- Persistent soreness

### Immunologic Features

- Demonstrated increase in the rate of upper respiratory tract infections in endurance athletes during periods of intense training that are not followed by adequate rest
- Decreased serum and secretory IgA
- Decreased natural killer cell function

## DIAGNOSIS

### Differential Diagnosis

- The diagnosis of OTS is clinically confirmed—there is no currently available confirmatory laboratory test. Accordingly, OTS **is considered a diagnosis of exclusion.**
- The differential diagnosis of overtraining is extremely broad and should be considered before a diagnosis of OTS is established. Infectious, inflammatory, malignant, endocrine, cardiopulmonary disease, renal disease, hematologic disease, myopathies, and psychiatric disease as well as substance abuse should be considered (Table 28.2 and Figs. 28.3 and 28.4).
- A broad laboratory evaluation is not initially recommended but may be necessary over the course of several visits to exclude many of the aforementioned conditions. Fig. 28.5 presents a possible evaluation strategy.

### Laboratory Analysis

- Although various hormonal and hematologic abnormalities have been elucidated in OTS, there is no one characteristic set

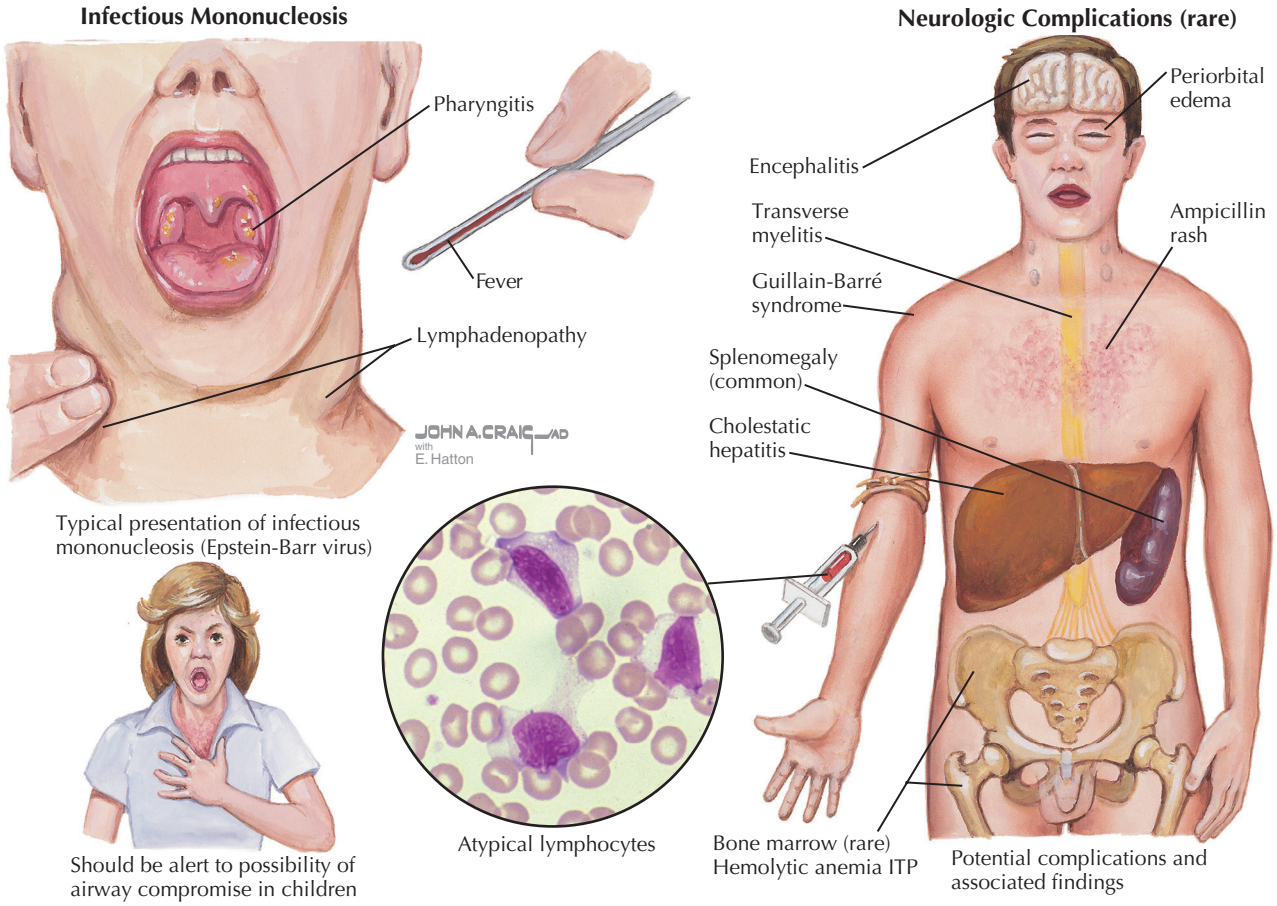


**Figure 28.3.** Infectious mononucleosis.

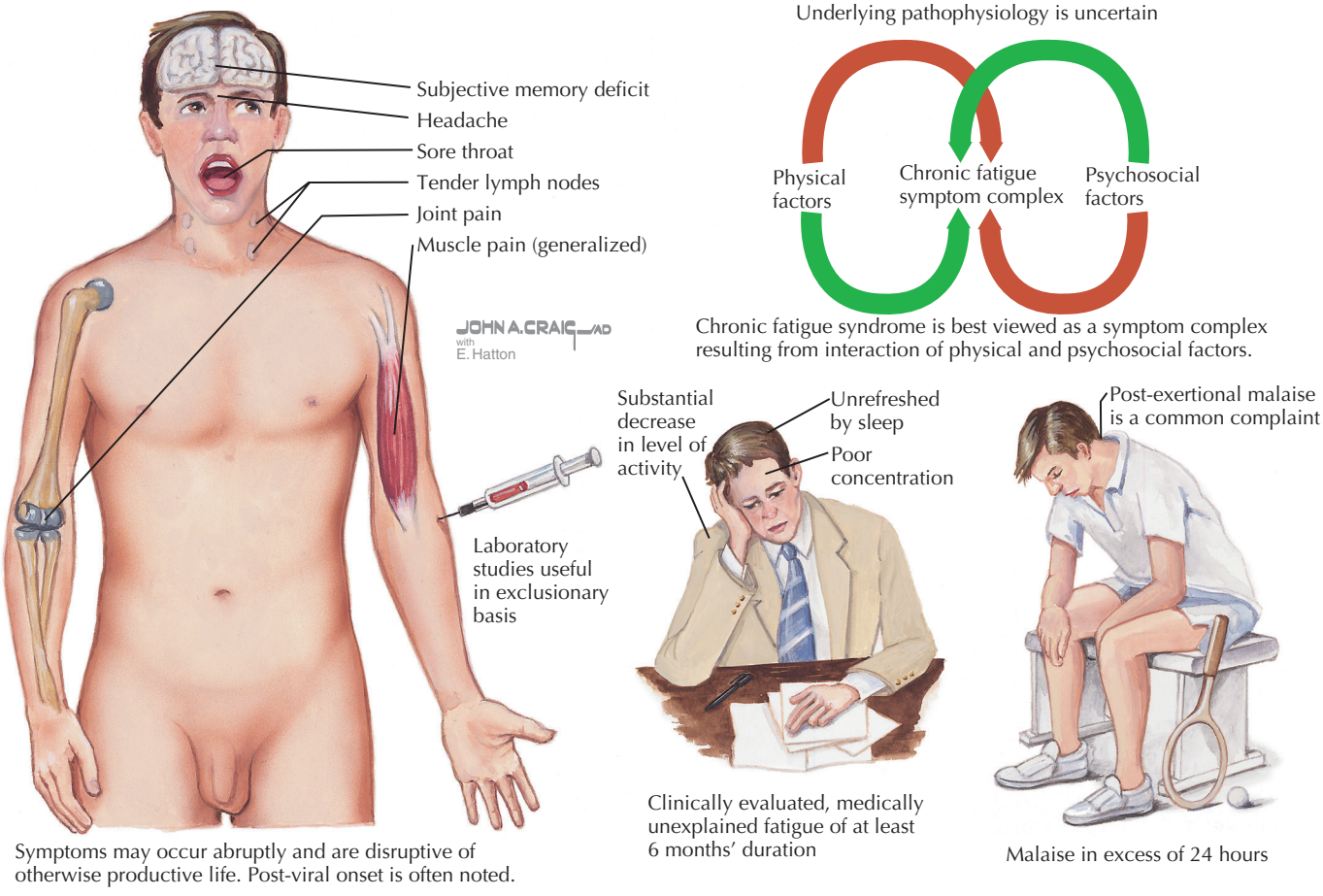


**Figure 28.4.** Signs and symptoms of chronic fatigue syndrome.

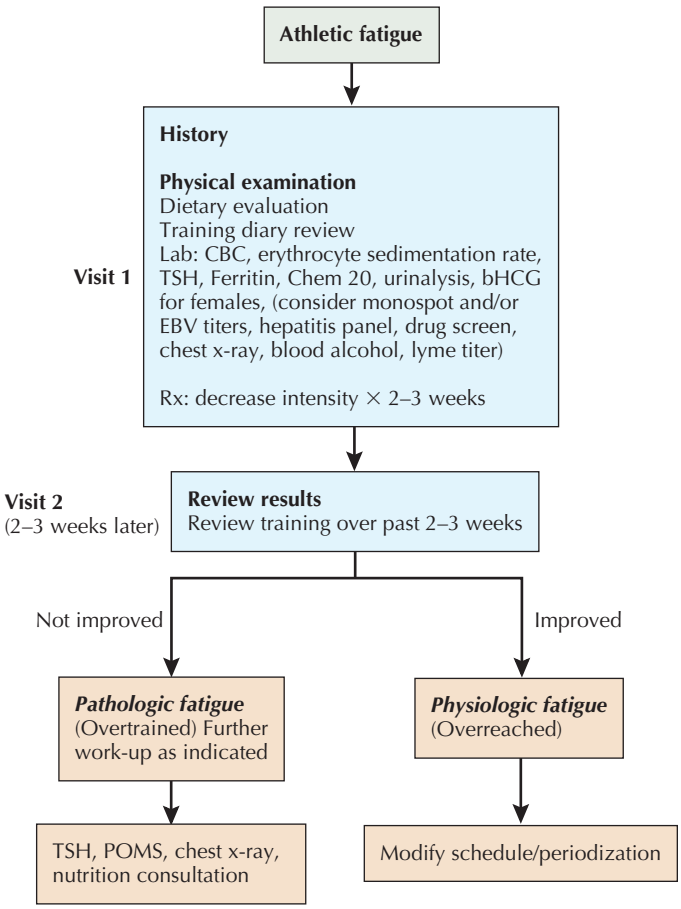


**Figure 28.5.** Proposed clinical evaluation of athletic fatigue.

of abnormalities and many changes may represent points along a continuum.

- Clinical and laboratory markers that have been proven to have poor sensitivity and specificity in evaluating OTS include body mass, resting heart rate and blood pressure, hematocrit, ferritin, creatinine kinase, and hormonal markers.
- Glutamine has recently been a frequent subject of study as a possible marker of overtraining. Glutamine is an amino acid that is depleted in catabolic states such as overtraining, infection, surgery, and acidosis. Moreover, several studies have demonstrated a decline in glutamine levels in overtrained states; however, there are multiple confounding factors, as discussed earlier, and glutamine testing is not yet widely available for practical use.

### Neuropsychiatric Testing

- Many of the earliest symptoms of overtraining are psychiatric, and a number of screening questionnaires have been developed to evaluate these. A commonly used questionnaire that has been validated for OTS screening and risk assessment is the Profile of Mood States (POMS).
- POMS is a survey wherein patients rate 65 adjectives on a 5-point scale in accordance with how well the adjective describes their feelings over the past week. POMS is designed to assess tension/anxiety, depression/dejection, anger/hostility, vigor/activity, fatigue/inertia, and confusion/bewilderment. The test is summarized in one global score, which subtracts the sum of points assessed for positive mood states from that of negative mood states.
- Other commonly used standardized tests include the Total Quality Recovery score by Kentta and Hassmen, the Daily Analyses of Life Demands for Athletes, which is used by Australian Olympic teams, and the Psycho-Behavioral Overtraining Scale, which has been used by certain British athletes.

### Performance Testing

- Preliminary research suggests that a diminished serum lactate concentration following performance testing is a consistent finding in overtrained athletes. However, lactate studies are often limited by the availability of prior baseline data.
- A previous study reported the diagnosis of OTS in athletes by using a two-bout exercise test protocol. The study included 10 underperforming athletes, five of whom were ultimately diagnosed with OTS and five with NFOR. After performing the two-bout maximal exercise protocol (two maximal exercise tests separated by a 4-hour interval), athletes with OTS exhibited reduced maximal lactate concentrations (concentrations ≤8 mmol/L) along with higher serum prolactin and adrenocorticotropic hormone (ACTH) concentrations. This two-bout exercise protocol may be a useful test for diagnosing OTS, but additional studies are warranted for validation.

## MANAGEMENT

- Because OTS is considered a diagnosis of exclusion and because assessing response to therapy requires time, the initial workup should be divided into two appointments, separated by a 2–3-week interval. The first appointment includes history and physical examination, dietary evaluated, and review of training diaries. At this time, a screening hematologic panel may be obtained to investigate other causes of decreased performance (see Fig. 28.5).
- The mainstay of treatment in OTS is rest. The athlete's response to imposed rest is also critical in achieving an accurate diagnosis. After the initial visit, a period of 2–3 weeks of rest from training is recommended. Athletes may remain active, but not in their chosen sport, and training volume should generally be decreased by at least 50%.
- At the 2- to 3-week follow-up visit, the athlete may resume training if symptoms have resolved and the laboratory workup is within normal limits, but the training regimen should be closely assessed for appropriate periods of recovery. If the athlete's symptoms have not resolved at the follow-up visit and the laboratory workup reveals no explanation, longer periods of imposed rest are necessary while tracking symptoms and mood scores.
- A multidisciplinary treatment approach is often useful and may include sports psychology and nutrition consultations. All four components of recovery must be considered: hydration and nutrition; sleep and rest; relaxation and emotional support; and stretching and active rest.
- **Glutamine:** There may be a role in the future for enteral or parenteral glutamine supplementation. To date, no study has demonstrated improved immune function or recovery with glutamine supplementation in healthy athletes.
- **Branched-chain amino acids (BCAAs):** BCAA supplementation may improve subjective reporting of mood and energy and positively affect POMS scores. However, no definitive improvement in athletic performance has been demonstrated with BCAA supplementation. Supplementation with BCAA is often limited by gastrointestinal side effects, with possible poor tolerance of doses >7–10 g/day.
- **Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs):** Numerous studies have demonstrated that dramatic increases in training volume lead to undesirable mood changes. Increases in fatigue, depression,

anger, and global mood disturbances are common and may lead to clinical depression. Care should be taken before initiating an SSRI or TCA if symptoms of fatigue predominate because increased central serotonin has been hypothesized to cause central fatigue early in OTS. As symptoms progress, particularly if the patient meets or is close to meeting diagnostic criteria for a mood disorder, a TCA or SSRI may be helpful.

## PREVENTION

- Once OTS has developed, there is no treatment other than rest, which indicates prevention as the best strategy. Coaches, athletes, physicians, and trainers should closely monitor athletes for early signs of overtraining. **Mood and sleep disturbances and resting heart rate elevation may be the earliest signs**. Athletes at a high risk of developing OTS may be monitored with daily training logs, frequent screening tests (POMS), and first A.M. basal heart rate monitoring. The normal resting heart rate of an athlete should be determined when he/she is healthy. Consistent elevation of >10 beats per minute above the baseline should raise a concern for OTS. Periodic assessment of athletic performance, such as time trials in a runner or swimmer, may also be performed to screen for decrease in performance associated with OTS.
- Care should be taken to maintain adequate sleep, nutrition, and hydration and to minimize external stressors. Training regimens should be individualized and flexible, with periodization of training and adequate recovery time. A decrease in training volume at the first sign of overtraining may facilitate a quicker recovery. Counsel athletes to avoid the tendency to *panic train* or train harder after a decline in their performance. Care must be taken to not allow development and progression of OTS because the required prolonged recovery time may significantly interfere with an athlete's preparation and participation.
- There has been tremendous recent interest in the role of HIT in the sports-conditioning community. A recent study in swimmers concluded that selective utilization of periodic HIT may, in fact, be a means to improve performance while decreasing training volumes that may lead to OTS. In this study, 41 elite swimmers were randomly assigned to either an HIT or a control group (CON). For 12 weeks, both groups trained for approximately 12 hours/week. The amount of HIT was approximately 5 and 1 hour and the total distance was approximately 17 and 35 km/week in the HIT and CON groups, respectively. After the 12-week intervention, compared with the CON group, general stress levels were 16.6% (2.6%–30.7%; mean and 95% CI) lower and general recovery levels were 6.5% (0.7%–12.4%) higher in the HIT group. These promising results indicate that increasing training intensity and reducing training volume can reduce general stress and increase general recovery levels and may be a strategy to assist in preventing OTS.
- Adolescent athletes may be particularly vulnerable to OTS and burnout with predictable short- and long-term consequences. The American Academy of Pediatrics has published consensus recommendations to assist in preventing OTS in this population. Common sense recommendations include: Encourage athletes to strive to have 1–2 days off per week from competitive athletics, sport-specific training, and competitive practice to allow them to recover both physically and psychologically and encourage athletes to take at least 2–3 months away from a specific sport during the year.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# INFECTIONS IN ATHLETES

# 29

Mark Stovak • Kyle Goerl • Leslie Greenberg

## GENERAL PRINCIPLES

- There is growing interest in the incidence of infectious disease outbreaks among persons engaging in competitive sports.
- Infectious outbreaks can affect sports participation and performance.
- Infections can easily spread among athletes and to surrounding team staff, spectators, relatives, and other contacts.
- Athletes face a variety of psychological, environmental, and physiologic stressors, which, when combined with inadequate diet and sleep deprivation, can cause immunosuppression and increased susceptibility to infections.
- Preventing disruption in sports participation and appropriately timing return to play can be challenging for both the physician and the patient.
- Appropriate diagnosis may help the patient return to health, foster sports success after appropriate timing of return to play, and quell a regional epidemic.
- An athlete's increased risk of contracting infections is attributed to several factors:
  - Contact with surfaces like a mat or artificial turf may cause skin breaks.
  - Athlete-to-athlete contact
  - Athletes may be more apt to be risk-takers, which can increase infections such as sexually transmitted infections.
  - Sharing personal toiletries and congregating in dormitories, locker rooms, or showers
  - Sports equipment, gloves, pads, and protective gear may be contaminated and difficult to sanitize.

### Epidemiology

- First published report of a sports-related infectious disease outbreak was of *Chlamydia trachomatis* in professional wrestlers in 1922.
- Barriers to recovery for athletes include some of the same reasons athletes are more susceptible to illness in the first place:
  - Difficulty taking time off from training to adequately recover
  - Often have inadequate caloric intake
  - Often sleep deprived

### Exercise Immunology

- In 1994, Nieman demonstrated a relationship between exercise and susceptibility to infections in the form of a J-shaped curve.
  - The model suggested that while engaging in moderate activity enhances immune function above sedentary levels, excessive amounts of prolonged high-intensity exercise have detrimental effects on immune functions (for additional details, refer to the J curve diagram in Chapter 18, "Aerobic Training").
- A majority of elite athletes suffer from upper respiratory tract infections (URTIs) at a rate similar to those in the general population, but the incidence of infections in such athletes is higher around competitions, with no seasonal variations.
- Faster marathon runners and those who train for longer distances per week experience more URTI symptoms.
- Tests have been performed on concentration of secretory immunoglobulin A (s-IgA), which is a major effector function of the mucosal immune system, providing a first line of defense against pathogens:
  - s-IgA binds to and opsonizes foreign organisms, including respiratory viruses.
  - Exercise studies have used salivary s-IgA as a marker of mucosal immunity.
  - *Long-duration and high-intensity exercise*, both acutely and chronically, *decreases* salivary s-IgA and is associated with increased incidence of URTIs.
  - *Acute and chronic moderate-intensity exercise increases* s-IgA levels and reportedly lowers the incidence of reported URTIs.
- Overall, the negative effects of intense exercise on the immune system are as follows:
  - Immune cell function: decreased effectiveness and number of white blood cells and increased production of free radicals
  - Stress hormone production: increased adrenaline, cortisol, growth hormone, and prolactin; in addition, such increases in these hormones depress immune cell function
  - Glutamine function: Glutamine assists in control of the rates of T- and B-lymphocyte proliferation and antibody synthesis. Intense exercise causes glutamine levels to decrease by about 20%. Castell found that oral glutamine supplementation after exercise improves immune function by increasing plasma glutamine levels.
- Gleeson found that postexercise immune function depression was most pronounced when exercise is moderate-to-high intensity, continuous, and prolonged.
- Leukocyte function may continue to remain depressed at 24 hours after the last exercise bout.
- If the recovery time between consecutive bouts of intense exercise is insufficient, chronic immunosuppression can occur, which is often observed in overtrained athletes.

## COMMON RESPIRATORY INFECTIONS

### Febrile Illness

- Fever is a normal physiologic response typically to infection with the intent to increase host survival and decrease the length of illness.
- The Centers for Disease Control and Prevention (CDC) defines fever as an internal temperature of >38°C.
- Fever can cause undesirable effects in the body; these may include increased insensible fluid losses, dehydration, increased metabolic demands, and dysregulation of body temperature.
- The combination of hyperthermia and dehydration leads to reduction in both cardiac output and blood pressure, which can be particularly concerning in athletes.
- A combination of these factors leads to reduced exercise tolerance, endurance, and muscle strength and an increase in perceived fatigue through cytokine-induced catabolism of muscles, which has been shown to potentially last for months after an illness.
- Continuing to exercise during a febrile illness can further potentiate negative effects from the disease process and can evolve into myocarditis, which could be potentially lethal.
- Athletes should be withheld from physical activity at least until their fever has resolved, and return to participation should be gradual.

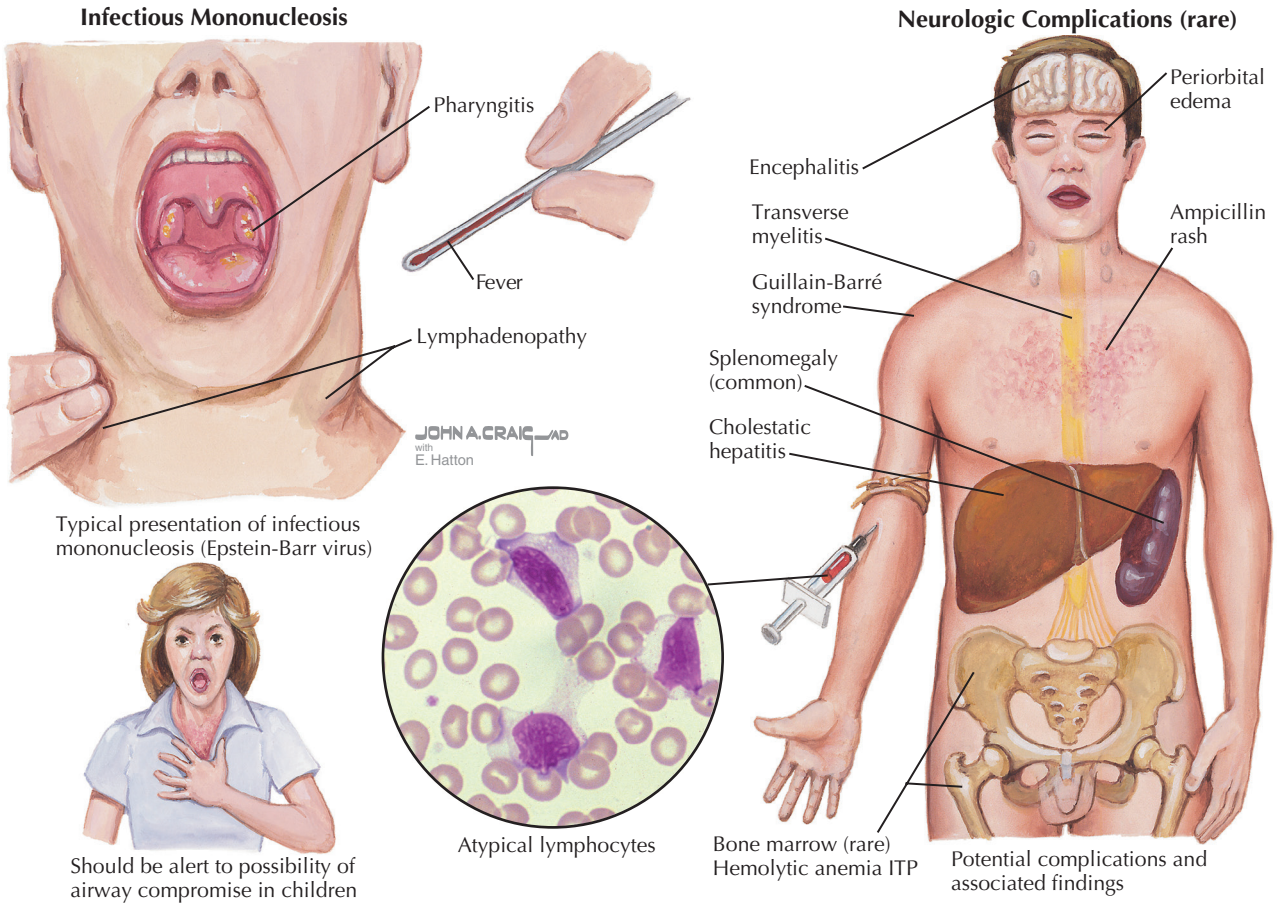


**Figure 29.1.** Infectious mononucleosis.

- To prevent the spread of febrile illnesses, the most common and effective tool is hand washing. It may be necessary to separate an athlete for a certain period of time from their teammates to prevent spread of infection to other members of the team.

## Upper Respiratory Tract Infections (URTIs)

**Etiology**: In general, URTI and common cold are used interchangeably. They are caused by respiratory viruses, typically **rhinoviruses**. Transmission is through nasal secretions by sneezing, coughing, and nose blowing.

**Epidemiology**: Leading reason for physician visits; cough is the 3rd whereas nasal congestion is the 15th most common presenting symptom in all office visits, and URTIs are the third most common primary care diagnosis

**Symptoms**: Common symptoms include fever, cough, rhinorrhea, nasal congestion, sore throat, headache, and myalgias. Most are self-limited, typically lasting up to 10 days.

**Examination and Diagnosis:** Fever, rhinorrhea, erythema, cobble stoning or swelling of the posterior oropharynx, fluid level behind the tympanic membrane, and cervical lymphadenopathy; diagnostic testing is not typically necessary unless there is suspicion for Group A streptococcal or mononucleosis infections

**Treatment**: Treatment is symptomatic and supportive. Effective modalities include antipyretic and anti-inflammatory medications, decongestants, nasal saline irrigation, intranasal ipratropium, and zinc. Antibiotics are not effective.

**Return to Play**: The **"neck check"** can be a helpful tool when considering whether physical activity is appropriate. Basically, an athlete with URTI symptoms "above the neck," with no systemic signs and symptoms, including no fever, should undergo 10–15 minutes of mild to moderate exercise and, if tolerated, may continue participation.

**Prevention**: Hand washing should be encouraged, and teammates should not share water bottles. Prophylactic vitamin C has been shown to modestly reduce the duration of cold symptoms but not the incidence.

## Infectious Mononucleosis (IM) (Fig. 29.1)

**Etiology**: Infectious mononucleosis (IM) or "mono" is the result of an infection with the **Epstein–Barr virus** (EBV). EBV is the primary cause in 90% of cases, but cytomegalovirus and toxoplasmosis can cause a similar syndrome as well. Transmission of the virus is through oral secretions; thus, the disease has been nicknamed as the "kissing disease." EBV has an incubation period of 30–50 days, and it causes a cell-mediated immune response, which leads to T lymphocyte proliferation. This results in lymphoid hyperplasia, lymphocytosis, and **atypical lymphocytes** on a peripheral blood smear.

**Epidemiology**: EBV is very common; 90%–95% of adults display immunity to EBV, indicating prior infection. There are approximately 500 cases per 100,000 persons per year in the United States, with the highest incidence in adults aged 15–24 years. Athletes are at no higher risk to contract the virus than nonathletes.

**Symptoms**: An infection with EBV can be clinically insignificant in children but results in a symptomatic illness in adolescent and adult patients. Typically, there is a 3–5-day prodrome

consisting of malaise, fatigue, and anorexia, which then progresses to the classic "triad" of IM with **pharyngitis, fever, and lymphadenopathy**. Rarely, laryngeal edema can occur, potentially leading to airway obstruction.

**Examination and Diagnosis**: The reactive lymphadenopathy classically leads to enlargement of **posterior cervical lymph nodes**, but axillary and inguinal nodes may also be enlarged. Posterior palatine petechiae, jaundice, exudative pharyngitis, rash (commonly seen in a patient who started penicillin for a suspected group A streptococcal infection), and splenomegaly are also possible. Jaundice is uncommon, but 90% of patients have mildly elevated AST and ALT. Exudative pharyngitis can be mistaken for a streptococcal pharyngitis, but concomitant infections do occur in up to 30% of patients. Splenomegaly and increased risk of rupture is concerning for athletes, but routine imaging is not generally regarded as useful in return-to-play decisions. Splenomegaly alone does not predict the risk of rupture. A **Monospot** test, which detects the presence of heterophile antibodies, is clinically utilized but has a false negative rate of 25% in the first week of illness. Testing for EBV early antigen (EA), IgM, and IgG (to EBV viral capsid antigens) may be useful if Monospot is negative. Atypical lymphocytes have a sensitivity of 75% and a specificity of 92%, and other laboratory abnormalities may include transient neutropenia, thrombocytopenia, and anemia.

**Treatment**: Treatment is supportive, including rest, hydration, and NSAIDs. In addition, given the negative effects IM can have on the liver, acetaminophen should be used with caution. Moreover, aspirin is best avoided for risk of bleeding and an association of IM with Reye syndrome in pediatric patients. In general, corticosteroids are not needed but may be considered if there is significant laryngeal edema or painful swallowing that interferes with drinking and eating.

**Return to Play**: Splenic rupture is the most concerning issue while making return-to-play decisions. All athletes should be withheld from competition until their symptoms have resolved. Occasionally, light aerobic activity can be initiated after 14 days if the athlete's symptoms have resolved, but published guidelines (expert opinion) generally recommend to withhold an athlete **from participation for 3 weeks**. Some experts advocate holding out contact athletes and anyone with splenomegaly for a total of 4 weeks. Follow-up laboratory results demonstrating normalization are not necessary. Athletes, parents, coaches, and trainers should be educated that in severe cases, it may take up to 3 months to make a full recovery.

**Prevention**: Hand washing should be encouraged, and teammates should not share water bottles.

## Influenza (Fig. 29.2)

**Etiology**: Influenza is an airborne viral illness that infects the body through the respiratory tract. It is a single-stranded RNA virus in the Orthomyxovirus family. **Influenza A** is the cause of most influenza illnesses. The virus has an incubation period of 18–72 hours, and viral shedding can occur anywhere from 24 hours before initiation of symptoms to up to 5–10 days thereafter.

**Epidemiology**: The World Health Organization (WHO) estimates, 3–5 million severe influenza cases are reported each year with 250,000–500,000 deaths worldwide. Older adults, young

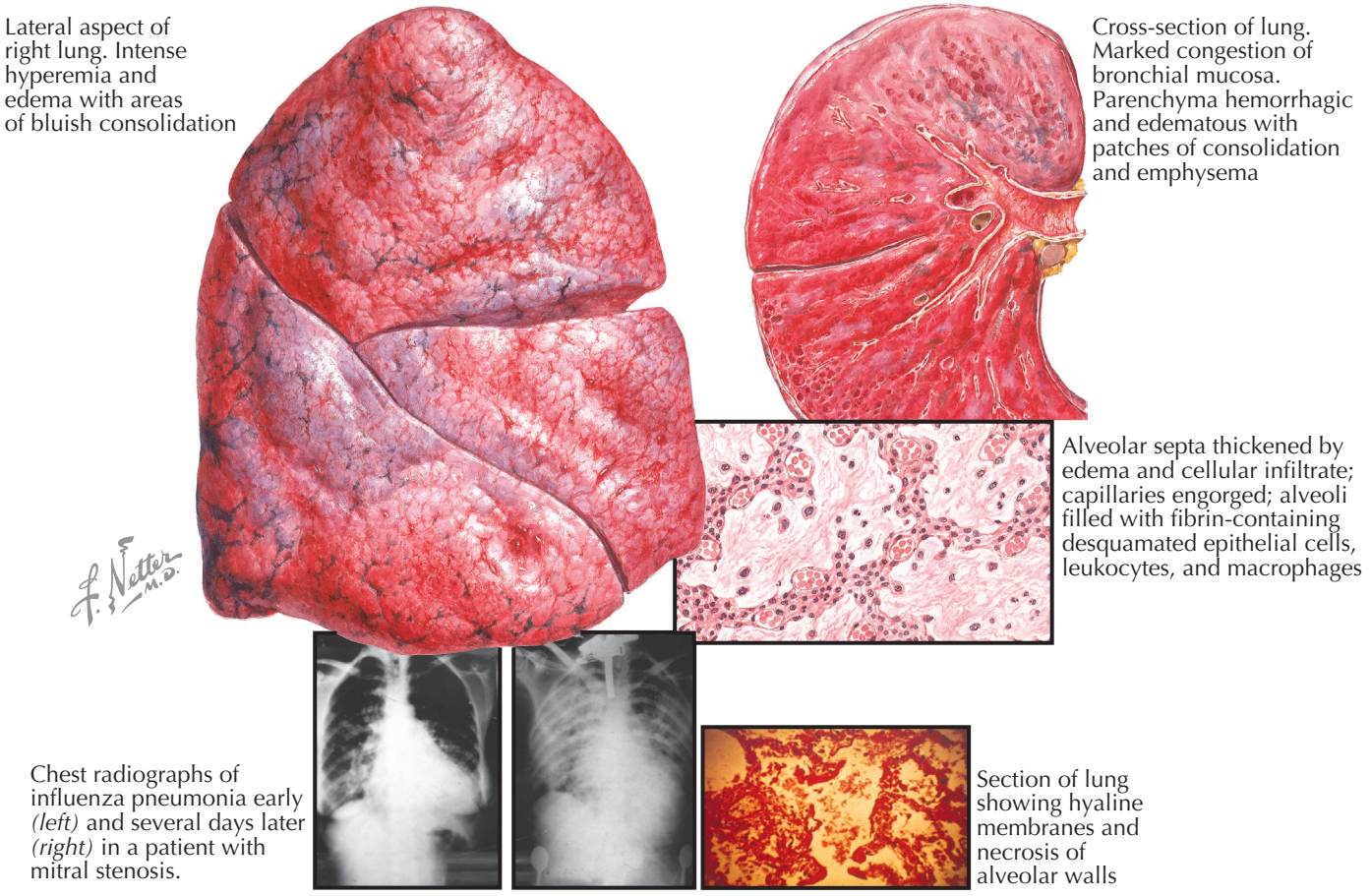


**Figure 29.2.** Influenza pneumonia.

children (<5 years), persons with chronic medical conditions, and pregnant women are at the highest risk of complications.

**Symptoms**: Classic symptoms include sudden onset of **fever, myalgias, headache**, malaise, rhinitis, sore throat, and cough. Severe complications are rare in healthy children and adults but can include pneumonia, encephalitis, respiratory failure, multiorgan failure, and death.

**Examination and Diagnosis**: Influenza is typically **clinically diagnosed based on the sudden onset of aforementioned symptoms**. Patients may have fever, rhinorrhea, erythema, cobblestoning or swelling of the posterior oropharynx, a fluid level behind the tympanic membrane, and cervical lymphadenopathy. Cardiac and pulmonary auscultation is typically normal unless there is associated pneumonia or cardiac complications such as pericardial effusion. Rapid tests have variable sensitivities (10%–70%); hence, a negative test does not rule out an infection but the tests are highly specific (95%).

**Treatment**: Treatment is typically **symptomatic** and includes anti-inflammatory and antipyretic medications, decongestants, and cough suppressants. Hydration should be encouraged. **Antiviral medications such as oseltamivir (Tamiflu) and zanamivir (Relenza) are not typically recommended in healthy children and adults without complications**. However, antivirals can be considered if symptoms have been present for <48 hours in order to shorten the duration of illness and potentially decrease the risk of complications, although this is questionable. This may apply to patients who are a part of team sports where transmission to other teammates may occur quickly. In this case, exposed teammates may also benefit from prophylactic antiviral treatment.

**Return to Play**: Athletes can return to play when their symptoms are well controlled, and they are no longer febrile off all medications.

**Prevention**: The CDC recommends **all people over 6 months of age to receive influenza vaccination**. Hand washing should be encouraged. Affected athletes should likely be isolated from teammates until their symptoms, particularly fever, have subsided to avoid transmission.

## COMMON SKIN AND SOFT TISSUE INFECTIONS

### Herpes Simplex Virus (HSV) Infection

**Etiology**: HSV-1. HSV on the skin, is often called "herpes gladiatorum" in wrestlers and "herpes rubeiorum" or "scrumpox" in rugby players. HSV-1 on the lips or "herpes labialis" is usually referred to as *cold sores*. Time lapse from virus exposure to vesicle appearance is 4–11 days.

**Epidemiology**: Most prevalent in sports with frequent, direct contact between players, such as rugby and wrestling; most common infection sites are head, face, and neck, followed by extremities and, last, the trunk. The most common mode of transmission is via direct contact with open wounds and skin tears. HSV is uncommon in lower extremities.

**Symptoms**: Mild flu-like symptoms or a prodrome of burning, tingling, or stinging may occur, followed by development of small clusters of painful papules or vesicles with possible fatigue, weight loss, pharyngitis, and lymphadenopathy in severe cases

**Examination and Diagnosis**: Small clusters of painful papules or vesicles (that may follow a dermotomal pattern, in case of shingles). Testing is not usually necessary, but Tzanck preparation can be confirmatory, revealing large, multinucleated giant cells. Culture and polymerase chain reaction testing have a high specificity as well.

**Treatment**: Antiviral medications (acyclovir, valacyclovir, or famciclovir) and covering lesions to decrease contagious spread

**Return to play**: **The National Federation of High School (NFHS) guidelines for Herpetic Lesions (Simplex, fever blisters/cold sores, Zoster, or Gladiatorum):** To be considered "noncontagious," all lesions must be scabbed over with no oozing or discharge, and no new lesions should have occurred in the preceding 48 hours. For primary (first episode of Herpes Gladiatorum), wrestlers should be treated and not allowed to compete for a minimum of 10 days. If general physical signs and symptoms such as fever and swollen lymph nodes are observed, the minimum period of treatment should be extended to 14 days. Recurrent outbreaks require a minimum of 120 hours or 5 full days of oral antiviral treatment as long as no new lesions have developed and all lesions are scabbed over. **The National Collegiate Athletic Association (NCAA) guidelines for Herpetic Lesions (Simplex, fever blisters/cold sores, Zoster, Gladiatorum**) require skin lesions to be surmounted by a FIRM ADHERENT CRUST at the time of competition and have no evidence of secondary bacterial infection. For primary (first episode of Herpes Gladiatorum) infection, a wrestler must have developed no new blisters for 72 hours before the examination; be free of signs and symptoms such as fever, malaise, and swollen lymph nodes; and have been on appropriate dosage of systemic antiviral therapy for at least 120 hours before and at the time of the competition. Recurrent outbreaks require a minimum of 120 hours of oral antiviral treatment as long as no new lesions have developed and all lesions are scabbed over. Active herpetic infections should not be covered to allow participation.

**Prevention**: Daily suppressive therapy is an option for recurrent infections. Valacyclovir (Valtrex) provides the easiest dosing of 500 mg BID during the sports "season." This easy dosing regimen may increase compliance and help prevent the spread of infection. Consider annual HSV serology testing to identify individuals who may need suppression-dosed antiviral medication before the start of the season. Because HSV can survive for several hours on inanimate objects or shared equipment, thorough cleaning of water bottles, fomites, and clothing is highly recommended. Nonabrasive uniforms may reduce skin abrasions/tears and may reduce transmission of infection. Although wrestling mats have not been shown to be a mode of transmission, regular cleaning of mats is still recommended. Prepractice and match screening of athletes for active lesions helps in preventing opponents and other teammates from contact with known vectors.

### Verrucae (Warts)

**Etiology**: Papillomaviruses

**Epidemiology**: Transmission is via direct contact, shared showers, or locker room floors

**Symptoms**: Plantar warts are sharp, well-defined hyperkeratotic lesions, either single or in clusters, with a smooth collar of thickened keratin. Plantar warts are painful as they grow inward because they exist on the plantar weight-bearing surface of the feet. Verrucae vulgaris are firm, rough papules or nodules that are 2–10 mm in diameter with punctate black dots within the lesion and appear on the rest of the body.

**Treatment**: Respond to numerous modalities; plantar warts may be treated with 30%–70% trichloroacetic acid, 40% salicylic acid, or taping with salicylic tape or duct tape for several days. Verrucae vulgaris may be treated with repeated cryotherapy with liquid nitrogen, 1% salicylic acid or lactic acid, or curettage with electrodessication.

**Return to Play**: **No NFHS limitations; NCAA guidelines for wrestlers with multiple digitate verrucae of their face will be disqualified if infected areas cannot be covered with a mask**

**Solitary or scattered lesions can be curetted away before a meet or tournament. Wrestlers with multiple verruca plana or verrucae vulgaris lesions must have the lesions adequately covered.**

**Tinea corporis ("ringworm")** - causes a ring-shaped rash, usually on the legs or trunk of the body.

**Tinea cruris ("jock itch")** - affects the inner thighs and groin. It causes pain and severe itching and usually a rash of red, ring-like patches that grow outward in the crease of the thighs. The patches usually have bumps and a different color than nearby skin.

**Tinea pedis ("athlete's foot")** - causes severe itching and a rash on bottoms of the feet and between the toes. Itching is often worst between the toes.

**Figure 29.3.** Tinea infections.

**Prevention**: Routine usage of protective rubber slides/flip-flops in public showers or locker rooms

## Tinea Infections (Fig. 29.3)

**Etiology**: Most commonly *Trichophyton rubrum* and *T. tonsurans*

Specific fungal infections are *tinea pedis* (athlete's foot), *tinea cruris* (jock itch), *tinea corporis* (ringworm), *tinea capitis* (cradle cap), and *tinea versicolor* caused by *Malassezia furfur*.

**Epidemiology**: Sweat-soaked clothing, suntan beds, communal showers, infrequently washed/sanitized clothing, occlusive clothing, and local skin trauma associated with form-fitting equipment can create common conditions in sports that put athletes at a risk of developing tinea infections.

**Symptoms**: Local pruritus, erythema, and burning

**Examination and Diagnosis**: Potassium hydroxide (KOH) preparation and microscopic examination of skin scrapings can be confirmatory very quickly, whereas culture takes longer.

- **Tinea pedis**: malodorous maceration of the skin of interdigital spaces with associated peeling, cracking, scaling, and pruritis
- **Tinea cruris**: itchy, red, often ring-shaped rashes on the skin of buttocks, genitals, and inner thighs
- **Tinea corporis**: circular or ring-shaped, scaly, raised plaque with irregular erythamatous borders and often with central clearing
- **Tinea capitis**: patchy scale with erythema leading to hair breaking off flush with the scalp, leaving patches of alopecia with follicular plugs that create a characteristic "black dot" appearance
- **Tinea versicolor:** hypopigmented, scaly patches in a diffuse pattern

**Treatment**: Antifungal medications may be topical or oral, and continuation of treatment is recommended for 2 weeks after clinical resolution for tinea pedis, tinea cruris, and tinea corporus. Tinea capitis requires only oral medication. Tinea versicolor can be treated with topical or oral antifungals.

Suppression and prophylactic doses have been successful and include fluconazole (100–200 mg once/week) or itraconazole (400 mg every other week). Appropriate sterilization of the sports environment—including the locker room, equipment, and clothing—is recommended daily.

**Return to play**: **NFHS guidelines for tinea lesions (ringworm on scalp or skin):** Oral or topical treatment for 72 hours on the skin but only oral treatment for 14 days on the scalp.

NCAA guidelines for tinea lesions (ringworm): Oral or topical treatment for 72 hours on the skin but only oral treatment for 14 days on the scalp. Wrestlers with solitary, or closely clustered, localized lesions will be disqualified if lesions are in a body location that cannot be adequately covered. **There are no NFHS or NCAA restrictions on return to sport for tinea versicolor because it has a very low transmission rate.**

**Prevention**: Appropriate clothing barriers, laundering of towels/uniforms/padding/equipment, use of shower footwear, and routine showering as well as prompt identification and treatment of all cases

## Community-Acquired Methicillin-Resistant *Staphylococcus aureus* (CA-MRSA)

**Etiology**: Colonization of the skin or exposure to MRSA through an open skin lesion

**Epidemiology**: Originally a hospital-acquired infection, MRSA has caused substantial morbidity and mortality in athletes since it emerged in the 1960s. Athletes often mistakenly believe the lesions to be spider or insect bites, which delays appropriate identification and treatment. High-risk sports for MRSA infection are those with frequent, repetitive, and direct skin contact. Transmission can also occur through environmental sources such as shared equipment, towels, and whirlpools.

**Symptoms**: Tender lesion on the skin usually rapidly progressing from a cellulitis to an abscess

**Testing**: Consensus guidelines recommend cultures in the presence of a purulent lesion and at least one of the following features: treatment with antibiotics, signs of systemic toxicity, multiple or large lesions, bite as the cause, association with water exposure, lack of response to initial treatment, or occurrence during a suspected outbreak. Culturing when antibiotics are used allows determination of sensitivities, which can guide antibiotic choices in the case of a poor response to the initial antibiotic.

**Treatment**: Lesion incision and drainage; culture samples from the wound

Current Infectious Disease Society of America guidelines recommend antibiotic treatment after incision and drainage

of suspected MRSA lesions for patients with severe or extensive disease, rapid progression of associated cellulitis, signs of systemic toxicity, lesions that are difficult to drain (e.g., in the groin or axilla), and a lack of response to incision and drainage alone. Oral antibiotics effective against CA-MRSA include trimethoprim-sulfamethoxazole, tetracycline, clindamycin (perform D-test if erythromycin-resistant strain is suspected), and linezolid. Decolonization of MRSA carriers has a limited role. Despite several whole-body antimicrobial decolonization protocols, which typically involve topical antimicrobial scrubs and nasal mupirocin, there is limited evidence available for the overall utility and accurate setting for these protocols. Decolonization can be considered for patients with recurrent MRSA infection, for close contacts who are culture-confirmed carriers, and in a MRSA outbreak in the training room setting. Decolonization can be achieved through a daily body wash with 4% chlorhexidine solution and 2% mupiricin ointment applied to each nostril 2 times per day for 5 days.

**Return to Play**: **NFHS guidelines for bacterial diseases (impetigo, boils):** To be considered "noncontagious," all lesions must be scabbed over with no oozing or discharge, and no new lesions should have occurred in the preceding 48 hours. Oral antibiotics for a minimum of 3 days is considered to achieve that status. If new lesions continue to develop or drain after 72 hours, a diagnosis of MRSA should be considered.

**NCAA guidelines for bacterial infections (furuncles, carbuncles, folliculitis, impetigo, cellulitis or erysipelas, staphylococcal infection, and CA-MRSA):** Wrestlers must not have any new skin lesion development for 48 hours before the meet or tournament; completed 72 hours of antibiotic therapy and have no moist, exudative, or draining lesions at the time of the meet or tournament. Gram stain of exudate from questionable lesions (if available). Active bacterial infections should not be covered to allow participation.

**Prevention**: Screen athletes regularly; teach them to report all skin injuries.

Keep skin injuries clean and covered until healed. Hand washing should be emphasized by all athletes and staff, as the CDC has reported contaminated hands as the primary mode of MRSA transmission. In addition, routine showering immediately after activity, cleaning of facilities and showers, and using liquid antibacterial soap and clean towels should be encouraged. Sharing of fomites, equipment, and razors should be discouraged.

## ATHLETIC PARTICIPATION AND OTHER INFECTIONS

**Human Immunodeficiency Virus (HIV) infection:** No available evidence to support that exercise and training of moderate intensity is harmful to an HIV-infected athlete. Exercise may be beneficial to HIV-infected athletes by reducing anxiety and depression. HIV-infected athletes should be allowed to participate in sports if they are asymptomatic and show no evidence of deficiencies in immunologic function.

**Hepatitis B Virus (HBV) Infection:** Participation should be determined based on signs or symptoms such as fever, fatigue, or hepatomegaly. There is no evidence that intense, competitive training is harmful to an asymptomatic HBV carrier (acute or chronic) without evidence of organ impairment.

**Hepatitis C Virus Infection**: There are no current CDC recommendations regarding athletic participation for athletes who have HCV infection, but participation based on signs and symptoms, similar to HBV, is probably safe (not are).

**Pericarditis (2015 AHA/ACC Scientific Statement):** No exercise or sports during acute phase; return to full activity with resolution of active disease (no effusion on echocardiogram and serum markers of inflammation normalized)

Chronic constrictive pericarditis is a contraindication to participation in sports.

**Myocarditis (2015 AHA/ACC Scientific Statement): Athletes with probable or definite myocarditis should not participate in competitive sports while active inflammation is present**. Recovery can take several months. There is a risk of lethal arrhythmias until recovery is complete. Current recommendations include evaluation using resting echocardiogram, 24-hour Holter monitor, and exercise stress test 3–6 months after initial illness before returning to competitive sport. May return to sport if:

- Ventricular systolic function has returned to normal
- Serum markers of myocardial injury, inflammation, and heart failure have normalized
- Arrhythmias such as frequent or complex repetitive forms of ventricular or supraventricular ectopic activity are absent on Holter monitor and graded exercise EKGs

## REPORTABLE DISEASES

- Reportable diseases are infections considered to be of great public importance that must be reported when they are diagnosed by doctors or laboratories.
- Reporting allows researchers to identify disease trends and track disease outbreaks. Every state has a reportable disease list.
- Is the responsibility of the healthcare provider, not the patient, to report disease cases; it is very important to be aware of how and when to report reportable diseases.
- Common reportable diseases are chicken pox, tuberculosis, HIV, Hepatitis A, Hepatitis B, Hepatitis C, meningitis, gonorrhea, and chlamydia.
- County or state health department will try to identify the source of several of these illnesses, and for sexually-transmitted diseases, the county or state will try to locate sexual contacts of infected people.
- Cooperation with state health workers can facilitate locating the source of infection and help quell the spread of an epidemic.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# GASTROINTESTINAL PROBLEMS

# 30

Joshua Baker • Jesse DeLuca • Anthony Beutler

## GENERAL PRINCIPLES

Exercise does not favor the gastrointestinal (GI) system. During exercise, blood flow is diverted from the splanchnic circulation and preferentially distributed to demanding peripheral muscles. An additional antagonist to normal GI functioning is the persistent mechanical movement of internal organs during exercise. These two factors are thought to be major underpinnings to various GI problems. Fortunately, despite almost 90% of some populations reporting exercise-associated GI complaints, the severity of illness is usually mild.

### Approach to GI Problems in Athletes

#### History

- Differentiate upper GI from lower GI symptoms: nausea, cramping, bloating, and diarrhea
- Determine severity of disease: hematochezia and melena
- Consider effects of foods and beverages, anxiety/stress, caffeine, tobacco, alcohol, nonsteroidal anti-inflammatory drugs (NSAIDs) or other medications, drugs of abuse or other supplements
- Assess possibility of undiagnosed systemic disease (e.g., celiac or inflammatory bowel disease)

### Physical Exam

- Signs of abnormal palpatory exam: localized or generalized abdominal tenderness, organomegaly, mass, hernia, and peritoneal signs
- Signs of volume depletion: orthostatic hypotension and tachycardia
- Signs of inflammatory bowel disease: oral ulcers, dermatologic and ocular signs, and joint manifestations
- Signs of thyroid disease: thyromegaly, altered reflexes, and dermatologic and ocular manifestations
- Signs of systemic wasting: temporal wasting, lymphadenopathy, hepatomegaly, and splenomegaly

### Differential Diagnosis (Table 30.1)

- Infectious: gastroenteritis and hepatitis
- Neoplastic: GI tract cancer and lymphoma
- Endocrine: hyperthyroidism, hypothyroidism, and pancreatic disease
- Autoimmune: Crohn's disease, ulcerative colitis, and celiac disease
- Trauma: GI and genitourinary organs
- Vascular: cardiac and mesenteric ischemia
- Other: peptic ulcer disease, irritable bowel syndrome, constipation, medication or supplement-induced disorder, and problems related to food or beverage intake

### Laboratory Data

- Determine the necessity of tests based on the severity of symptoms. Tests may include cell blood count, iron studies, hepatic function panel, *Helicobacter pylori* testing, electrolytes, thyroid studies, and **occult blood in stool** and other stool studies

## UPPER GI PROBLEMS

- Upper GI pain related to training and competition often presents a diagnostic dilemma as both serious and benign etiologies often have similar presentations. Common symptoms include epigastric pain, dysphagia, dyspepsia, nausea, vomiting, and heartburn.
- During exercise, central blood volume is maintained by redirecting blood away from internal organs, particularly the splanchnic bed. Studies have revealed that splanchnic blood flow declines from 1.56 L/minute at rest to 0.3 L/minute at maximal exercise.
- **Possible negative effects of exercise-induced shunting:** Decreased esophageal motility, erosive hemorrhagic gastritis, delayed gastric emptying, diarrhea, and intestinal bleeding
- **Angina or cardiac ischemia:** Should always be entertained as a possible diagnosis in epigastric pain, particularly in older athletes
- **Gastroesophageal reflux (GER)**
  - Vigorous exercise causes GER in healthy subjects, notably in runners, bicyclists, and weightlifters.
  - Frequency, amplitude, and duration of esophageal contractions decline with increasing exercise intensity.
  - Hypoperfusion resulting from physiologic arterial shunting to muscles and skin may cause reduced esophageal motility.
  - Treatment: $H_2$-blockers or proton pump inhibitors 4 hours before exercise, standard medical management for GER, alteration of oral intake (avoid symptom-triggering foods and beverages, no food for 3 hours before exercise)
- **Gastritis:** Erosive gastritis may be induced by exercise-related hypoperfusion, mechanical forces, or NSAIDs: often hemorrhagic. Treat with proton pump inhibitors, $H_2$-blockers, and antacids (Fig. 30.1).
- **Peptic ulcer disease** is as common in runners as in the general population; use standard medical management (Fig. 30.2).
- **Delayed gastric emptying** may be related to bloating, reflux, or both; likely only at severe exercise; may be caused by hypoperfusion, resulting from arterial shunting away from the splanchnic bed
- **Upper GI bleeding:** May be related to hemorrhagic gastritis or peptic ulcer disease (see the Gastritis section); mechanical cause is proposed in some cases. Gastric fundus is the most common site for gastric bleeding because the shearing force from the adjacent diaphragm may be the source.
  - Evaluate and treat using standard upper GI methodologies.
  - Improve hydration before and during performance. Increased plasma volume may reduce ischemia.
- **Exercise-associated transient abdominal pain (ETAP):** Often referred to as a "side-stitch"; presents as pain, most often in the lateral aspect of the midabdomen. Incidences decline with age. Proposed mechanisms include diaphragm spasm, exertional irritation of parietal peritoneum, compression of

thoracic intercostal nerves, and trapped gas in hepatic or splenic flexure. Avoid solid meals before exercise.

## LOWER GI PROBLEMS

### Runner's Diarrhea ("Runner's Trots")

- Stimulated by intense endurance running, with or without accompanying GI bleeding
- Descriptive data from a study by Priebe of 425 runners in a 10K race showed the following:
  - Incidence: 30% of runners in the race
  - Characteristics of syndrome: 85% passed semiformed or watery stools; 60% had low abdominal pain or rectal urgency, generally relieved by defecation; 15% had multiple stools; 13% had large-volume stools; and **12% had frank blood in stool**.
  - Other data: 30%–42% of serious runners have an urge to defecate; 14%–30% report running-induced diarrhea; direct relationship between the severity of symptoms and level of physical exertion; diarrhea more common in running than in other sports
- See Box 30.1 for general recommendations for prevention/treatment.

#### Proposed Etiologies

- Anxiety-induced diarrhea (see Anxiety and Stress Reaction section)
- Increased GI motility: exercise increases the secretion of gastrin, motilin, other hormones that affect motility; irritable bowel syndrome
- Dietary factors: high-fiber diet may cause exercise-induced diarrhea in a small subset of runners; a higher incidence of

**TABLE 30.1 GASTROINTESTINAL PROBLEMS**

| Upper GI Problems | Lower GI Problems | General GI Problems |
|---|---|---|
| Angina/Cardiac Ischemia | Runner's Diarrhea | Anxiety/Stress |
| Gastroesophageal Reflux | Lower GI Bleed | Acute Gastroenteritis |
| Gastritis | Ischemic Colitis | Traveler's Diarrhea |
| Peptic Ulcer Disease | Cecal Slap Syndrome | Celiac Disease |
| Delayed Gastric Emptying | Celiac Artery Compression Syndrome | Irritable Bowel Syndrome |
| Upper GI Bleed | | Inflammatory Bowel Disease |
| Exercise-Associated Transient Abdominal Pain | | |

**BOX 30.1 GENERAL RECOMMENDATIONS FOR PREVENTION/TREATMENT OF DIARRHEA**

- Encourage bowel movement. Have a light meal a few hours before competition and then jog to stimulate gastrocolic reflex.
- Improve hydration before and during performance: increased plasma volume may decrease ischemia (with or without diarrhea).
- Dietary manipulations:
  - Athletes on low-fiber diet may improve by adding fiber to absorb intraluminal fluid.
  - Athletes on high-fiber diet may improve by reducing fiber to decrease stimulation of intestinal motility. Several athletes prefer a high-fiber diet because it stimulates intestinal motility, thus reducing intraluminal contents.
  - Eliminate foods that trigger bowel symptoms in individual athletes—lactose, fructose, sorbitol, and caffeine.
- Antimotility medications may be helpful in cases of diarrhea that appear to be a form of functional bowel syndrome and in those that remain undiagnosed: Imodium (risk of hyperthermia) and Lomotil.
- Decrease training and competition level by 20%–40% in both mileage and intensity, then build back up slowly. May be able to cross-train.
- Surgery involving division of obstructing diaphragmatic fibers and denervation of celiac ganglion may benefit patients with celiac compression syndrome but remains controversial.

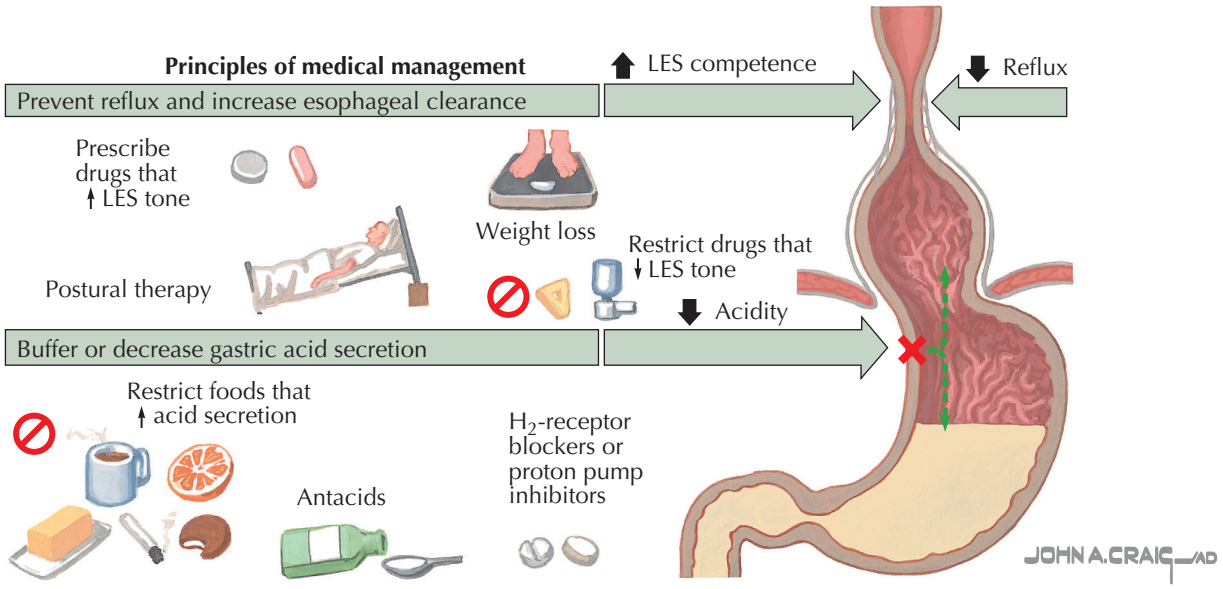


Figure 30.1. Gastroesophageal reflux.

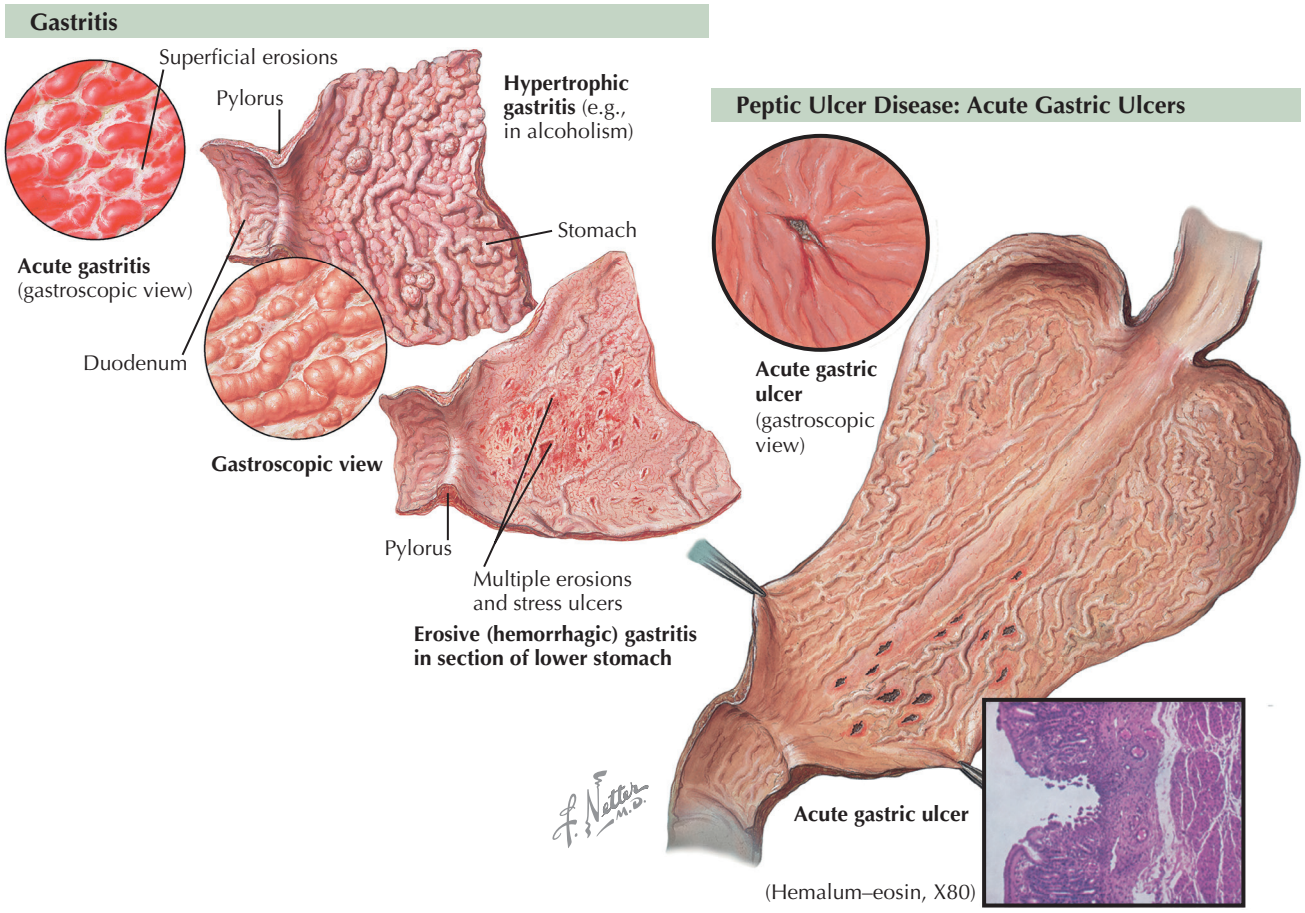


**Figure 30.2.** Upper GI problems.

lactose intolerance in patients with exercise-induced diarrhea than in general population; symptoms may occur only during exercise; sorbitol or fructose intolerance with fruit-intensive diets; large doses of caffeine or vitamin C
- Possible immune system etiology: Variant of exercise-induced anaphylaxis; generalized urticaria including urticarial lesions in intestines
- Endotoxins have been proposed as a cause of GI symptoms.

## Lower GI Bleeding

- Grossly bloody diarrhea stimulated by intense performance: large amounts of red, maroon, or clotted blood may be seen; severe abdominal pain
- Microscopic increases in fecal hemoglobin after intense running are more common than gross blood and have been noted in endurance bicyclists; may be caused by upper or lower GI bleeding; transient in endoscopic studies

### Proposed Etiologies

- Crampy abdominal pain combined with frank blood in stool should be an indication to stop exercise and rest, and likely seek further medical attention.
- **Ischemic colitis:** hypoperfusion caused by shunting blood flow from mesentery to muscles and skin; strenuous exercise may decrease flow by up to 80% (may be magnified by dehydration). Relative gut ischemia, particularly in watershed areas of colon, causes focal areas of necrosis and ulceration. In addition, ischemia may cause intestinal malabsorption, thereby leading to diarrhea; most often seen in endurance athletes, and prolonged ischemia can lead to colectomy
- **Cecal slap syndrome:** mechanical trauma from running reported to cause hemorrhagic cecal lesions and diarrhea
- **Celiac artery compression syndrome** (median arcuate ligament syndrome): rare and controversial cause of chronic recurrent abdominal pain reported in nonathletes.
  - External compression of celiac and occasionally superior mesenteric artery (SMA) by the median arcuate ligament of diaphragm (left crus or fibrous band connecting left and right crus), particularly during exhalation (Fig. 30.3)
  - Diagnosis of exclusion: all other GI pathology must be ruled out, with a high index of clinical suspicion
  - No clear diagnostic criteria and no imaging modality predicts response to surgery
  - Most common in young individuals, those with recent weight loss, and women
  - Pain may be sharp, dull, steady, or crampy and may worsen after meals, weight loss, or position changes.
  - **Loud systolic bruit** may be appreciated in the epigastrium.
  - **Four proposed mechanisms for pain:** compression of celiac and SMA, resulting in mesenteric ischemia; extensive collateral circulation "steals" from primary distribution of SMA in the fed state; inadequate collateral circulation, resulting in nonocclusive ischemia during hypotension or decreased perfusion; irritation of celiac plexus or overstimulation of sympathetic nerve fibers
  - **Arteriography** confirms clinical diagnosis of celiac artery compression syndrome; duplex ultrasonography recently

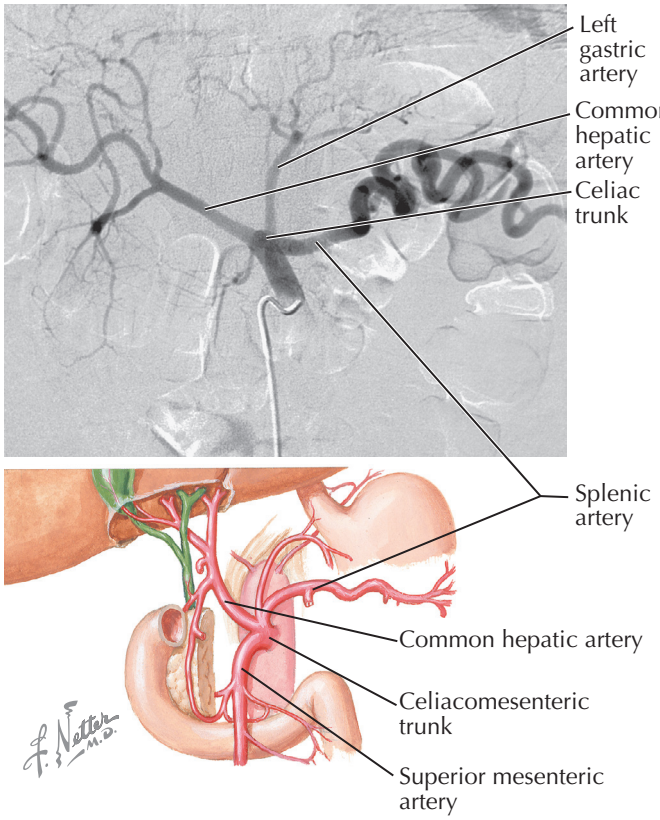


**Figure 30.3.** Celiac trunk: normal and variant.

used to measure flow velocities in SMA and celiac arteries

## GENERAL GI PROBLEMS IN ATHLETES

### Anxiety and Stress Reaction

- Performance anxiety
- Inhibitory effects on upper GI function: decreased acid secretion in the stomach, diminished motor activity, and reduced blood flow
- Continued anxiety may result in acid hypersecretion
- Stimulant effect on lower GI activity: increased motility and decreased transit time
- **Symptoms:** dry mouth, dyspepsia ("knot in stomach"), heartburn, reflux, abdominal cramping, and diarrhea
- **Treatment:** reassurance and education, behavior modification, and relaxation exercises

### Acute Gastroenteritis

- Incidence second only to that of upper respiratory tract infections in adolescents and young adults
- Etiologic agents: viral (most common, including rotavirus and Norwalk and Noro virus), bacterial, and protozoan *(Giardia lamblia)*
- Peak incidence: winter in cities and summer in rural or outdoor sports
- **Symptoms:** nausea, vomiting, abdominal cramps, diarrhea, fever, and myalgia

#### Treatment

- Usually self-limited (2–3 days)
- Clear and electrolyte-containing fluids (e.g., sport drinks) are cornerstone; replace fluid loss liter for liter
- Assess the severity of dehydration (body weight, urine output, and blood pressure) before strenuous practice or game
- Antimotility drugs may be effective for abdominal cramps but may also prolong the carrier state of certain organisms: loperamide (Imodium) and diphenoxylate hydrochloride with atropine (Lomotil)

### "Traveler's Diarrhea"

- 90% caused by bacteria, with *E. coli* being the predominant organism; other bacteria include: *Salmonella*, *Shigella*, *Vibrio*, and *Campylobacter jejuni*. Viruses comprise 5%–10%, and parasites comprise a small percentage.

#### Treatment

- Depends on severity of illness
- Adequate fluid replacement essential, and most cases are self-limiting and resolve within 3–5 days
- Antibiotics may be warranted in certain circumstances, and therapy may be guided by the region of travel. Antibiotics shorten disease course and severity but may have side effects, yet many prefer treatment with antibiotics.
- Recommended azithromycin 1000 mg PO × 1 over traditional fluoroquinolone owing to equal efficacy and the association of fluoroquinolone use with tendinopathy and tendon rupture; recommended Imodium with antibiotic administration for symptomatic relief
- Indications for antibiotics may include severe diarrhea, cramping, fever, pus, or mucus in the stool, but bloody diarrhea can reflect an enterohemorrhagic *E. coli* infection, an uncommon cause of traveler's diarrhea, for which, antibiotic treatment has been associated with an increased risk of hemolytic–uremic syndrome In addition, *Salmonella*, *Shigella*, and *Campylobacter* may cause bloody diarrhea but are uncommon with traveler's diarrhea and are typically self-limiting.
- Stool studies are recommended before antibiotic administration. Monitor for persistent symptoms (antibiotic resistance) or worsening colitis *(Clostridium difficile)*.

#### Prevention

- Consider rifaximin for prophylaxis over bismuth salicylate. Large doses of bismuth salicylate required for prevention and increased risk of salicylate toxicity. Moreover, bismuth may reduce the absorption of antibiotics.
- Return to competition limited only by hydration status, infective nature of problems, symptom complex (i.e., frequent diarrhea), and reconditioning
- Probiotics have shown potential in small-scale studies to reduce GI symptoms caused by intraluminal floral shifts that occur 1–2 weeks after antibiotic use.

### Celiac Disease

- Hereditary autoimmune disorder to gluten that damages small intestinal mucosa, which results in villous atrophy and decreased nutrient absorption; includes a wide variety of symptoms: diarrhea, bloating, vomiting, fatigue, anemia, arthralgia, and myalgia (Fig. 30.4)
- Initial work up should include tissue transglutaminase antibody level. The gold standard for diagnosis is a biopsy of the small intestine that demonstrates blunted villi.
- Avoidance of gluten is the only known treatment.
- Gluten-free diets have not been shown to enhance performance or decrease exercise-associated GI symptoms in athletes with no celiac disease.

### Irritable Bowel Syndrome

- Exercise has a known benefit on regulation of IBS.
- Divided into various types based on predominance of symptoms: constipation predominant, diarrhea predominant, mixed constipation, and diarrhea
- Constipation predominant: consider fiber or polyethylene glycol. Diarrhea predominant: consider Loperamide as first line treatment. For significant pain: consider dicyclomine or hyoscyamine Be wary of side effects of anticholinergic medications; TCA's and SSRI's may be considered as well.

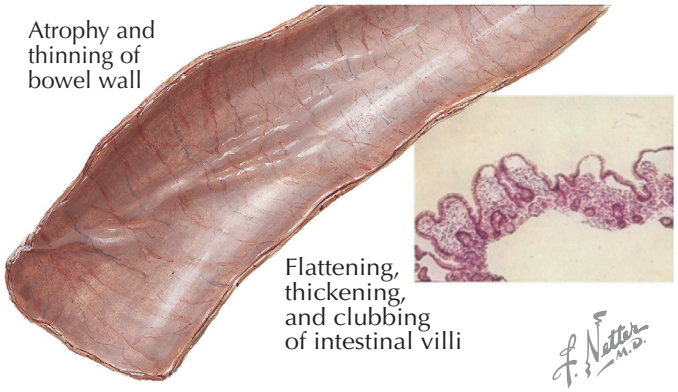


**Figure 30.4.** Celiac disease enteropathy.

## ACKNOWLEDGMENT

The opinions in this chapter are those of the authors and should not be construed as official policy of the Department of Defense, the Department of the Army or the Department of the Air Force.

## RECOMMENDED READINGS

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## RECOMMENDED READINGS


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# 31 HEMATOLOGIC PROBLEMS IN ATHLETES

Matthew R. Pollack • Jason A. Robertson • Tracy R. Ray

## SPORTS/DILUTIONAL ANEMIA

**Description:** Known as *dilutional anemia* or *pseudoanemia*

**Epidemiology:** The most common cause of anemia found in the athletic population. Dilutional pseudoanemia is not pathologic but rather an adaptation to endurance training and normalizes after training cessation. It is postulated to occur as a result of plasma volume expansion. It is hypothesized that dilutional anemia enhances the efficiency of oxygen delivery by decreasing blood viscosity and increasing cardiac output.

**Etiology:** Dilutional anemia occurs when the plasma expansion is greater than the red blood cell (RBC) mass increase. Typically, there is no change or an actual increase in RBC mass and total RBCs. Within 3–5 hours after exercise, plasma volume levels equilibrate, and subsequently, volume expansion occurs secondary to an increase in renin, aldosterone, and vasopressin levels, along with an increase in albumin production.

**Laboratory tests:** Laboratory test results reveal mild anemia (hemoglobin levels: 13 g/dL in men and 11.5 g/dL in women), which resolves within 3–5 days of discontinuing exercise.

**Treatment:** Dilutional anemia should not negatively affect athletic performance, and no treatment is required.

## IRON-DEFICIENCY ANEMIA

**Epidemiology:** Iron-deficiency anemia is the most common true anemia in athletes and the most common nutritional deficiency in the United States, affecting 3%–5% of women and <1% of men.

**Presentation:** It may be asymptomatic but often presents with weakness, lassitude, palpitations, shortness of breath, and pica (craving for starch, ice, or clay). Paleness, glossitis, angular cheilitis, and koilonychias (spoon-shaped nails) may be found in severe cases (Fig. 31.1).

**Etiology:** It is primarily caused by insufficient iron uptake from the gut or increased loss of iron; it is important to differentiate the cause. Most commonly, the blood loss results from menstruation or the gastrointestinal tract, but other causes include sweating and iron sequestration in response to inflammation. Hepcidin, a peptide hormone that inhibits iron absorption, may increase in response to exercise. In addition, nonsteroidal antiinflammatory (NSAID)-induced gastritis may frequently occur in athletes.

**Laboratory testing:** A low hemoglobin level in adults (<12 g/dL in women and <14 g/dL in men), mean corpuscular volume <75 fL, a hypochromic and microcytic blood smear, low serum iron with high total iron-binding capacity, and serum ferritin levels <12 µg/L

**Treatment:** Oral iron therapy in the form of elemental iron, 50 mg three times a day.

Absorption is best between meals, and orange juice or ascorbic acid increases absorption. Treatment should be continued for 6–12 months to completely replenish iron stores. Empiric treatment in borderline cases is warranted for 8 weeks, with the aim of a 1-g increase in hemoglobin. Women with documented cases of recurrent iron deficiency should be administered prophylactic doses of iron.

## IRON DEFICIENCY WITHOUT ANEMIA

**Overview:** Iron deficiency without anemia affects 12%–16% of premenopausal women and 2% of adult men. Importance in athletic performance is unclear and controversial with certain studies demonstrating an effect on $\dot{V}O_2$ max and several others reporting a decline in physical and cognitive function in deficient athletes, specifically in endurance athletes such as marathoners.

**Laboratory testing:** Laboratory test results will show decreased serum ferritin levels with normal hemoglobin.

**Treatment:** There are conflicting studies on the effects of iron deficiency without anemia on athletic performance. If an athlete presents with iron deficiency and borderline or low hemoglobin, a trial of oral iron therapy may be considered. Several studies have advocated supplementation if basal ferritin is <35 mg/L. Unnecessary treatment with iron can lead to gastrointestinal disturbances and occasionally more serious issues such as hemosiderosis.

## FOOT-STRIKE HEMOLYSIS/EXERCISE-INDUCED HEMOLYSIS/HEMOGLOBINURIA

**Overview:** Rarely severe enough to cause clinically significant anemia or iron deficiency

Typically, healthy individuals can counteract the degree of hemolysis with reticulocytosis.

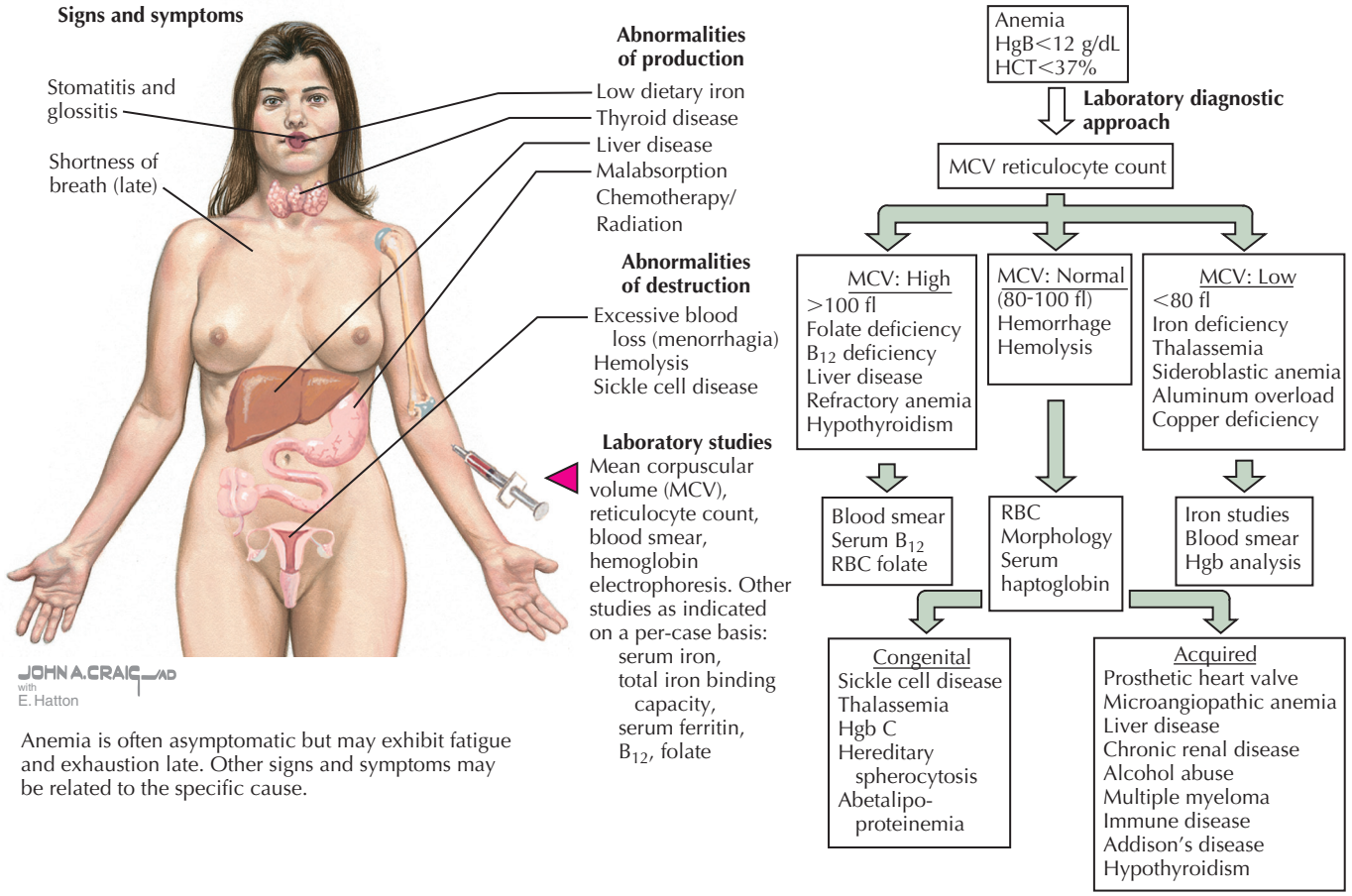
**Etiology:** Mechanical forces from muscle contractions and heel-strike have been shown to be a reproducible cause of RBC hemolysis leading to hemoglobinuria. Studies have suggested that hemolysis in the plantar vessels is caused by a combination of poorly cushioned shoes, running style, and hard running surfaces. In addition, elevated body temperature aids in increased RBC fragility, leading to "runner's macrocytosis" secondary to the loss of the older microcytes. Hemolysis has been documented in swimmers, dancers, rowers, and triathletes. Once hemolysis occurs, hemoglobin is released into the intravascular space. Haptoglobin, an intravascular protein, binds to the free hemoglobin; once haptoglobin becomes saturated, the free hemoglobin spills into the urine, producing hemoglobinuria.

**Laboratory studies:** Urinalysis reveals hemoglobinuria, which resolves within 3–5 days with exercise cessation. Laboratory test results typically reveal a macrocytic anemia with an increased reticulocyte count. Haptoglobin levels may be low or immeasurable, leading to urinary loss of iron.

**Treatment:** Treatment is not necessary but includes changes in gait, shoes, running surfaces, and intensity of the training regimen.

## SICKLE CELL TRAIT (SCT)

**Overview:** Sickle cell trait (SCT) is present in 5%–8% of the African-American population. In SCT, both normal hemoglobin A and abnormal hemoglobin S are produced, which does not seem to affect exercise capacity. With exercise, acidosis occurs to improve oxygen delivery, which leads to an increase in deoxygenated hemoglobin levels. The NCAA recommends confirming the sickle cell status of all athletes during preparticipation physical evaluation to allow more targeted precautions for those at risk. Certain organizations do not support this position due to a concern for bias, discrimination, and lack of cost effectiveness and instead, recommend precautions be applied to all athletes. Factors that can put athletes at a risk of SCT collapse include dehydration, heat, altitude, and asthma. Moreover, individuals with SCT can exhibit hyposthenuria




Anemia is often asymptomatic but may exhibit fatigue and exhaustion late. Other signs and symptoms may be related to the specific cause.

**Figure 31.1.** Anemia. *HCT*, Hematocrit value; *HgB*, hemoglobin; *RBC*, red blood cell.

(inability to concentrate urine), which may put them at an even greater risk of dehydration.

**Laboratory testing:** Hemoglobin electrophoresis will reveal one gene for normal hemoglobin A and another for hemoglobin S.

**Risks:** Athletes with SCT have been shown to have an increased risk of sudden cardiac death (SCD), splenic infarction, and hematuria. In 1987, the US military found that recruits with SCT had a 28 times increased risk of SCD compared with African-American recruits who tested negative for SCT.

**Exercise guidelines:** Regular exercise provides several benefits for those with SCT, including the potential to reduce oxidative stress, inflammation, and blood viscosity. Guidelines to decrease the risks associated with SCT seek to prevent overexertion and dehydration while gradually acclimatizing athletes to conditioning in the heat and with altitude changes through activity and conditioning modifications. Educating the athletes as well as the coaches and medical staff is paramount in avoiding SCT-related collapse. Recommendations for exercise include encouraging preseason training and conditioning as well as a gradual progression of exercise, allowing for longer periods of recovery between repetitions and workouts.

## EFFORT-INDUCED THROMBOSIS

**Overview:** Also known as *Paget–Schroetter syndrome*, effort-induced thrombosis is a rare cause of upper extremity deep venous thrombosis but is more common in young athletes. It falls under the venous subset of thoracic outlet syndrome.

**Epidemiology:** Typically presents in athletes involved in strenuous upper extremity activities as well as "industrial athletes" (e.g., overhead workers and manual laborers)

Theoretically, veins of upper extremities become compressed by hypertrophied anterior scalene muscles or a cervical rib during strenuous upper extremity motion, which typically occurs in the dominant extremity. It can occur with normal anatomy if the arm is in extreme abduction/external rotation.

**Presentation:** Unilateral upper extremity symptoms, including pain and swelling, which worsen with activity; in cases of chronic compression, symptoms may not present until complete occlusion occurs.

**Diagnostics:** Ultrasonography is the initial noninvasive test of choice, but MRI and CT may also be used. A hypercoagulability workup should be conducted to rule out any underlying disease.

**Treatment:** May involve a combination of surgical decompression, anticoagulation, or thrombolytic therapy; symptoms should ultimately resolve with treatment

## VENOUS THROMBOEMBOLISM (VTE)

**Epidemiology:** Venous thromboembolism (VTE) affects 1 in 1000 people per year. Virchow's triad includes stasis, endothelial injury, and hypercoagulability and helps identify those with risk factors for thrombosis.

**Risk factors:** Risk factors for thrombosis in the athletic population include trauma, immobilization after injury, frequent travel,

dehydration and hemoconcentration after exertion, and oral contraceptives.

**Treatment:** Some studies have recommended the use of low-molecular-weight heparin (LMWH) for the prevention of VTE after an injury that requires immobilization or before air travel in those at risk, but it is expensive. Aspirin is relatively inexpensive but much less effective. Basic precautions during flights include hourly aisle walks, not crossing legs, wearing loose clothing, adequate hydration, and using aspirin or LMWH if at a risk of developing thrombosis. Those with risk factors should consider compression stockings and/or LMWH for flights longer than 6 hours. Treatment should be continued for 3 months in those with reversible risk factors and for 6 months in idiopathic cases. Recurrent episodes should be treated for 12 months, and after three episodes, patients should receive anticoagulants indefinitely.

**Exercise guidelines:** Athletes with an acute VTE on anticoagulation should not participate in contact or collision sports but may participate in noncontact sports and exercise with appropriate counseling.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# RENAL AND GENITOURINARY PROBLEMS

# 32

Jack Spittler • Kevin E. Burroughs

## ANATOMY

**Genitourinary system:** Composed of internal and external organs of the urinary and genital/reproductive systems. Both systems are contained in the abdominal and pelvic regions.

**Urinary system:** Comprises kidneys, ureters, bladder, and urethra

**Reproductive system:** Male (penis and testicles) and female (ovaries, fallopian tubes, uterus, vagina, and vulva)

**Female genitourinary system:** Situated within the pelvis, except for the vulva, which is external (Fig. 32.1A)

**Male genitourinary system**: The prostate and internal portion of the male urethra located within the pelvis; the penis, scrotum, and testes are located externally and are most vulnerable in men (see Fig. 32.1B)

**Kidneys:** Located in the retroperitoneal region from T12 through L3 vertebral level (Fig. 32.2); the right kidney lies slightly lower than the left owing to the position of the liver

The kidneys are contained in the cushion of pericapsular fat and protected by the posterior abdominal wall musculature (erector spinae muscles and latissimus dorsi).

**Ureters:** Run along the posterior peritoneal wall from the kidneys to the urinary bladder; also protected by muscles of the posterior abdominal wall; most vulnerable where they cross the brim of the pelvis

**Bladder:** When empty, lies completely within the pelvis and is protected anteriorly by pubic rami; therefore, is most vulnerable when full

## PHYSIOLOGY

- The major function of the kidney is to maintain the stable composition of the blood via regulation of fluid and electrolytes.
- Renal blood flow at rest is approximately 1100 mL/min and is approximately 20% of the cardiac output.
- Oxygen consumption of the kidneys at rest is 26 mL/min and is approximately 10% of the resting metabolism.
- Daily urine volume may vary from 500 mL to 15 L.
- Urine volume determined primarily by antidiuretic hormone (ADH)
  - ADH regulates water reabsorption by increasing permeability of distal tubules of the nephron and collecting duct.
  - ADH is released from the posterior pituitary in response to signals from the hypothalamus.
  - Primary stimuli for ADH release:
    - Increased plasma osmolality
    - Decreased blood volume
    - Decreased blood pressure
  - ADH release may increase three-fold during heavy exercise in order to prevent free water loss and dehydration.
- At rest, 15%–20% of renal plasma flow is continuously filtered by the glomeruli.
  - Results in 170 L of filtrate per day
  - 99% is reabsorbed in the tubular system
- Exercise results in a reduction of renal blood flow proportional to the intensity of activity owing to shunting of blood to the exercising muscles.
  - Mechanism is via constriction of afferent and efferent arterioles owing to increased circulating levels of epinephrine and norepinephrine.
  - Moderate exercise (50% $\dot{V}O_2$ max) results in a 30% reduction; strenuous exercise leads to a 40%–50% decrease in both renal blood flow and glomerular filtration rate (GFR).
  - During maximal exercise (65% $\dot{V}O_2$ max), there is a 75% reduction in renal blood blow, which is approximately 1% of the cardiac exercise output.
  - Decreases even more if an individual is dehydrated or uses NSAIDs
  - Usually returns to pre-exercise levels within 60 minutes

## HEMATURIA

- Can be gross or microscopic (Fig. 32.3)
- Microscopically defined as >3 red blood cells (RBCs) per high-power field.
- In athletes, rates of hematuria can be as high as 75%–80% and can occur in both contact and noncontact sports.
- Although hematuria typically resolves within 48–72 hours after exercise or athletic events, resolution may take up to 7 days in ultraendurance athletes.

### Etiology

Etiological factors can be categorized by location (kidney vs. bladder):

**Nontraumatic renal:** Decreased renal blood flow (RBF) (up to 50% reduction, with the decrease proportional to the intensity) leads to ischemia in the nephron, increased permeability, and subsequent passage of RBCs.

Also increased glomerular filtration pressure, secondary to efferent vasoconstriction, leads to passage of RBCs at the glomerulus.

Clots are usually not renal in etiology; dysmorphic cells are suggestive.

**Traumatic renal:** Direct contact (e.g., helmet, ski pole, or balance beam) vs. indirect trauma (e.g., jarring during running or jumping)

**Bladder:** Sports hematuria of bladder origin is almost always traumatic.

Can be due to a single, large blunt trauma or multiple lesser forces; repetitive contact of flaccid posterior bladder wall against anterior wall (trigone) is known as a "bladder slap"; it can occur in athletes participating in intense physical activities such as long-distance running, track, swimming, lacrosse, and football. Cystoscopy shows damage of the superficial urothelium or contusion. Incidence can be decreased if the bladder is partially filled and adequate hydration is maintained.

**Prostate/urethra:** Usually traumatic, most often observed in cyclists because of repetitive jarring or direct trauma from bicycle top tube; inappropriate seat height, tilt, and fore/aft adjustments may contribute

Other causes of hematuria:

- Nephrolithiasis
- Urinary tract infections
- Sickle cell anemia or other blood disorders
- Malignancy
- Drug or medication use (including penicillin, cephalexin, thiazides, allopurinol, nonsteroidal anti-inflammatory drugs [NSAIDs], aspirin, furosemide, and oral contraceptives)
- Rhabdomyolysis (hematuria on urinalysis [UA] with absent RBCs on microscopy)

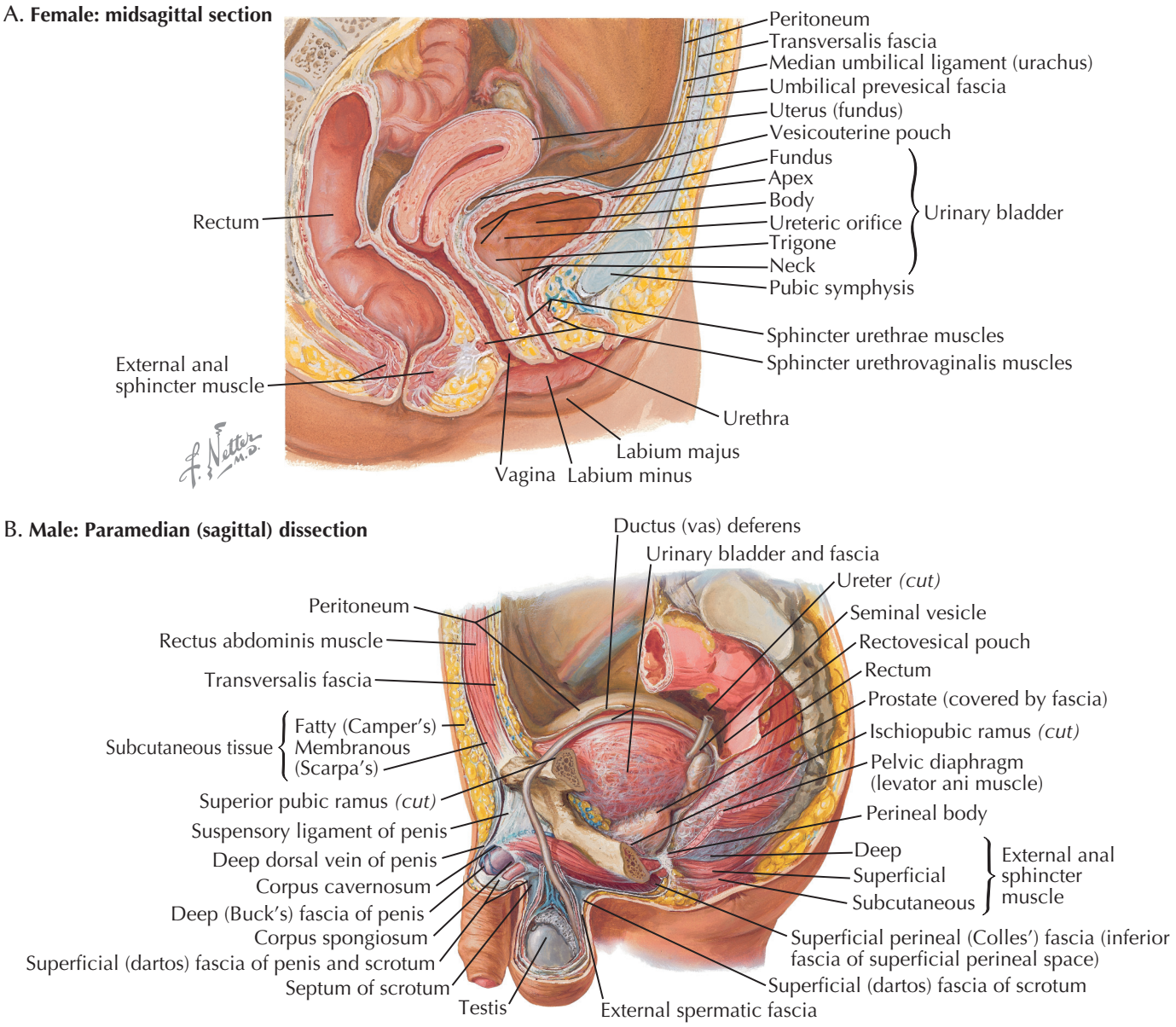


**Figure 32.1.** Genitourinary system.

- Red urine w/o RBCs attributed to beets, berries, food coloring, phenazopyridine, phenytoin, ibuprofen, nitrofurantoin, sulfamethoxazole, and rifampin

## Diagnostic Considerations

- Assess using urine dipstick and confirm with microscopy
- Timing of hematuria during urination is important to consider:
  - Initial hematuria (beginning of urination) often urethral in origin
  - Terminal hematuria (end of urination) may originate in the bladder or prostate (men)
- Mean corpuscular volume (MCV) and RBC morphology may help establish the origin of hematuria:
  - MCV <72 fL considered to be of glomerular origin
  - MCV >72 fL considered to be of nonglomerular origin
  - Casts or dysmorphic appearance of cells consistent with glomerular origin

## Treatment

- If no concerning history, physical examination or diagnosis, stop exercise and/or suspected medications and repeat UA after 24–72 hours
  - If urine clears after 24–72 hours and patient is <40 years old, is likely exercise-induced hematuria
  - If gross or microscopic hematuria persists or patient is >40 years old, further evaluation indicated:
    - Urine: culture, cytology
    - Blood: blood urea nitrogen (BUN), creatinine, PT/PTT, CBC, sickle cell screen (in African Americans)
    - Cystoscopy to exclude bladder lesions
  - If testing remains normal and hematuria persists, must consider intrinsic renal disease:
    - Creatinine clearance and protein excretion should be measured.
    - Upper urinary tract may be imaged using CT urography, renal ultrasound, and intravenous urography.

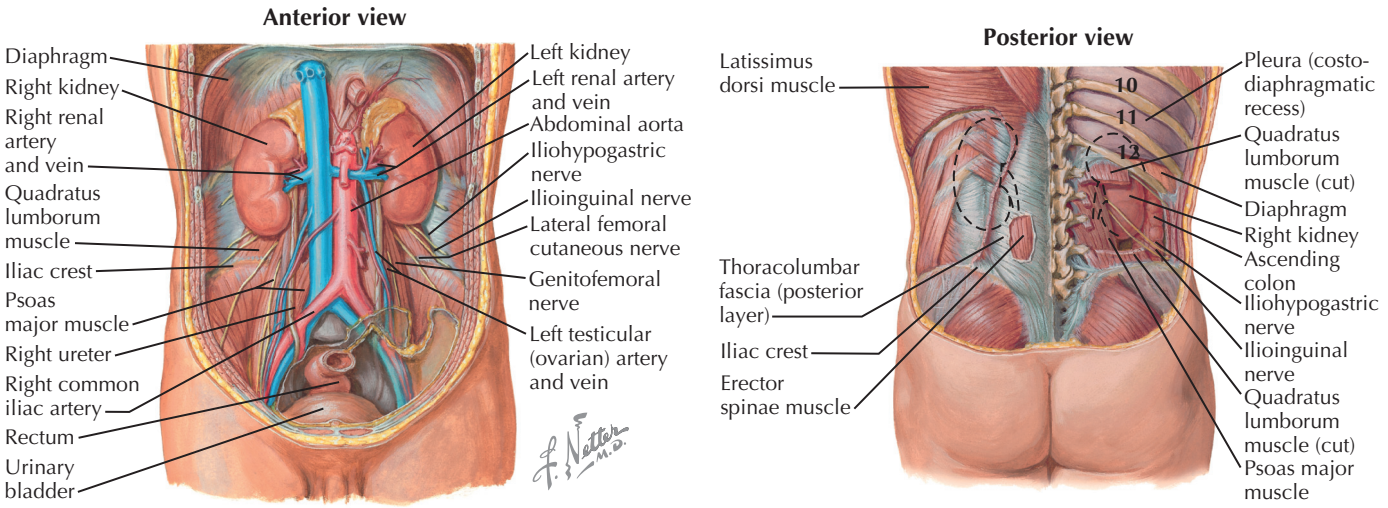


**Figure 32.2.** Kidneys in situ.

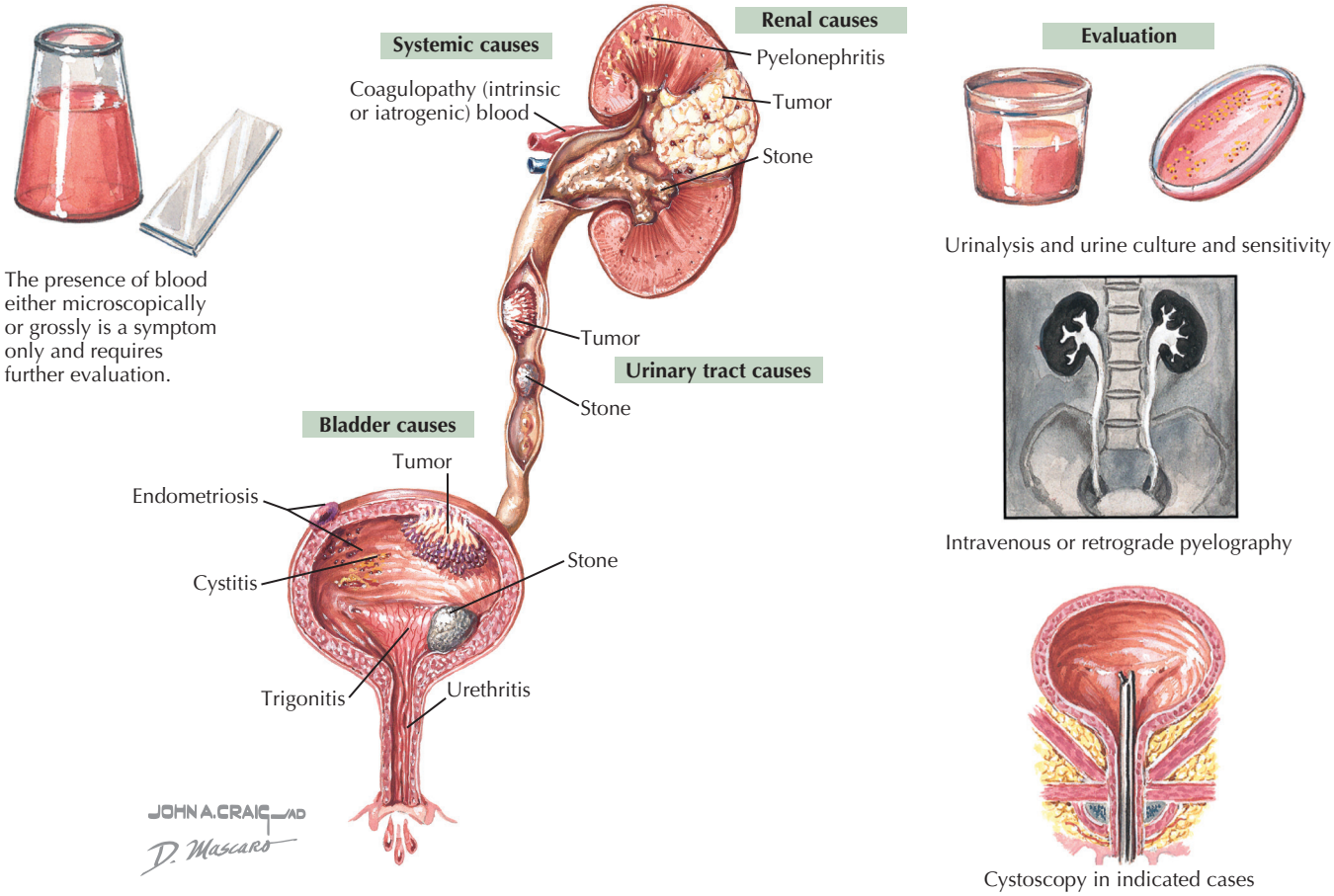


**Figure 32.3.** Hematuria.

- Nuclear renal scans, arteriography, retrograde pyelography, and voiding cystourethrography are less common, used when clinically indicated.
- Renal biopsy occasionally needed to establish medical cause and subsequent treatment of hematuria

## Return to Play

- Athletes with benign hematuria secondary to exercise may return to activity once hematuria resolves.
- Return to play for other causes of hematuria is diagnosis dependent.

### Prevention

- Athletes, particularly those with previous episodes of hematuria, should be encouraged to drink fluids before and during exercise to avoid dehydration.
- Keeping a slightly distended bladder during exercise can help prevent "bladder slap."

## PROTEINURIA

- May be present in up to 70% of athletes after exertion
- Exercise-induced proteinuria is a function of the intensity of exercise.
- Strenuous exercise can cause protein excretion to reach 1.5 mg/minute but usually does not increase beyond 1–2 g/day (normal 150–200 mg/day).
- Usually occurs within 30 minutes of exercise and clears in 24–48 hours

### Etiology

- Cause unknown, but the renin–angiotensin system (RAS) and prostaglandins (PGs) play a major role:
  - Plasma concentration of angiotensin II increases during exercise, resulting in increased protein filtration through glomerular membrane.
  - Moreover, strenuous exercise activates the sympathetic nervous system, which releases catecholamines, thereby increasing glomerular membrane permeability.
- In patients with underlying chronic kidney disease (CKD), low-intensity exercise does not increase proteinuria or lead to progression of CKD.

### Diagnostic Considerations

- Urine dipstick is a good screening tool but poorly quantifies the degree of proteinuria.
- Screening for proteinuria is not recommended during pre-preparticipation evaluation because the diagnostic utility is low.
- If routine UA shows protein and was collected within 24 hours of intense exercise, repeat testing in the absence of prior exercise to determine transient vs. persistent proteinuria
- Other causes of false-positive proteinuria on UA:
  - Highly concentrated urine (specific gravity >1.030)
  - Contamination with antiseptics
  - Pyridium (phenazopyridine HCl) use
  - Highly alkaline urine (pH > 8)
- Thorough history should be taken, including personal and family history of:
  - Renal disease
  - Anemia
  - Hypertension
  - Diabetes
  - Medication use (e.g., NSAIDs or antibiotics)
- Further workup may include:
  - First step is to quantify degree of proteinuria or microalbuminuria by using spot urine for protein, microalbumin, and creatinine (protein/creatinine, microalbumin/creatinine ratios)
  - Serum tests for renal function (BUN, creatinine)
  - 24-hour urine for total protein, creatinine, and creatinine clearance (sometimes indicated)
  - Fasting blood glucose or hemoglobin A1c
  - CBC or other tests, as medically indicated
  - Imaging: renal ultrasound and CT
  - Renal biopsy

### Treatment

- If proteinuria is transient or exercise-induced, no treatment is indicated.
- Otherwise, treatment depends on underlying cause.

### Return to Play

- If proteinuria clears after 24 hours, can return to activity or exercise
- If significant proteinuria persists, further evaluation as indicated before clearance

### Prevention

- No known prevention strategies needed because it is a normal physiologic process
- Exercise-induced proteinuria does not decrease with regular physical training, even in high-level athletes.

## ACUTE KIDNEY INJURY

- Acute kidney injury (AKI) is uncommon in sports, but occurrence is well documented.
- Becoming more prevalent in athletes with rise of ultraendurance events

### Etiology

- Combined effects of exercise-to-exhaustion, dehydration, hyperpyrexia, and rhabdomyolysis culminate in renal dysfunction by release of muscle enzymes and myoglobin that precipitates in renal tubules (Fig. 32.4).
- Over 25% of participants in ultraendurance races have evidence of AKI (at least 1.5–2× normal Cr).
- Underlying renal disease, dehydration, heat stress, genetic predisposition (i.e., sickle cell trait), and NSAID use are risk factors.

### Diagnostic Considerations

- AKI is defined as an increase in serum creatinine by ≥0.3 mg/dL or an increase that is ≥1.5 times the baseline value.
- Creatine kinase (CK) levels correlate well with myoglobin concentrations in the blood.
- Increased muscle breakdown increases the levels of circulating myoglobin, resulting in rhabdomyolysis, which becomes clinically significant when myoglobin precipitates in renal tubules.
- There is no commonly accepted CK threshold to guide hospitalization or treatment of rhabdomyolysis.
- One published algorithm uses CK levels of 20,000 IU/L as a threshold for treatment initiation in order to prevent renal failure, but most experts do not rely on a specific threshold.
- Levels >100,000 IU/L have been reported in asymptomatic individuals during/after exercise, and significant kidney injury can occur with CK levels of <20,000 IU/L.
- Hydration, serum creatinine, and urine flow should be carefully monitored in all athletes with rhabdomyolysis and potential kidney injury.
- AKI with rhabdomyolysis may also be associated with:
  - Disseminated intravascular coagulation (DIC)
  - Hyponatremia, hyperkalemia, hyperphosphatemia, and hyperuricemia
  - Hypocalcemia—caused by precipitation of calcium phosphate in muscles from hyperphosphatemia

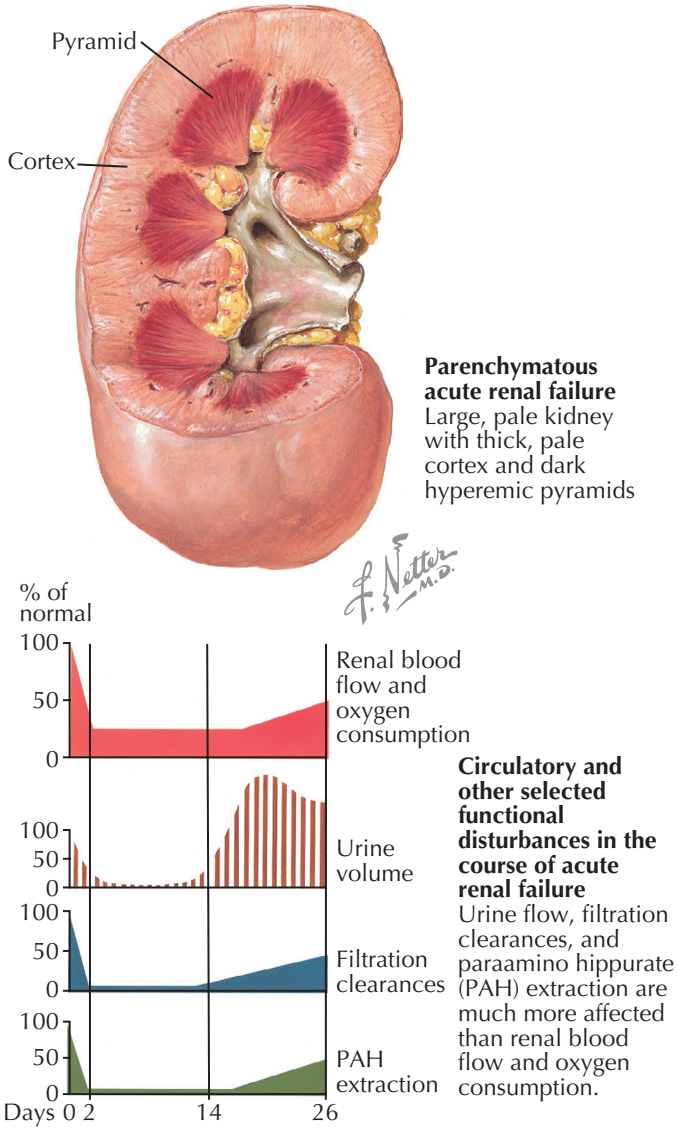


**Figure 32.4.** Acute renal failure.

## Treatment

- Treatment goal is to prevent complications, particularly renal failure
- Aggressive hydration, correction of electrolyte abnormalities, and maintenance of good urinary output are mainstays of therapy.
- Limited evidence supports that urinary alkalinization may reduce kidney damage in rhabdomyolysis.
- Loop diuretics are reserved to treat patients with volume overload.
- Dialysis is occasionally needed.

## Return to Play

- Serum creatinine, CK, electrolytes, and urinary output should return to baseline before return to play is initiated.

## Prevention

- Prevention is primarily related to appropriate hydration before and during exercise.
- Adequate training, heat acclimatization, and avoidance of NSAIDs can also be utilized in prevention.

# MEDICATION SIDE EFFECTS

## NSAIDs

- NSAIDs may have deleterious effects because of their ability to inhibit cyclooxygenase within the kidney (Fig. 32.5).
- Cyclooxygenase is the rate-limiting enzyme for synthesis of PGs.
- Vasodilatory $PGE_2$ and $PGI_2$ play a protective role in the kidney by modulating renal vasoconstriction caused by:
  - Increased renal sympathetic activity
  - Renin-angiotensin II
  - Circulating catecholamines
- Under stressful conditions (salt restriction, dehydration, and heat), ibuprofen decreases GFR after 45 minutes of exercise at 65% $VO_2$ max in comparison with placebo or acetaminophen.
- Both indomethacin and celecoxib have shown decrease in free water clearance, which in certain environments or conditions, can predispose to hyponatremia.

## Creatine

- Creatine monohydrate is widely used as an ergogenic aid for muscle performance.
- Increases muscle stores of creatine, leading to greater ATP synthesis.
- In healthy individuals, there is no known link between creatine ingestion and renal dysfunction if used for a short duration at typical doses (loading dose 20 g/day × 5 days and then ≤3 g/day as maintenance dose).
- There have been case reports of acute renal failure in long-term, high-dose users.
- Avoid if known renal disease
- End product of creatine is creatinine, may affect reliability of using creatinine as a marker of renal function
- Alternative measure of renal function is albumin excretion rate (normal <20 mcg/min).

# STRESS INCONTINENCE

- Defined as involuntary loss of urine during physical exertion
- Women twice as likely to be affected as men
- 28%–47% of regularly exercising women report some degree of incontinence.

## Etiology

- Cause is bladder outlet incompetence, history of childbirth common
- Urethral sphincters:
  - Internal sphincter composed of smooth muscles and under *involuntary* control
    - Prevents urine leakage via contraction under sympathetic stimulation
  - External urethral sphincter composed of skeletal muscles and under *voluntary* control
    - Prevents urine leakage via contraction under somatic nervous system stimulation
- Sports requiring jumping, running, or prolonged Valsalva maneuver may exacerbate

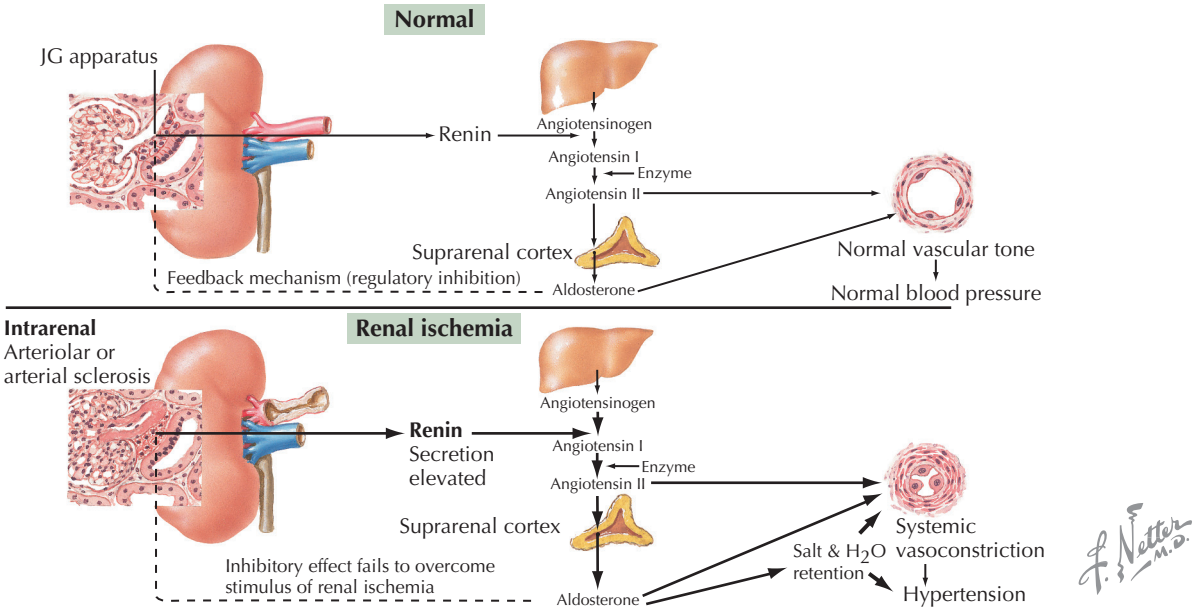


**Figure 32.5.** Renovascular hypertension.

## Diagnostic Considerations

- Usually diagnosed clinically and does not require further testing

## Treatment

- **Behavioral:** emphasis on establishing appropriate pelvic muscle function:
  - Kegel exercises, other pelvic floor exercises
  - Vaginal cones (or vaginal weights)
- **Pharmacologic:** Alpha-adrenergic agents (e.g., phenylpropanolamine) often effective in controlling the internal urethral sphincter
- **Surgical:** often effective, but should be considered only if conservative measures fail

# INGUINAL HERNIA

- Protrusion of abdominal contents into the inguinal canal
- More likely to occur in men than in women owing to residual space left from descent of testicles into scrotum from the abdomen

## Etiology

- Caused by weakness in the abdominal wall in conjunction with elevated abdominal pressure
- Most contain only soft tissues but may contain omentum or intestines.

## Diagnostic Considerations

- May be symptomatic or asymptomatic
- Men often have a swollen scrotum and women may have a bulge in the labia.
- Palpable mass felt in the inguinal canal, often more prominent with Valsalva
- Signs of strangulated hernia: sudden pain, nausea, and vomiting

## Treatment

- Symptomatic hernias often require surgical treatment at some point.
- May be monitored if asymptomatic and does not significantly limit activity
- Emergent treatment indicated for strangulated hernia

## Return to Play

- Athletes with asymptomatic inguinal hernias may participate in all sports.
- If symptomatic, may return to play based on severity of symptoms

# RENAL/URINARY TRACT INJURY

## Renal

- Kidney most common traumatic genitourinary injury
- Exact incidence in athletics unknown, but approximately 245,000 cases of traumatic renal injury reported worldwide/year

### Etiology

- Most cases in sports caused by a direct blow to the flank
- In athletics, these are often isolated injuries, opposed to motor vehicle accidents or falls, which often result in multiple traumatic injuries.

### Diagnostic Considerations/Treatment/Return to Play

- May have flank pain, CVA tenderness, ecchymosis, and hematuria (Fig. 32.6)
- No correlation between the amount of hematuria and degree of injury
- Hypovolemic shock may result from extensive bleeding; hence, it is important to monitor vital signs and hemodynamic status.
- Ultrasound (US) may identify renal lacerations but cannot definitively assess depth/extent.
- US helps to identify patients that require more aggressive radiologic investigation.

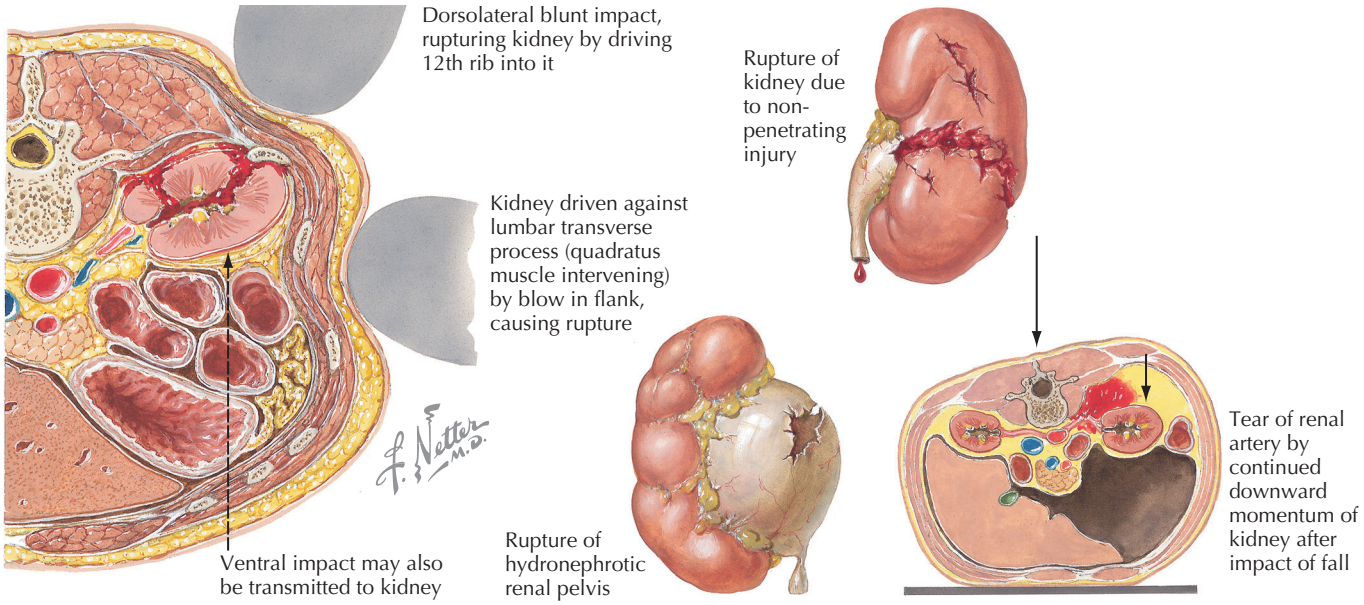


**Figure 32.6.** Renal trauma—nonpenetrating trauma of kidney.

- CT of abdomen with IV contrast (gold standard) or IVP needed for definitive diagnosis.
- Examining physician should also check for injury to other abdominal organs.
- Focused Assessment with Sonography for Trauma (FAST) is a rapid bedside US examination that can evaluate abdominal organs and heart for bleeding:
  - Positive FAST will show a dark ("anechoic") collection of fluid
  - FAST exam has a sensitivity and specificity of >90%

### Five Classes of Renal Injury

Graded from 1 to 5 based on severity by the American Association for the Surgery of Trauma (AAST) (Fig. 32.7):

**Grade 1:** Contusion or subcapsular hematoma without parenchymal laceration; a majority of sports-related renal injuries. Hematuria (microscopic or gross) and flank pain may be present. Treatment usually comprises observation, bed rest, and repeat UA. No sports until hematuria clears and no contact sports for 6 weeks.

**Grade 2:** Nonexpanding perirenal hematoma (confined to renal retroperitoneum), or renal or cortical laceration <1 cm deep without urinary extravasation. IVP shows extravasation of dye. Radiographs may have loss of psoas shadow. Treatment is often observation, bed rest, and repeat UA. No sports until hematuria clears and no contact sports for 6 weeks.

**Grade 3:** Parenchymal laceration >1 cm deep into the cortex without collecting system rupture or urinary extravasation; IVP shows intact capsule with intrarenal extravasation and disruption of the pelvicalyceal system. Angiography can be used to further delineate surgical vs. nonsurgical cases. Treatment involves observation or surgery in more severe cases.

**Grade 4:** Major parenchymal laceration extending through the corticomedullary junction and into the collecting system (complete renal fracture), injury to main renal artery or vein with contained hemorrhage; rare in sports. IVP shows separation of pelvicalyceal system with intrarenal and extrarenal dye extravasation.

**Grade 5:** Multiple major lacerations resulting in a shattered kidney or renal pedicle injury; patient will present in hypovolemic shock; rare in sports; kidneys usually not visualized on IVP.

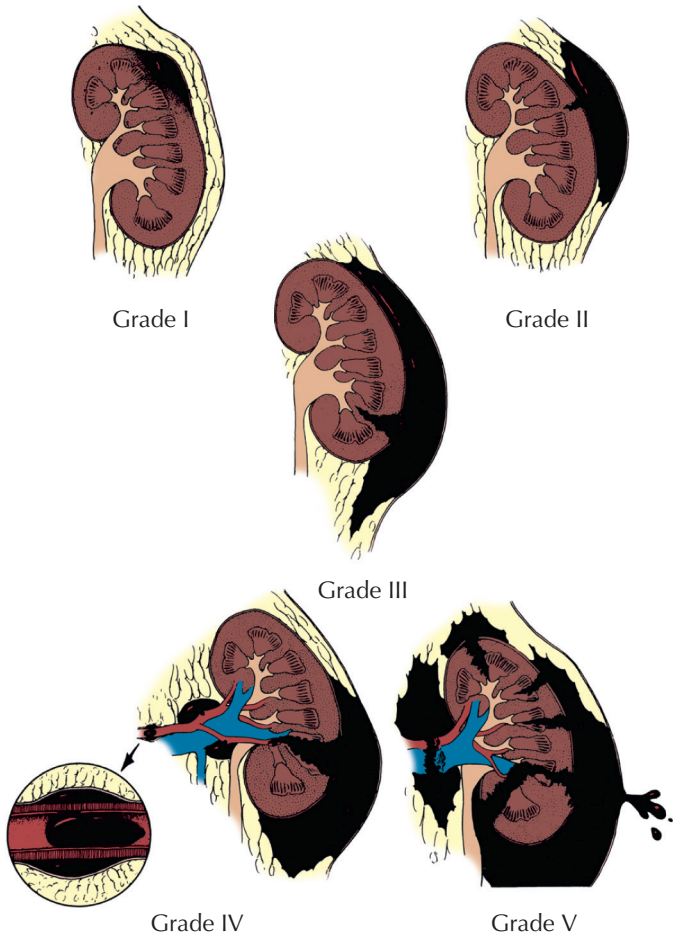


**Figure 32.7.** Classification of renal injuries by grade. Based on the organ injury scale of the American Association for the Surgery of Trauma. (From McAninch JW, Santucci RA. Genitourinary trauma. In: Walsh PC, Retik AB, Vaughan ED Jr, et al., eds. *Campbell's Urology*. 8th ed. Philadelphia: Elsevier; 2002.)

Selective renal arteriogram will show renal vascular damage. Treatment always involves surgery.

### Ureter

- Ureteral injuries rare in sports and represent only 1% of all genitourinary trauma cases

#### Etiology

- Usually a result of blunt trauma in sports
- Most often associated with significant renal damage
- Must also consider fracture of pelvic and lumbar vertebrae

#### Diagnostic Considerations

- No typical signs or symptoms, but hematuria is present in almost half of the patients
- Diagnosed by IVP or CT abdomen with IV contrast showing extravasation of contrast dye

#### Treatment

- Almost always requires surgery
- May involve nephrectomy if associated severe renal injury

#### Return to Play

- As this injury is very rare in sports, there are no standard return-to-play guidelines.

### Bladder

- Significant bladder injury is rare in sports.
- Major bladder injury is present in <5%–10% of pelvic fractures.

#### Etiology

- Bladder injuries most often related to blunt trauma of distended bladder.
- Two types trauma: contusion and rupture
- Repetitive contact of flaccid posterior bladder wall against anterior wall (trigone) is known as "bladder slap."
- Can occur in athletes participating in intense physical activities such as long-distance running, track, swimming, lacrosse, and football

#### Diagnostic Considerations

- Most patients with significant bladder trauma present with suprapubic tenderness and hematuria.
- May also have inability to void, suprapubic bruising, and abdominal distention
- Cystogram and retrograde pyelogram helpful for definitive diagnosis
- Contusion:
  - May have suprapubic pain and guarding
  - May pass small clots and have dysuria and hematuria
  - Degree of hematuria does not correlate well with the severity of injury
- Bladder rupture:
  - Rare in sports
  - Usually associated with pelvic fracture

#### Treatment

- Severe contusions may require use of indwelling catheter for 7–10 days with concurrent antibiotics.
- Bladder rupture requires immediate surgery.

#### Return to Play

- Return to play from contusion only after symptoms and hematuria resolve
- Return to play from rupture dependent on surgical outcome

#### Prevention

- Keeping bladder partially full (but not over distended) can help protect against bladder slap or bladder contusion/rupture.

## GENITAL INJURY

- More common in men compared to women because of anatomic differences with prominence of external genitalia

### Male Injuries

#### Testicles/Scrotum

- Testicles are paired organs that are contained within the scrotum.
- Significant testicular trauma can lead to impaired fertility and atrophic testes.

##### ETIOLOGY

- Direct trauma to scrotum may cause testicular contusion, rupture, and/or scrotal hematoma.
- May also develop testicular torsion due to twisting of the spermatic cord

##### DIAGNOSTIC CONSIDERATIONS

- May present with testicular pain, pallor, nausea, anxiety, and syncope
- If pain severe or persists for >1 hour, rule out testicular torsion and rupture
- Cremasteric reflex usually absent with torsion
- Doppler US can assess intra-/extratesticular hematoma, contusion, rupture, and torsion.

##### TREATMENT

- Ice and elevation utilized to control bleeding and swelling for scrotal hematoma or testicular contusion
- Surgical intervention for testicular rupture, tense hematoma, or testicular torsion

##### RETURN TO PLAY

- After symptoms of scrotal hematoma and testicular contusion resolve
- Depends on surgical intervention for more severe injuries
- Participation in contact sports with one testicle (either congenital or following trauma/orchiectomy) controversial because of the risk of infertility with damage to the remaining testicle; shared decision making between the athlete and the physician recommended

##### PREVENTION

- Use of a protective cup in contact sports may reduce the risk of injury.

#### Penis

- Penile injuries uncommon in sports

##### ETIOLOGY

- Trauma caused by straddle injuries or direct blow to pubis
- Partial or complete urethral rupture possible but complete rupture not described in sports

##### DIAGNOSTIC CONSIDERATIONS

- May present with immediate pain, swelling, and perineal ecchymosis
- Diagnostic retrograde urethrogram may be needed to check for significant injury.
- Erect penis susceptible to fracture of tunica albuginea:
  - Area of fracture swollen and ecchymotic
  - Penis bent to the affected side

- Penile frostbite described in runners wearing inadequate clothing in cold weather
- Traumatic irritation of pudendal nerve causing penile issues is not uncommon:
  - Particularly common in bicycle racers or touring cyclists
  - May cause priapism, impotence, paresthesia, or ischemic neuropathy

TREATMENT
- Complete urethral rupture is a urologic emergency that requires repair.
- Penile facture is also an emergency that requires hematoma evacuation and tunica albuginea repair.

PREVENTION
- Use of an athletic protective cup in contact sports may reduce the risk of penile injury.

### Female Injuries

- Vulvar hematoma may occur with trauma owing to prominent vascularity.
- May require evacuation if large enough
- Fall while water skiing may force water into the vagina ("water-skiing douche")
- This may lead to vaginal laceration and bleeding.

## SPECIAL POPULATIONS

### Wheelchair Athletes

- Neurologic control of urinary tract often lost after spinal cord injury (Fig. 32.8)
- Paraplegics and quadriplegics at a significant risk of:
  - Bladder and kidney infections
  - Kidney stones
  - Bladder distention
  - Urethral fistula
- Renal failure secondary to infection is a primary cause of death in people with spinal cord injury.
- Wheelchair athletes have demonstrated a lower incidence and frequency of urinary tract complications when compared with sedentary wheelchair users.
- Autonomic dysreflexia:
  - Athletes with spinal cord injury above T6 level are at a risk
  - Uncontrolled sympathetic response from noxious stimuli below the level of the lesion
  - Common symptoms include headache, hypertension, flushing, diaphoresis, and bradycardia.
  - Despite the risks associated with autonomic dysreflexia, certain athletes attempt to induce it for a competitive advantage, referred to as "boosting".
  - Autonomic dysreflexia self-induced by overdistending the bladder, sitting on sharp objects, or using tight leg straps
  - Theoretically, elevated blood pressure increases cardiac output and results in improved racing performance

### Cyclists

#### Pudendal Neuropathy

- Prevalence of perineal and penile/labial numbness is 50%–91% in competitive cyclists.
- Risk factors include age >50 years, high body weight, increased cycling time (>10 years, >3 hours/week).

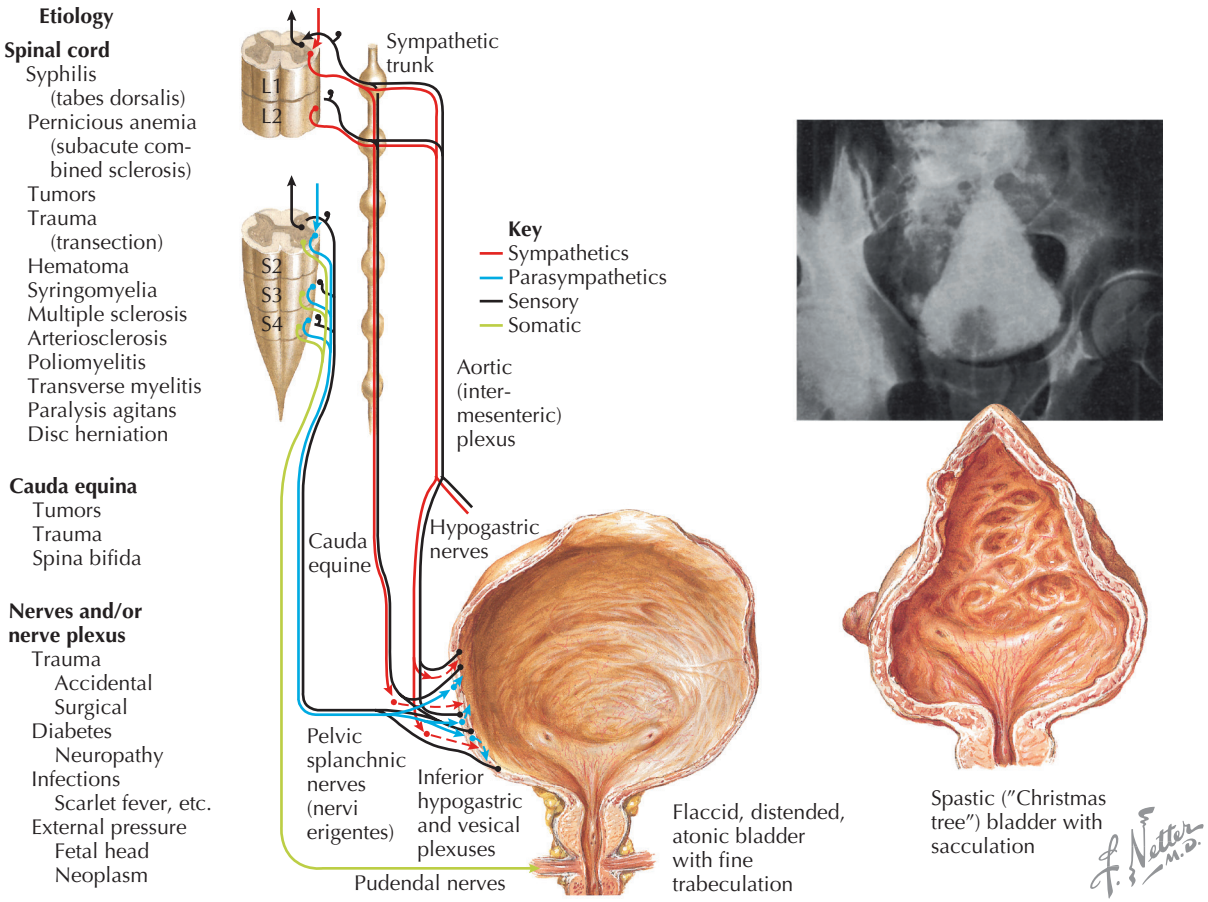


**Figure 32.8.** Neurogenic disorders of the urinary bladder.

- Treatment is cessation until numbness resolves:
  - Upon return to riding, intermittently rising from the saddle during rides
  - Appropriate bike fit important
  - Saddle individualized to riders, make sure is level, some utilize wide saddles (rear) with or without central cutout or nose.

### Erectile Dysfunction

- Risk factors are similar to pudendal neuropathy and etiology likely neurovascular.
- Central saddle cutouts popular but no evidence that decreases incidence
- Appropriate bike fit important; consider adjustments in handlebar height and saddle position

## Athletes Missing a Paired Organ

- No absolute contraindication to sports if single kidney, ovary, or testicle
- American Academy of Pediatrics Council on Sports Medicine and Fitness participation recommendations:
  - Solitary kidney: "qualified yes"
    - Individual assessment for contact, collision, and limited-contact sports
    - Incidence of unilateral renal agenesis is 1 : 500 to 1 : 1800, so likely that several athletes are unaware of having one kidney; most find it incidentally
    - Most physicians agree that athletes should be excluded from contact/collision sports if remaining kidney is ectopic, multicystic, or has any degree of obstruction or impairment of function.
    - Protective equipment may reduce the risk of injury to the remaining kidney
  - Solitary ovary: "qualified yes"
    - Risk of severe injury to remaining ovary is minimal
  - Solitary testicle: "qualified yes"
    - Certain sports may require use of a protective athletic cup.
- Participation is best decided on a case-by-case basis after careful education and shared decision-making between the athlete and the physician.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE ATHLETE WITH DIABETES

# 33

George D. Harris • Russell D. White

## GENERAL PRINCIPLES

- Diabetes mellitus (DM) is a chronic group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion leading to an absolute insulin deficiency due to beta-cell destruction (Type 1), by defects in insulin action due to progressive insulin secretory loss along with increasing insulin resistance (Type 2), or by other causes such as pregnancy, neonatal diabetes, maturity-onset diabetes (monogenic diabetes), or diseases of the exocrine pancreas or because of drug- or chemical-induced causes.
- A majority of patients with DM have Type 2 (90%). Athletes with DM range in sports participation from youth to competitive Olympic and professional.
- Each sport and the type of exercise have their own effects on DM management. Numerous factors affect glucose levels, including stress, level of hydration, rate of glycogenolysis and gluconeogenesis, and secretion of counter-regulatory hormones.
- Management includes excellent clinical care, continuous patient self-management, patient education, and longitudinal support to prevent long-term complications (renal failure, blindness, peripheral vascular disease, and peripheral neuropathy).

### Anatomy and Pathophysiology

- **Type 1 DM (T1DM**) has two subtypes: Type 1A and Type 1B. Each type can occur at any age, but typical onset is before the age of 30 years, with peak incidence during adolescence.
- Type 1A is an autoimmune disease characterized by cellular antibodies that may form against islet cells (ICA), insulin (IAA), and glutamic acid decarboxylase (GAD65). Type 1B is an idiopathic, nonautoimmune disease state with loss of beta-cell function. Both types are caused by loss of insulin secretion due to progressive loss of insulin production.
- These physiologic changes lead to increased hyperglycemia, weight loss, possible ketoacidosis, and possible death if insulin is not administered.
- **Type 2 DM (T2DM)** occurs in most patients older than 40 years and is characterized by defects in both insulin secretion and resistance to insulin action.
- Impaired insulin secretion, increased hepatic glucose production, and decreased muscle glucose uptake lead to increased levels of insulin production and eventual insulin resistance.
- Both genetic (family history or familial hyperlipidemia) and environmental (sedentary lifestyle or inappropriate diet with increased caloric intake) factors are involved in the development of insulin resistance. Insulin resistance is associated with obesity, hypertension, and hyperlipidemia and may precede the onset of diagnosed DM by 10–20 years.
- T2DM occurs in athletes with an increased body mass for their particular sport (football lineman or rugby players) or those who do not remain fit (e.g., certain baseball players).
- Patients with T1DM and T2DM benefit from regular exercise:
  - Exercise decreases the insulin resistance of peripheral tissues and alleviates the defect of insulin-stimulated glycogen metabolism in skeletal muscles;
  - It improves postprandial hyperglycemia and possibly postprandial insulin secretion.
- For patients with T2DM, both aerobic and resistance exercises can benefit glycemic control.

### Epidemiology

- The incidence of DM is increasing worldwide. It is estimated to affect 439 million individuals worldwide by the year 2030, and it is predicted that prediabetes and DM will affect 52% of the United States (US) population by 2020. Currently, approximately 24 million patients have DM, and 90%–95% of them have T2DM.

## GENERAL MEDICAL CONCERNS

- In comparison with inactive individuals, the additional demands of training and competition affect glucose homeostasis in athletes with DM, thus creating additional challenges.
- Challenges include the athlete's safety during athletic participation, adequate glucose monitoring, and diet and insulin adjustments for safe and effective athletic performance.
- Most competitive athletes learn to manage their DM during training and competition by trial and error while sharing personal experiences with other athletes.
- Medical concerns include unsafe dietary patterns, using nutritional supplements with no benefit or even detrimental effects, and using illegal drugs.
- Other concerns include the female athlete triad (amenorrhea, osteoporosis, and eating disorder), rapid weight loss to "make weight" in the respective wrestling or gymnastic competition, and excessive consumption of a single macronutrient (carbohydrate, protein, or fat) in certain athletes such as football players.
- In sports with weight categories (wrestling, boxing, and weightlifting), insulin is often omitted so that athletes can lose weight before weigh-ins; the consequence is poor glucose control and the risk of ketoacidosis.

### Athletes With Type 1 Diabetes Mellitus

- Appropriate hydration and maintenance of glucose levels can maximize performance. The excitement of competition can increase catecholamine release, resulting in hyperglycemia, but adjustment of insulin dosing is usually unnecessary.
- Athletes with T1DM learn their personal requirements from training and recognize that they always require certain amount of insulin supplementation.
- When an athlete with T1DM is insulin deficient, hyperglycemia occurs with a risk of further elevation in glucose levels, which may exacerbate or precipitate ketoacidosis. Moreover, osmotic diuresis with relative dehydration occurs as well.
- Morning endurance sports are less likely to cause hypoglycemia owing to physiologically elevated diurnal cortisol and growth hormone levels. Events that occur later in the day require adjustments in food and insulin.
- Certain athletes with T1DM intentionally avoid achieving good blood glucose control before their competitive event to have increased lipid utilization, which prevents exercise-induced hypoglycemia.

### Athletes With Type 2 Diabetes Mellitus

- The duration the athlete has been diagnosed with T2DM and specific sport will determine management. Endogenous insulin production or secretion early in the disease requires little, if any, exogenous insulin. These athletes maintain the ability to

physiologically decrease or increase endogenous insulin secretion and are generally able to achieve optimal glucose levels remain in the desired range.
- With progression of the disease, endogenous insulin secretion diminishes and exogenous insulin administration becomes necessary and must be adjusted to prevent hypoglycemia.
- It is not uncommon for such athletes to decrease exogenous insulin requirement by ≥50% with competition.
- Because athletes with T2DM also frequently use medications (e.g., insulin secretagogues and sensitizers), such medications are often decreased as training and insulin sensitivity increases and body fat decreases, resulting in an overall increase in lean body mass.

## SPORT-SPECIFIC ISSUES

### Endurance Athletes (Distance Cyclists and Runners)

- Appropriate glucose management before, during, and after exercise is crucial to care of an athlete with DM.
- Endurance runners often strive for optimal prerun blood glucose levels of 120–180 mg/dL, use minimal insulin, and estimate a glucose reduction of 10–15 mg/dL/mile.
- When running for 30–60 minutes, self-monitoring of blood glucose or continuous glucose monitoring (CGM) during the training period will delineate the athlete's predicted response.
- Distance cyclists and runners may choose to set the basal rate of insulin infusion via continuous subcutaneous insulin infusion (CSII) or insulin pump at a lower rate or may select a decreased long-acting basal insulin dosage (not uncommon to have to decrease by 50%). Carbohydrates are then gradually ingested to match energy utilization with exercise. By creating a steady-state balance between exercise requirements, basal insulin infusion, and ingestion of energy (carbohydrates), glucose levels are held constant (e.g., 130–150 mg/dL) over several hours.
- When a cyclist or runner with T1DM encounters a demanding section of a course, they will either adjust the basal rate of insulin downward or ingest additional carbohydrates to maintain optimal glucose levels.
- For runners and cyclists who are prone to hypoglycemia, performing a series of anaerobic sprints or resistance exercise before the aerobic endurance event may help prevent subsequent hypoglycemia.
- Running sports pose special problems for athletes with DM and include skin breakdown or other lesions due to vascular compromise or neuropathy.
- Athletes should travel with at least two pairs of well-fitting shoes that (i) are well broken-in, (ii) have no areas of material weakness or breakdown, and (iii) cause no discomfort with exercise. In addition, (iv) athletes should have their feet examined by the ATC/healthcare provider on a weekly basis, (5) avoid switching to a new pair of shoes on competition day, and (6) avoid using any alcohol-based lotion on the feet that may cause skin drying.

### Altitude Sports

- Glycemic control decreases in athletes with T1DM and may affect certain patients with T2DM at high altitudes. Hyperglycemia is due to (i) increased sympathetic tone with subsequent increased hepatic glucose production, (ii) increased insulin resistance, and (iii) loss of appetite at high altitude.
- Other problems encountered include variation in reliability of glucose monitors and adequate insulin protection from temperature extremes. As insulin temperature approaches freezing temperatures, it becomes less active.
- High-altitude athletes should maintain insulin close to their body to maintain optimal insulin temperature.

### Water Sports, Swimming, or Scuba

- Water sports limit the use of insulin pumps and continuous glucose monitors depending on the specific device and company model. While insulin pumps are water resistant and one model is waterproof to 12 feet for up to 24 hours, other models do not withstand pressure.
- Insulin pumps usually are removed before showering or bathing, whereas implanted glucose monitor skin sensors can remain in place; however, separate external glucose monitor devices are not waterproof.
- Scuba diving is discouraged for athletes with T1DM, although several accomplished divers dive with partners who are aware of their condition. Since recreational dives usually last 30–45 minutes, glucose monitoring can be accomplished on an interval basis at the surface level.

### Ice Hockey, Wrestling, or Football

- Use of external devices is discouraged in these sports. Wrestling and football competitions can cause damage to insulin pumps or external glucose monitors. Hockey players cannot wear any equipment that would be harmful to another player. A letter from a healthcare provider is required for any special medical equipment.
- Athletes often resort to intermittent insulin injections and glucose monitoring outside the competition area.

### Baseball or Softball

- These sports are ideal for athletes with DM because they require bursts of anaerobic energy without prolonged exercise periods.
- In the dugout, during each inning, athletes should test their glucose levels, ingest carbohydrates, and maintain hydration as needed.
- There has been some success in using insulin pumps with these sports.

### Triathlons

- Most diabetic triathletes have T1DM and, through their training, have learned to satisfactorily manage their insulin and glucose levels, making insulin adjustments on competition days based on training experiences.
- During the swimming element, either a waterproof pump is worn or the insulin pump and glucose monitor are removed.
- After exiting the water, athletes may attach an external glucose monitor and insulin pump. The process requires ≤10 seconds.
- Athletes may suspend, continue, or increase the same infusion based on glucose monitor readings without ceasing activity.
- With special permission from race officials, along the course route, athletes may be provided with glucose supplements.

## RISK OF EXERCISE

- Hypoglycemia is a major risk in athletes with T1DM, with additional risks of exacerbation of hyperglycemia and ketoacidosis. All diabetics are at an increased risk of coronary artery and peripheral vascular disease.
- Other associated risks include retinopathy, injury from neuropathy (ankle and foot injuries), and autonomic dysfunction (abnormal sweating mechanisms, affecting heat dissipation or abnormal heart rate response to exercise) (Fig. 33.1).
- Running is prohibited in patients with active proliferative retinopathy, which must be treated, stabilized, and cleared by an ophthalmologist before resuming activity.

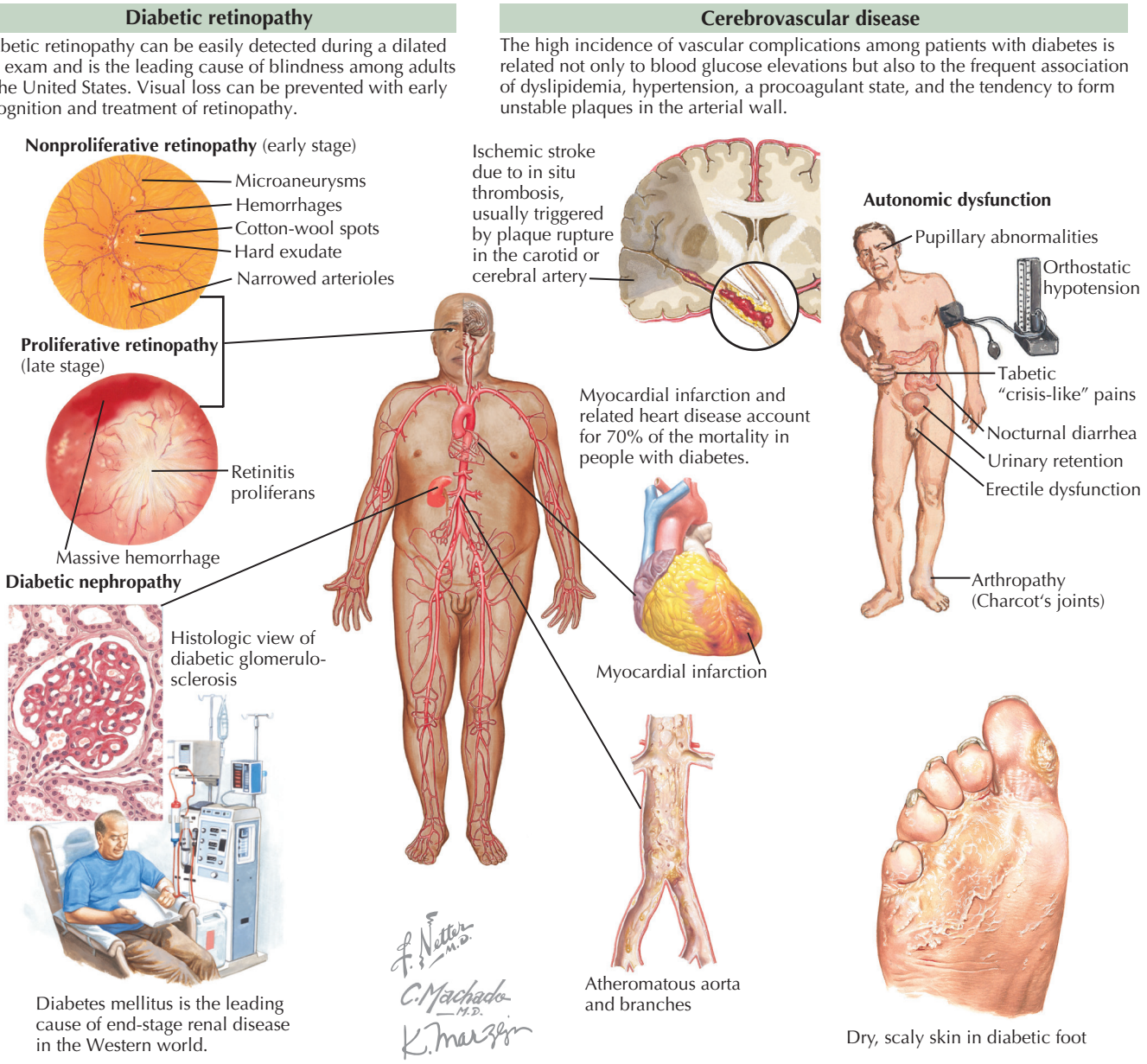


**Figure 33.1.** Signs of neuropathic joint disease and peripheral vascular disease.

## SPECIAL EQUIPMENT

- CSII is a device that contains a reservoir of rapid-acting insulin within a pump along with a small computer and catheter connected to a quick-release device inserted into the subcutaneous tissue of the athlete (abdomen). Basal insulin is provided in the form of a continuous slow infusion. A bolus of insulin can be administered to cover carbohydrate intake. The device can be interrupted or suspended for short periods of time (<60 minutes) without and adverse effects.
- In addition, CGM can be used to monitor glucose levels. A small sensor is implanted in the subcutaneous tissue, which analyzes glucose levels in the interstitial fluid. Severe low or high levels are signaled, allowing the athlete to have more control over his/her DM management and exercise programs.

## TREATMENT

### Principles of Glucose Management in Diabetes Mellitus

- The American Diabetes Association recommends following glycemic goals:
  - Fasting glucose of 90–130 mg/dL
  - 2-hour postprandial glucose <180 mg/dL
  - Limit postprandial glucose versus premeal glucose to ≤50 mg/dL
- The total daily dose (TDD) of insulin = 0.7 × weight in kg
  - Basal insulin dose per 24 hours = 50% of TDD
  - Bolus insulin dose per 24 hours = 50% of TDD
  - Percentages vary from individual to individual based on glucose measurements and activity, work, and exercise schedule.

**TABLE 33.1 BOLUS INSULIN PREPARATIONS**

| | Chemical | Onset of Action | Peak (Hours) | Duration (Hours) |
|---|---|---|---|---|
| **Rapid-Acting Class** | | | | |
| Aspart (NovoLog) | Insulin analog | 5–15 minutes | 1–2 | 3–4 |
| Glulisine (Apidra) | Insulin analog | 5–15 minutes | 1–2 | 3–4 |
| Lispro (Humalog) | Insulin analog | 5–15 minutes | 1–2 | 3–4 |
| **Short-Acting** | | | | |
| Regular | Human insulin | 30–60 minutes | 2–4 | 6–8 |
| **Intermediate-Acting** | | | | |
| NPH | Human insulin | 1–4 hours | 6–10 | 10–16 |
| **Premixed** | | | | |
| 70/30 (70% NPH/30% Reg) | Human insulin | 0.5–1.0 hour | 0.5–1.0 hour<br>1st peak-2–3<br>2nd peak hours later | 10–16 |
| 50/50 (50 NPH/50 Reg) | Human insulin | 0.5–1.0 hour | 0.5–1.0 hour<br>1st peak-2–3<br>2nd peak hours later | 10–16 |

**TABLE 33.2 BASAL INSULIN PREPARATIONS**

| | Chemical | Onset of Action | Peak | Duration (Hours) |
|---|---|---|---|---|
| **Basal Insulin Class** | | | | |
| Insulin glargine (Lantus) | Human insulin | 1–4 hours | None | 24 |
| Insulin detemir (Levemir) | Human insulin | 1–4 hours | None | 20–24 |
| Insulin degludec (Tresiba) | Human insulin | 30–90 minutes | None | >24 |
| **Premixed** | | | | |
| Humalog Mix 75/25 (75% insulin lispro protamine/25% insulin lispro) | Insulin analog | 15 minutes | 0.5–1.2 | 10–16 hours |
| Insulin degludec/Aspart (Ryzodeg 70/30) | Insulin analog | 10 minutes | None | >24 hours |
| BiAsp 70/30 (70% protamine aspart/30% aspart) | Insulin analog | 15 minutes | 0.5–1.2 | 10–16 hours |
| Lispro mix 50/50 | Insulin analog | 15 minutes | 0.5–1.2 | 10–16 hours |

- Prandial insulin can be determined by weight (0.1 units/ kg/ meal, see Example 1) or can be determined by pre-meal glucose measurements and calculated grams of carbohydrate in the upcoming meal (1 unit for each 30 g of glucose in the meal, see Example 2).
  - Example 1: 70 kg male × 0.1 units/meal = 7 units with that meal
  - Example 2: Pre-meal glucose = 150 mg% (Target level is 120 mg%)
  - For blood glucose levels above a described target number, i.e., one unit for each 15 g of glucose (150–120 = 30 mg%/15 = 2 units)
    PLUS
    60 g glucose meal (60 g/30 = 2 × 1 unit for each 30 g of glucose in meal = 2 units)
    Total units given pre=meal = 4 units.
- Patients with T1DM require bolus insulin before meals (Table 33.1) and basal insulin (Table 33.2) throughout the day.
- CSII (insulin pump therapy) is very effective in distance events. Several athletes wear a CGM system that renders current glucose levels and a trend graph. Distance athletes learn how to balance ingested carbohydrates and administered insulin with exercise and competition demands.
- Patients with T2DM *may* require insulin to achieve initial glycemic control but are typically managed with oral agents alone such as metformin (Table 33.3 and Fig. 33.2). Exercise and appropriate diet are beneficial to disease management and may lead to decreased medication doses with progressive training.

### Management of Hypoglycemia

- Hypoglycemia should be treated with 15–20 g of fast-acting carbohydrates, preferably glucose tablets designed to treat hypoglycemia or a sugar-sweetened beverage or juice.
- Repeat treatment if no improvement in symptoms or glucose levels after 15 minutes.
- If concern for recurrent hypoglycemia once the athlete resumes play, additional complex carbohydrates should be consumed before returning to activity.
- Hypoglycemia provokes a counter-regulatory hormonal response; thus, excessive carbohydrate intake should otherwise be avoided to prevent a significant hyperglycemic rebound.
- Severe hypoglycemia with cardiac arrhythmias, unconsciousness, or seizures is a life-threatening condition that requires an emergency response and parenteral glucagon or IV glucose administration of 1–3 ampules of 50% dextrose.

## PREVENTION

The best approach in management of hypoglycemia during exercise is to prevent its occurrence.

### Hypoglycemia

- Exercise, stress, level of hydration, rate of glycogenolysis and gluconeogenesis, and secretion of counter-regulatory hormones can affect glucose levels.
- Hypoglycemia in diabetic athletes can be either immediate or delayed.

**TABLE 33.3 PHARMACEUTICAL AGENTS AND MECHANISM OF ACTION FOR TYPE 2 DIABETES MELLITUS** (Fig. 33.2)

| Agents (Generic Name) | Mode of Action | Side Effects |
|---|---|---|
| 1. Sulfonylureas (SU)<br>Glyburide (DiaBeta, Micronase)<br>Glipizide (Glucotrol)<br>Glipizide-GITS (Glucotrol XL)<br>Glyburide (Micronized glynase)<br>Glimepiride (Amaryl)<br>Glipizide/metformin (Metaglip)<br>Glyburide/metformin (Glucovance)<br>Pioglitazone/glimepiride (Duetact) | Insulin secretagogues | Hypoglycemia in elderly; renal insufficiency; weight gain |
| 2. Biguanides<br>Metformin (MF) (Glucophage)<br>Metformin XL (Fortamet)<br>Metformin XR (Glucophage XR)<br>Glumetza (Metformin oral suspension) | Decreases hepatic glucose production and glucose absorption from GI tract. Increases peripheral glucose utilization | Caution in elderly and in renal failure. Withhold for contrast studies. Improves fertility in PCOS |
| 3. Alpha-glucosidase inhibitors<br>Acarbose (Precose)<br>Miglitol (Glyset) | Slows gut absorption of CHOs by inhibiting alpha-glucosidase enzymes | Hypoglycemia<br>Avoid in malabsorption syndromes; inflammatory bowel disease, bowel obstruction |
| 4. Thiazolidiones (TZDs)<br>Pioglitazone *(Actos)*<br>Rosiglitazone *(Avandia)* | Increases tissue sensitivity to insulin in skeletal muscles by activation of intracellular peroxisome proliferator-activated receptors. | Weight gain, resumption of ovulation, avoid in patients with Stage III/IV CHF; increased fracture risk in women |
| 5. Glitinides<br>Replaglinide *(Prandin)* | Rapid-acting insulin secretagogue with short duration of action sulfonylureas (same mechanisms but different binding site on pancreatic cells) | More effective in reducing postprandial glucose when compared with MF, SU, and TZDs. |
| 6. D2-dopamine agonist<br>bromocriptine *(Cycoset)* | Resets dopaminergic in patients with T2DM; can be used with all other oral agents within the central nervous system | Reduces glucose, triglycerides, insulin resistance, and insulin; should be taken within 2 hours of arising; ideal for shift workers who have disruption of their SCN (suprachiasmatic nucleus) pace-maker (body's circadian clock) |
| 7. Bile acid sequestrant<br>Colesevelam *(Welchol)* | Uncertain; may affect secretion of GLP-1 | NOT indicated as monotherapy in T2DM; may be used with metformin or metformin and sulfonylurea. Considered off-label use in prediabetes to reduce LDL and to preserve beta-cell function. May be considered in T2DM patients who have elevated LDL cholesterol |
| 8. DPP-4 inhibitors<br>Sitagliptin (Januvia)<br>Sitagliptin + metformin (Janumet/ Janumet XR)<br>Linagliptin (Tradjenta)<br>Linagliptin + metformin (Jentadueto)<br>Alogliptin (Nesina)<br>Alogliptin + metformin (Kazano)<br>Alogliptin + pioglitazone (Oseni) | Block the action of DPP-4 enzymes, resulting in a two- to three-fold increase in plasma endogenous GLP-1 levels | Skin rash and rhinitis; doses are based on renal status with the exception of linagliptin. Does NOT increase risk of CAD, HF, hospitalization for CHF |
| 9. GLP-1 receptor agonists<br>Exentatide (Byetta)<br>Liraglutide (Saxenda) (Victoza)<br>Exentatide QW<br>Lixisenatide (Lyxumia) | Enhance nutrient-stimulated secretion biomarkers. Exentatide is injected BID with meals; liraglutide is injected once daily without regard to meals; exentatide QW is injected once weekly. | Weight loss; favorable effect on CV; preserve B-cell function via activation of GLP-1 receptors on B-cells; inhibit glucagon secretion. Positive effect on endothelial cell function in humans; no cause of pancreatitis; delay gastric emptying; nausea is common side-effect; lower risk of hypoglycemia when used with insulin/insulin secretagogues |
| 10. Synthetic amylin analog<br>Pramlintide *(Symlin)* | Exogenous replacement with insulin deficiency. Injected pre-prandially; caution in patients with hypoglycemic unawareness | Adjunct therapy in amylin, which is deficient in proportion. T1DM or T2DM who have not achieved glucose control despite optimal insulin therapy |
| 11. SGLT2 inhibitors<br>Canagliflocin (Invokana)<br>Dapaglifozin (Farxiga)<br>Empagliflozin (Jardiance) | Reduces renal threshold of glucose. Inhibits sodium-glucose transporter 2 in tubules of glomeruli. Glucose is not absorbed into the plasma producing a decrease in FPG and postprandial glucose. | Urinary frequency, glycosuria, UTIs, decreases urine absorption from the SGL2 co-transporter GU tract; agents require dose-adjustment if co-administrated with other meds or in renal failure |

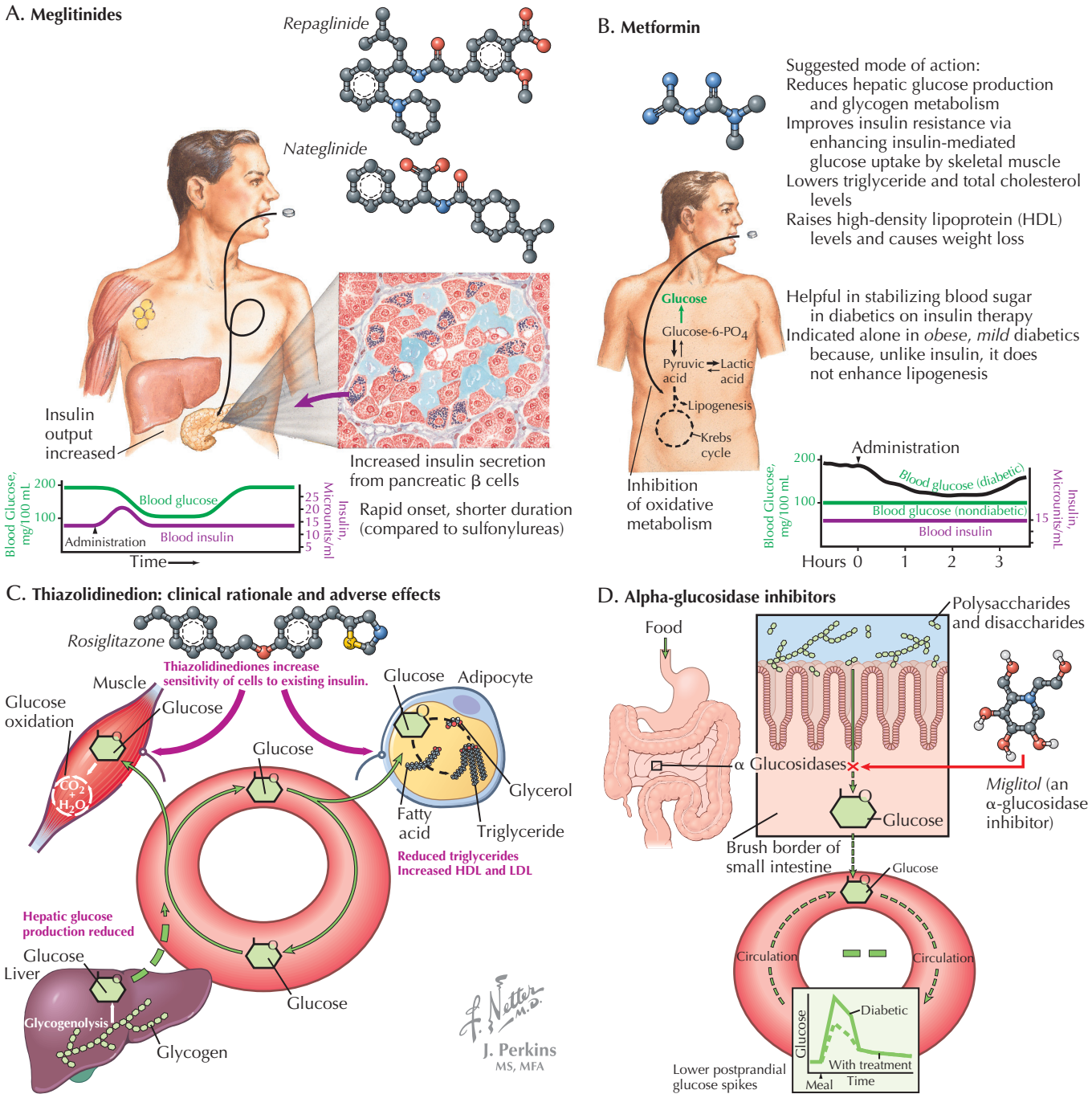


**Figure 33.2.** Medications that may contribute to hypoglycemia.

- Immediate hypoglycemia occurs during or shortly after exercise and is most common in patients with T1DM owing to inadequate caloric intake to meet metabolic demands.
- Other causes include excessive exogenous insulin administration or injection of insulin into the site of an exercising muscle or overlying subcutaneous tissue, resulting in increased rate of absorption.
- Symptoms and signs are primarily because of adrenergic causes (from the release of sympathomimetic mediators) and neuroglycopenic causes (from an inadequate amount of glucose being available to support brain activity).
- Adrenergic signs and symptoms consist of hunger, anxiety, sweating, tremor, tachycardia, palpitations, and/or a feeling of impending doom.
- Neuroglycopenic symptoms and signs include weakness or fatigue, slow or slurred speech, impaired performance of tasks, lack of coordination, blurred vision, odd behavior, confusion, vertigo, paresthesias, stupor, seizures, and loss of consciousness.
- To prevent immediate hypoglycemia, avoid injection of insulin into an exercising muscle (e.g., use of anterior thigh in a runner) and instead inject into the abdominal area.

- Calories should be continually replaced during periods of prolonged activity.
- Careful glucose monitoring during activities allows athletes to determine individual caloric requirements and make appropriate adjustments.

### Delayed Hypoglycemia (Nocturnal Hypoglycemia)

- Usually occurs 6–12 hours after exercise and has been reported to sustain for up to 28 hours after exercise.
- Pathophysiology involves vigorous exercise that severely depletes body glycogen stores, followed by inadequate replacement of glycogen stores in the postexercise interval ("golden replenishment period").
- Athletes either underestimate caloric requirements or ingest limited calories and defer eating until later. Over ensuing hours, liver and muscle tissues extract circulating blood glucose to replenish depleted glycogen stores and glycogen synthetase is activated.
- Peripheral muscle tissues are more sensitive to any available insulin after exercise.
- Subsequent severe and persistent delayed hypoglycemia often requires parenteral glucagon and continuous caloric intake over several hours to correct and replenish the depleted glycogen stores.
- Adequate replenishment of glycogen stores (1.5 g of carbohydrate per kg of body weight) immediately after exercise; it is critical to replace glycogen stores during the "golden recovery period" in order to avoid the risk of delayed hypoglycemia

## GUIDELINES FOR AVOIDING EXERCISE-ASSOCIATED HYPOGLYCEMIA

- These guidelines are only estimates that must be modified for a given person.
- Measure blood glucose (BG) before exercise and ingest carbohydrates using the following criteria if the intensity is low to moderate. Realize that high-intensity exercise leads to increased BG levels and hence, lesser carbohydrates, not more, are needed.
  - If BG is <120 mg/dL, initially consume 15 g of carbohydrate and then 30 g of carbohydrate per 60 minutes of light to moderate exercise (60% $VO_2$max).
  - If BG is 120–180 mg/dL, consume 30 g of carbohydrate per 60 minutes of light to moderate exercise.
  - If BG is 180–250 mg/dL, do not consume food before exercise. If the exercise is heavy and the duration exceeds 30 minutes, take second blood glucose reading and use the criteria stated in first two items of this list.
  - If BG is ≥250 mg/dL, check urine for ketones. Exercise is permissible if no ketones are present, but do not exercise if ketones are present or BG levels are >300 mg/dL because the action of counter-regulatory hormones may cause blood glucose to rise during exercise and increase ketone levels as well.
- **Easily digested carbohydrates should be readily available for supplemental feeding** with the athletic trainer or coach and on the athlete's person (e.g., pocket of running shorts or pack on bicycle). Glucose gel or tablets elicit faster rise in blood glucose than do fruit or fruit juices.
- **Decrease the dosage of insulin that peaks during exercise.** Rapid-acting analog insulin normally taken before meals might be decreased or deleted.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:
- eAppendix 33-1, Benefits of Exercise
- eAppendix 33-2, Exercise Guidelines
- eAppendix 33-3, Contraindications to Exercise

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

### eAppendix 33-1 Benefits of Exercise

- An athlete's motivation to improve athletic performance may enhance his or her diabetic management through more frequent blood glucose assessment and closer attention to diet.
- Consistent and good control of blood glucose reduces microvascular complications in T1DM and T2DM. A certain degree of reversal of these conditions is possible with prolonged near-normal glucose control.
- Physical trainability and performance are probably optimized when blood glucose is consistently good because of more normal substrate utilization, reduced protein degradation (which leads to greater muscle hypertrophy and possibly greater mitochondrial enzymes), greater muscle and liver glycogen, and increased body water with increased heat tolerance.
- Psychological effects of exercise include improved self-esteem and self-confidence.
- Reduction in cardiovascular disease risk factors, including reduced total cholesterol and low-density lipoprotein cholesterol, increased high-density lipoprotein cholesterol, reduced triglyceride level, reduced blood pressure, increased fibrinolysis, and reduced stress can be attributed to exercise.
- Increased insulin sensitivity: Exercise augments the action of glucose transporter 4 (GLUT-4).
- Reduced insulin or oral hypoglycemic medication doses often result; medications for certain patients with T2DM may be discontinued
- Moderate-intensity, high-volume resistance training also improves insulin sensitivity in patients with T2DM.

### eAppendix 33-2 Exercise Guidelines

- Establish good glucose control before starting an exercise program.
- Adjustment of diet and medication must be individualized while exercising. Consider consulting a physician trained in sports medicine with an understanding of DM management during exercise and sports.
- Blood glucose should be measured before, during, and after exercise to determine an individual's glycemic response to exercise.
- Determine individual blood glucose patterns in an athlete with tournaments, successive days of hard training, and reduced training days before competition.
- If ketosis exists before exercise, ketone production often increases. This effect is attributed to counter-regulatory hormones and often relative insulin deficiency. If blood glucose exceeds 250 mg/dL, check urine for ketones; if ketones are present, then DO NOT exercise.
- Pregame anxiety may mimic hypoglycemic symptoms, and the athlete may overeat and subsequently reduce insulin/medications, which produces hyperglycemia. Instead, measure blood glucose to define cause of symptoms and current blood glucose levels.
- Hypoglycemia is more likely to occur during exercise in the evening and LEAST likely to occur in the morning secondary to diurnal variations in endogenous cortisol and growth hormone, which elevate glucose levels.
- Reduce rapid-acting insulin before exercise as below:
  - Exercise lasting <1 hour—10% reduction
  - Exercise lasting 1–2 hours—40% reduction
  - Exercise lasting ≥2 hours—50% reduction
  - Further adjustments are based on historical glucose response to exercise.
- Consistent absorption and more predictable pharmacodynamics are achieved with rapid-acting insulin analogs (insulin lispro, insulin aspart, and insulin glulisine) and basal long-acting insulin analogs (insulin glargine and insulin detemir).
- Before special competition times, athletes should experiment with exercise, food intake, and medications at the same time of day as the scheduled competition.

### eAppendix 33-3 Contraindications to Exercise

- Presence of any additional risk factors for coronary artery disease requires evaluation.
- Exercise electrocardiogram is indicated in patients with any of the following:
  - Age >35 years
  - Age <25 years and:
    - T1DM for >15 years
    - T2DM for >10 years
  - Presence of any additional risk factor for coronary artery disease
  - Presence of microvascular disease
  - Peripheral artery disease
  - Autonomic neuropathy
- Proliferative retinopathy precludes:
  - Anaerobic exercise
  - Exercise involving jarring, straining, or Valsalva-type activity (e.g., weight-lifting, contact sports, high-impact aerobics, gymnastics, running, boxing, and volleyball)
  - Activity that dramatically increases heart rate and systolic blood pressure >170 mmHg
  - Inverted exercises (certain yoga positions, standing on head, and hanging upside down)
  - Scuba diving at deep depths, but is permitted at shallow depths (should be cleared by ophthalmologist following careful eye exam)

## RECOMMENDED READINGS


1. ADA Standards of Medical Care in Diabetes—2016. *Diabetes Care*. 2016;39(1).
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16. Yardley JE, Kenny GP, Perkins B, et al. Effects of performing resistance exercise before versus after aerobic exercise on glycemia in type 1 diabetes. *Diabetes Care*. 2012;35(4):669-675.

# 34 ECG INTERPRETATION IN ATHLETES

Henry F. Pelto • Jonathan A. Drezner

## INTRODUCTION

- Sudden death related to cardiovascular disorders is the most common cause of death among athletes during sports and exercise.
- Genetic cardiovascular disorders, such as cardiomyopathies and ion channelopathies, are the primary causes of sudden cardiac death (SCD) in athletes.
- Most of these disorders can be identified through characteristic changes on a resting 12-lead electrocardiogram (ECG)
- The heart undergoes physiologic changes in response to regular exercise (so-called "athlete's heart").
- These physiologic adaptations can manifest as changes seen on an ECG.
- Analyzing the ECG of an athlete requires careful interpretation to distinguish pathologic from physiologic patterns (see Fig. 34.1).
- The evolution of modern interpretation standards that account for the physiologic changes in an athlete's ECG have greatly improved specificity (i.e., lowered the false-positive rate) while maintaining sensitivity to detect conditions at an increased risk for SCD.

## NORMAL CARDIAC ADAPTATIONS IN ATHLETES

### Introduction

- Cardiac adaptations develop as a result of regular, sustained exercise.
- Electrical manifestations of this on an ECG reflect increase in cardiac chamber size and/or vagal tone.
- ECG findings related to increased cardiac chamber size include voltage criteria for left ventricular hypertrophy (LVH) and right ventricular hypertrophy (RVH) as well as incomplete right bundle branch block (RBBB).
- Findings consistent with increased vagal tone include early repolarization, sinus bradycardia, sinus arrhythmia, first-degree atrioventricular (AV) block, Mobitz type II second-degree AV block, as well as junctional and ectopic atrial rhythms.

### Specific Findings Related to Physiologic Adaptations to Exercise

- QRS voltage criteria for LVH
  - Definition: Sokolow–Lyon criteria: sum of the S wave in V1 and the R wave in V5 or V6 (using the largest R wave) as >3.5 mV
  - 64% of athlete ECGs will meet these criteria
  - Isolated finding with no other ECG abnormalities in <2% of individuals with hypertrophic cardiomyopathy (HCM)
  - When pathologic LVH is present such as in HCM, the voltage criteria for LVH are usually associated with other abnormal ECG findings (i.e., T-wave inversion, ST-segment depression, and pathologic Q waves).
- QRS voltage criteria for RVH
  - Definition: Sokolow–Lyon criteria: R wave in V1 + largest S wave in V5 or V6 >10.5 mV
  - 13% of athletes meet these criteria
  - Very poor correlation with arrhythmogenic right ventricular cardiomyopathy (ARVC), pulmonary hypertension, or other RV pathology
- Incomplete RBBB
  - Definition: QRS duration 100–120 ms with an RBBB pattern: terminal R wave in lead V1 (commonly characterized as an rSR' pattern) and wide terminal S wave in leads I and V6
  - Caused by delayed conduction owing to physiologic increase in RV size
- Early repolarization
  - Definition: elevation of the QRS-ST junction (J-point) by ≥0.1 mV often associated with late QRS slurring or notching (J wave), often affecting the inferior and/or lateral leads
  - Common in general population and more common in young, male, and black individuals
  - Available evidence suggests that all early repolarization patterns are normal in young athletes.
- Repolarization in black athletes
  - Up to 13% of black/African athletes demonstrate specific repolarization variant: convex ST segment and J-point elevation in the anterior leads (V1–V4) followed by T-wave inversion (see Fig. 34.2).
  - No association with cardiomyopathy after 5-year follow-up evaluation
  - T-wave inversion that extends to lateral leads (V5–V6) should always be considered abnormal and warrants careful evaluation to exclude cardiomyopathy.
- "Juvenile" ECG pattern
  - Definition: T-wave inversion in anterior precordial leads
  - T-wave inversions in V1–V3 in asymptomatic athletes aged <16 years (or prepubertal) should not trigger further evaluation.
  - T-wave inversion beyond V2 in those aged >16 years should raise concerns regarding ARVC.
- Sinus bradycardia
  - Definition: heart rate <60 beats per minute (bpm)
  - Present in 80% of highly trained athletes
  - Heart rate >30 bpm considered normal in trained athletes
  - Sinus bradycardia should resolve with exertion.
- Sinus arrhythmia
  - Definition: fluctuation of heart rate with breathing
  - Heart rate should correlate with breathing, without sustained acceleration or decelerations, and should resolve with exertion.
  - Not associated with symptoms to differentiate from sick sinus syndrome
- Junctional escape rhythm
  - Occurs with QRS rate faster than resting sinus rate caused by increased vagal tone
  - Narrow complex QRS with QRS morphology similar as sinus rhythm
  - Should have a rate <100 and regular RR interval
  - Sinus rhythm resumes with exercise and increase in heart rate.
- Ectopic atrial rhythm
  - P waves are present but different in morphology compared with sinus rhythm
  - 8% of athletes have ectopic atrial rhythm or junctional escape rhythm
- First-degree AV block
  - Definition: PR interval >200 ms
  - Occurs in 7.5% of athletes
  - Caused by increased vagal tone and should resolve with exertion

- Mobitz Type I (Wenckebach) second-degree AV block
  - Definition: progressive PR lengthening until a nonconducted P wave occurs (no QRS after P wave)
  - Caused by increased vagal tone: 1:1 P:QRS conduction should return with exertion

**ECG Findings In Athletes**

**Normal ECG Findings**
- Increased QRS voltage for LVH or RVH
- Incomplete RBBB
- Early repolarization/ST segment elevation
- 1° AV Block
- Mobitz Type I 2° AV Block
- Sinus bradycardia
- Sinus arrhythmia
- Ectopic atrial or junctional rhythm
- ST elevation followed by T wave inversion in V1-V4 in black athlete
- T wave inversion in V1-V3 if ≤ 16 years old

**Borderline ECG Findings**
- Complete RBBB
- Left axis deviation
- Left atrial enlargement
- Right axis deviation
- Right atrial enlargement

**Abnormal ECG Findings**
- T wave inversion
- ST segment depression
- Pathologic Q waves
- Complete LBBB
- QRS ≥ 140 ms duration
- Ventricular pre-excitation
- Prolonged QT interval
- Brugada Type 1 pattern
- Sinus bradycardia < 30 bpm
- PR interval >400 ms
- Mobitz Type II 2° AV block
- 3° AV block
- ≥ 2 PVCs
- Atrial tachyarrhythmias
- Ventricular arrhythmias

**Figure 34.1.** Normal ECG findings represent physiologic adaptations and do not require additional evaluation. Abnormal ECG findings may represent an underlying pathologic cardiac disorder and warrant further evaluation. Presence of two or more borderline findings warrants further evaluation, whereas a single borderline finding found in isolation and without other clinical markers of concern does not require additional testing.

## ECG CHANGES SUGGESTIVE OF CARDIOMYOPATHY

### Introduction

- Cardiomyopathies are a heterogeneous group of heart muscle diseases.
- Collectively, they are the leading identified cause of SCD in young athletes.
- Individuals with these diseases may be asymptomatic: often the presenting sign is sudden cardiac arrest.
- ECG is abnormal in 85%–95% of individuals with such diseases.

### Hypertrophic Cardiomyopathy (HCM)

- Heart muscle disease characterized by asymmetric LVH or septal hypertrophy, small LV chamber size, diastolic dysfunction, and myocyte disarray on histopathology. Some cases of HCM have LV outflow tract obstruction at rest or provoked by exercise.
- Hypertrophy is most common at the intraventricular septum but can also be found in the apex or in a concentric fashion.
- Prevalence of approximately 1:500 in adults (1:800–2600 in young competitive athletes)
- ECG findings are abnormal in >90% of patients with HCM.
- ECG findings suggestive of HCM (see definitions below): T-wave inversions in the lateral or inferolateral leads, ST-segment depression, pathologic Q waves, complete left bundle branch block (LBBB), nonspecific intraventricular conduction delay, and multiple PVCs
- Markedly abnormal ECG patterns involving deep T-wave inversion with ST-segment depression in the lateral (V5–V6, I, and aVL) or inferolateral (V5–V6, I and aVL, and II and aVF) leads warrants exclusion of apical HCM, which can be difficult to assess by echocardiogram alone; cardiac MRI is recommended when such ECG patterns are noted (see Fig. 34.3).

### Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- Heart muscle disease characterized by fibrofatty replacement of RV myocardium

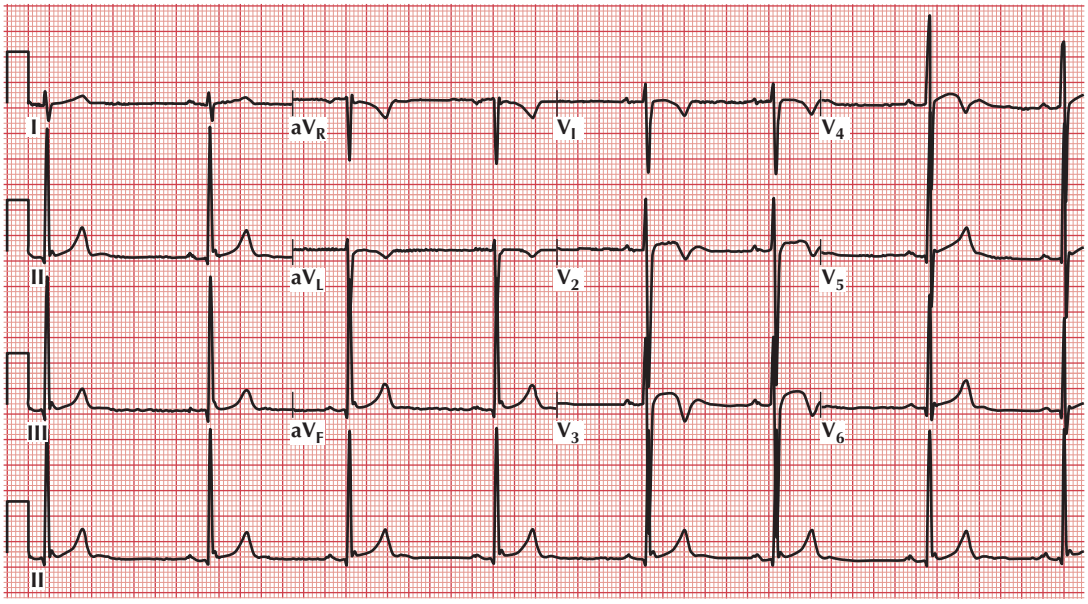


**Figure 34.2.** ECG from a 17-year-old asymptomatic black athlete. ECG demonstrates J-point elevation with a convex "domed" ST segment elevation followed by T-wave inversion confined to leads V1–V4. This is a normal repolarization variant in black athletes.

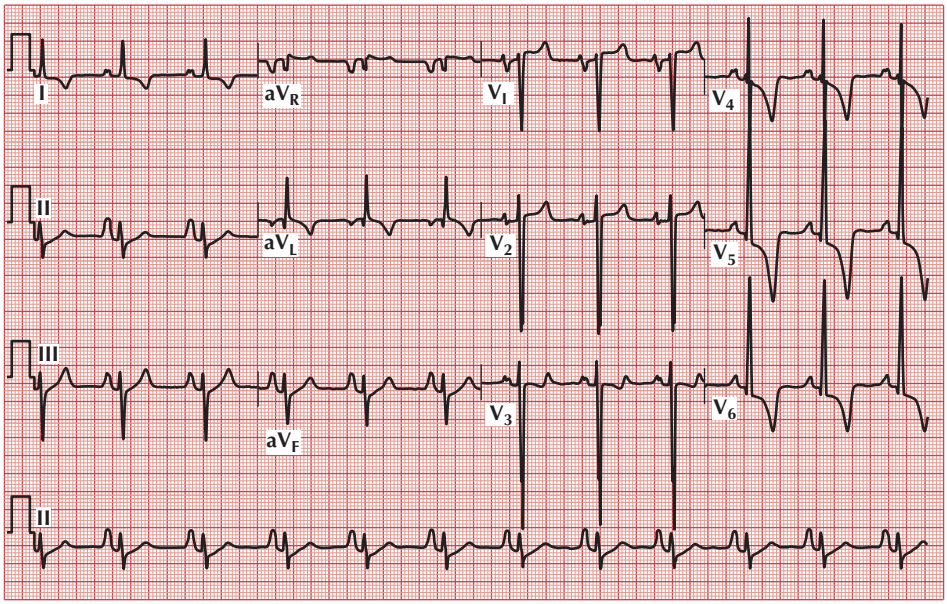


**Figure 34.3.** ECG from a patient with hypertrophic cardiomyopathy demonstrates deep T-wave inversion and ST-segment depression predominantly in the lateral leads (I, aVL, and V4–V6), left and right atrial enlargement, and left axis deviation.

- ECG findings suggestive of ARVC (see definitions below): T-wave inversion in anterior leads, ST-segment depression, multiple PVCs, epsilon wave, delayed S-wave upstroke, and low limb lead voltage
  - Epsilon wave: small negative deflection just after QRS in leads V1–V3
  - Delayed S-wave upstroke: > 55 ms from nadir of S wave to the end of the QRS complex
    - Must be in the absence of RBBB
  - Low limb lead voltage: QRS voltage <5 mm in leads I, II, and III

## Idiopathic Dilated Cardiomyopathy (DCM)

- Heart muscle disorder wherein weakened contractile function leads to heart chamber enlargement without associated increase in wall thickness and decrease in ejection fraction
- ECG findings suggestive of DCM (see definitions below): T-wave inversions in the anterior, lateral, and inferolateral leads, ST-segment depression, pathologic Q waves, complete LBBB, and nonspecific intraventricular conduction delay
  - Goldberger's triad (criteria suggestive of DCM): deep S waves in the anterior precordial leads, low limb lead voltage, and poor precordial R-wave progression

## Left Ventricular Noncompaction (LVNC)

- Heart muscle disorder characterized by poorly organized muscle fibers leading to deep trabeculations and thinning of the compacted myocardium
- Hypertrabeculations are more common in male and African-American athletes; thus, the clinical diagnosis usually requires LV dysfunction.
- ECG findings suggestive of LVNC (see definitions below): T-wave inversions in the lateral and inferolateral leads, ST-segment depression, pathologic Q waves, complete LBBB, and nonspecific intraventricular conduction delay

## Myocarditis

- Acquired disorder and thus not an inherited cardiomyopathy, but ECG abnormalities overlap with findings found in other myocardial diseases
- Acute presentation may include fever, chest pain, flu-like viral syndrome, and change in exercise tolerance.
- ECG findings suggestive of myocarditis (see definitions below): T-wave inversions, ST-segment depression, pathologic Q waves, complete LBBB, and premature ventricular contractions (PVCs)

## Specific ECG Findings Suggestive of Cardiomyopathy

### T-Wave Inversions

- >1 mm of depth in two or more contiguous leads V2–V6, II and aVF, or I and aVL
  - Excludes leads III, aVR, and V1
  - Excludes the repolarization variant in black athletes confined to V1–V4
  - Excludes juvenile T-wave inversion in leads V1–V3 when age is <16 years
- Disease-specific findings
  - HCM:
    - Commonly seen in lateral (V5–V6, I, and aVL) and inferior (II and aVF) leads
    - Seen in approximately 52%–64% of individuals with HCM
  - ARVC:
    - Commonly seen in the anterior precordial leads (V1–V4); usually a flat or depressed ST segment prior to T-wave inversion, which distinguishes it from the repolarization variant in African-American athletes with J-point elevation and ST segment elevation prior to T-wave inversion
    - Seen in approximately 85% of individuals with ARVC
    - Can also be seen in lateral (V5–6) and inferior (II and aVF) leads

- DCM
  - T-wave inversion seen in 25%–45% of individuals
- LVNC
  - T-wave inversion seen in approximately 41% of individuals
- Recommended follow-up evaluation:
  - In lateral or inferolateral leads: echocardiogram and cardiac MRI to exclude HCM or apical HCM; exercise ECG test and 24-hour ECG monitor in cases of grey-zone hypertrophy (13–15 mm)
    - These patients require annual re-evaluation as two separate studies have found that 6% of patients with initial normal cardiac imaging subsequently develop clinical cardiomyopathy; thus, the ECG manifestations of cardiomyopathy can occur before the morphologic changes in certain patients
  - Inferior leads alone: echocardiogram
  - Anterior leads: echocardiogram, cardiac MRI, exercise ECG test, 24-hour ECG monitor, and signal-averaged ECG to exclude ARVC

### ST-Segment Depression

- ≥0.5 mm in depth in two or more contiguous leads
  - This finding is rare in healthy athletes; thus, its presence requires the exclusion of cardiomyopathy.
- Disease-specific findings
  - HCM:
    - Reported in 46%–50% of individuals
  - ARVC:
    - Can be seen but not a part of diagnostic criteria for ARVC
  - LVNC
    - Seen in approximately 51% of individuals
- Recommended follow-up:
  - Echocardiogram

### Pathologic Q Waves

- Q/R ratio ≥0.25 or ≥40 ms in duration in two or more contiguous leads (except III and aVR)
  - Q waves are commonly seen in young athletes, specifically long "skinny" Q waves related to overall increases in QRS amplitude.
  - The new definition of pathologic Q waves is based on international consensus recommendations.
- Disease-specific findings
  - HCM:
    - Reported in 32%–42% of individuals
  - ARVC:
    - Q waves are not a common finding in ARVC
  - DCM
    - Present in 10%–25% of individuals
  - Coronary artery disease (CAD)
    - Could represent premature atherosclerotic CAD; a CAD risk factor assessment is indicated
- Recommended follow-up:
  - Echocardiogram and CAD risk factor assessment in older athletes

### Complete Left Bundle Branch Block (LBBB)

- QRS ≥120 ms, predominantly negative QRS complex in lead V1 (QS or rS), and upright notched or slurred R wave in leads I and V6
- Disease-specific findings
  - HCM:
    - Present in approximately 2% of individuals
  - ARVC:
    - LBBB is not a common finding in ARVC
  - DCM:
    - Present in 9%–25% of individuals
- Recommended follow-up:
  - Echocardiogram, cardiac MRI with a perfusion study; a comprehensive evaluation to exclude cardiac pathology must be performed in the presence of LBBB.

## CHANGES SUGGESTIVE OF PRIMARY ELECTRICAL DISEASE

### Introduction

- Inherited arrhythmia syndromes and an important cause of SCD in young individuals
- Ventricular pre-excitation is also an electrical disorder at risk for SCD.
- Individuals may be asymptomatic or present with syncope, palpitations, or sudden cardiac arrest
- These primary electrical diseases usually exhibit detectable ECG abnormalities

### Congenital Long QT Syndrome (LQTS)

- A genetic cardiac arrhythmia syndrome characterized by a prolonged QT segment, indicating a delay in ventricular repolarization
- Predisposes individuals to syncope, seizures, and sudden arrhythmic cardiac death (due to torsades de pointes)
- The QT interval is affected by the heart rate; thus, measured QT intervals are corrected to a heart rate of 60 bpm (QTc) using validated formulas (most commonly Bazett's)
  - Bazett's heart rate correction formula is $QTc = QT/\sqrt{RR}$ (the RR interval is measured in seconds)
- Significant overlap in QTc duration exists between individuals with and without LQTS
  - Thus, a single prolonged QTc on an ECG does not equate with a diagnosis of LQTS; subsequent evaluation is needed (see below).
- ECG cut-off values suggestive of LQTS: >470 ms in males; >480 ms in females
  - A QTc of >500 ms is markedly prolonged, carries a greater risk of ventricular arrhythmias, and should trigger more aggressive management.
  - Care should be used when measuring the QT segment
    - Lead II or V5 usually provide the best demarcation of the end of the T wave and thus allow most accurate measurement of QT interval
    - U waves should not be included in QT measurement
    - A straight line drawn on the downslope of the T wave to the isoelectric line should mark the end of the T wave for measurement purposes.

### Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)

- An inheritable ion channelopathy that predisposes to sudden death via ventricular arrhythmias triggered by exercise or emotional stress.
- Resting ECG is typically normal in CPVT.
- Graded exercise stress test is the routine method to diagnose CPVT and should be considered in children and adolescents who develop syncope during exercise or from emotional stress.
  - Polymorphic ventricular ectopy increasing in frequency with higher intensity on stress testing and leading to polymorphic ventricular tachycardia is diagnostic.

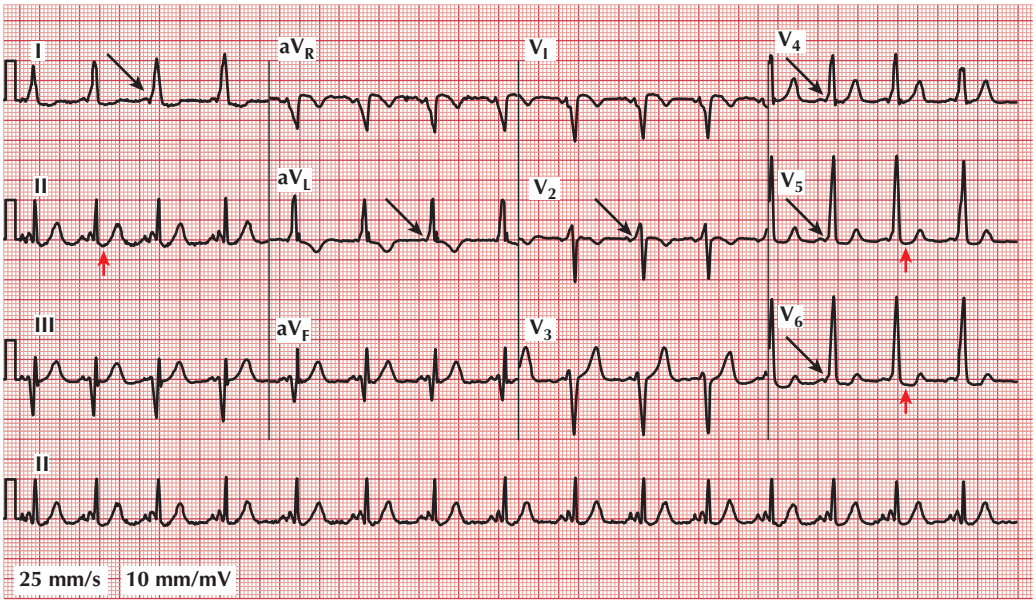


**Figure 34.4.** ECG demonstrating the classic findings of Wolff–Parkinson–White patterns with a short PR interval (<120 ms), delta wave with slurred QRS upstroke *(black arrows)*, and prolonged QRS (>120 ms). ST-segment depression, another common finding in WPW pattern, is also present *(red arrows)*.

## Brugada Syndrome (BrS)

- A genetic cardiac arrhythmia syndrome that predisposes to SCD via ventricular fibrillation
- Ventricular fibrillation due to BrS most commonly occurs during rest or sleep
- ECG findings suggestive of Type 1 BrS: high take-off ST elevation (≥2 mm) with a down-sloping ST segment elevation, followed by a negative symmetric T wave in ≥1 leads in V1–V3

## Ventricular Pre-Excitation/ Wolff–Parkinson–White Pattern

- Cardiac conduction abnormality wherein an accessory pathway bypasses the AV node leading to abnormal electrical conduction to the ventricle.
- A rapid conducting pathway in the presence of atrial fibrillation or flutter can lead to ventricular fibrillation.
- ECG findings suggestive of ventricular pre-excitation: short PR interval (<120 ms), slurring of the QRS upslope (delta wave), and widening of the QRS complex (>120 ms); ST-segment depression is also frequently present (see Fig. 34.4).
- Evaluation of WPW pattern begins with an exercise stress test; abrupt cessation of pre-excitation at higher heart rates suggests a low risk pathway. If pre-excitation does not resolve on a stress test, an electrophysiologic study should be considered to assess the characteristics of the bypass pathway, with possible ablation of high-risk tracts.

## Specific ECG Findings Suggestive of Primary Electrical Disease

### Long QTc interval

- >470 ms in males and >480 ms in females
- >500 ms indicates a markedly prolonged QTc
- Recommended follow-up:
  - Repeat ECG testing on a separate day, review of symptoms and family history, medication review, exclusion of laboratory abnormalities, first-degree relative screening ECGs, referral to a heart rhythm specialist, exercise stress test, and genetic testing

### Ventricular Pre-Excitation/WPW Pattern

- PR interval <120 ms with a delta wave (slurred upstroke in the QRS complex) and a wide QRS (≥120 ms)
- Recommended follow-up:
  - Echocardiogram and exercise ECG stress test

### Brugada Type 1 Pattern

- Initial ST elevation ≥2 mm (high take-off) with down-sloping ST segment elevation, followed by a negative symmetric T wave in ≥1 leads in V1–V3
- Recommended follow-up:
  - Referral to a heart rhythm specialist

## Other ECG Abnormalities

### Profound Nonspecific Intraventricular Conduction Delay (IVCD)

- QRS >140 ms
  - Regardless of QRS morphology
- Disease-specific findings:
  - Concerning for myocardial disease
- Recommended follow-up:
  - Echocardiogram

### Profound Sinus Bradycardia

- Resting heart rate <30 bpm
- Disease-specific findings:
  - Concerning for primary SA node disease if heart rate does not appropriately respond to exercise
- Recommended follow-up:
  - Repeat ECG after mild aerobic activity

### Profound First-Degree Heart Block

- PR interval >400 ms
- Disease-specific findings:
  - If PR does not shorten with exercise, there is a concern for primarily electrical disease or cardiomyopathy.

- Recommended follow-up:
  - Repeat ECG after mild aerobic activity and exercise ECG test

### High-Grade AV block

- Mobitz type II second-degree AV block: intermittently nonconducted P wave with a fixed PR interval
- Third-degree AV block (complete heart block): more P waves than QRS complexes and no association between the two; QRS complexes are regular
- Disease-specific findings:
  - Concern for primarily electrical disease or cardiomyopathy
- Recommended follow-up:
  - Cardiology consultation, echocardiogram, 24-hour ECG monitor, and exercise ECG test

### Multiple Premature Ventricular Contractions (PVCs)

- >2 PVCs per 10 second of ECG tracing
  - Indicates the potential for a high 24-hour PVC burden
- Disease-specific findings
  - In athletes with >2000 PVCs in 24 hours, 30% have structural heart disease
  - In high-dynamic athletes (e.g., cyclist, triathletes, and rowers), pathologic remodeling of the right ventricle may occur with high training volumes over a long period of time, leading to exercise-induced ARVC. Consideration should be given to further evaluation of individuals with an ECG that contains even one PVC
    - Specifically, a PVC with an LBBB morphology and superior axis (negative in inferior leads) is more concerning for a RV origin
- Recommended follow-up:
  - Echocardiogram, 24-hour ECG monitor, and exercise ECG test

### Atrial Tachyarrhythmias

- Heterogeneous group of tachycardic conditions wherein the electric impulse originates from the SA node, atrium, or AV node
- Heart rate >100 bpm
- Includes sinus tachycardia, supraventricular tachycardia (SVT), atrial fibrillation, and atrial flutter
- Disease-specific findings:
  - Can be associated with myocarditis, congenital heart disease, and any cardiomyopathy
- Recommended follow-up:
  - Echocardiogram, 24-hour ECG monitor, exercise ECG test, and cardiology consultation

### Ventricular Arrhythmias

- Couplets, triplets, and nonsustained ventricular tachycardia
- Do not independently predispose to SCD
- Disease-specific findings:
  - Can be associated with myocarditis, HCM, ARVC, DCM, CPVT, and myocardial infarction
- Recommended follow-up:
  - Echocardiogram, 24-hour ECG monitor, exercise ECG stress test, cardiac MRI, and cardiology consultation

## BORDERLINE ECG FINDINGS IN ATHLETES

### Introduction

- Emerging data suggest that several ECG findings previously thought to be abnormal may represent physiologic changes in athletes and are not predictive of diseases predisposing to SCD.
- These changes include complete RBBB, left and right axis deviation, and left and right atrial enlargement.
  - When these findings are found in isolation, with no other ECG abnormalities or clinical markers of concern, no further evaluation is warranted.
  - However, when these findings are found in combination (≥2 borderline findings) or with other compelling personal or family history, further evaluation is warranted.
- The recommended follow-up for two or more borderline findings is an echocardiogram.

### Specific Borderline ECG Findings

- Complete RBBB
  - rSR′ pattern in lead V1 and S wave wider than R wave in lead V6 with a QRS duration of ≥120 ms
- Left axis deviation
  - −30° to −90°
- Left atrial enlargement
  - Prolonged P-wave duration of >120 ms in leads I or II with the negative portion of the P wave ≥1 mm in depth and ≥40 ms in duration in lead V1
- Right axis deviation
  - >120°
- Right atrial enlargement
  - P-wave amplitude ≥2.5 mm in leads II, III, or aVF

## ADDITIONAL CONSIDERATIONS

### Athletes >30 Years of Age

- The number of masters athletes is increasing.
- The most common cause of SCD in this age group is atherosclerotic CAD.
- Although the benefits of exercise in this age group are unequivocal, exercise can be a trigger for acute coronary artery events
- Most "silent" CAD do not display ECG changes; however, some findings may be present on a resting ECG that could indicate changes from prior myocardial ischemia.
- These potential findings include:
  - Pathologic Q waves, ST-segment depression, T-wave inversion, left or RBBB, abnormal R-wave progression, left anterior hemiblock, and atrial fibrillation
    - In addition to excluding the presence of cardiomyopathy for these findings, in athletes aged >30 years, stress test should be considered to evaluate for occult CAD.

### Abnormal ECGs Requiring Serial Follow-Up

- Some ECG abnormalities may precede the phenotypic manifestation of structural heart disease such as HCM, ARVC, and DCM.
  - The most concerning is lateral or inferolateral T-wave inversion and ST-segment depression, wherein 6% of athletes with initially normal cardiac imaging have been found to develop overt cardiomyopathy on subsequent follow-up evaluations.
- Serial ECGs and repeat structural imaging should be performed in patients with ECG abnormalities suggestive of cardiomyopathy.
- Specific ECG findings requiring serial follow-up:
  - Abnormal T-wave inversion, ST-segment depression, pathologic Q waves, LBBB, and profound IVCD
    - Ideally, annual follow-up is warranted.

## SUMMARY

- SCD is the most common cause of death in young athletes during sports

- ECG changes are present in approximately 60% of disorders that predispose athletes to SCD.
- It is critical to accurately differentiate pathologic ECG changes from those caused by physiologic adaptations from regular training.
- Once an ECG abnormality is identified, appropriate further evaluation is important for clarifying an athlete's cardiac status, health, and safety.
- Collaboration with a cardiologist knowledgeable about the physiologic cardiac adaptations to exercise and the diseases associated with SCD in athletes is a critical component in this process.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


1. Corrado D, Biffi A, Basso C, et al. 12-lead ECG in the athlete: physiological versus pathological abnormalities. *Br J Sports Med*. 2009; 43(9):669-676.
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# CARDIAC DISEASE IN ATHLETES

# 35

Kimberly G. Harmon

## THE ATHLETE'S HEART

### Definition

Intense regular physical exercise can induce physiologic and morphologic cardiac changes known as "athlete's heart". These adaptations are considered a normal response to repetitive exercise training.

### Physiologic Changes

- Increased vagal tone
- Morphologic changes, including left ventricular (LV) enlargement, increases in LV wall thickness, and increases in LV mass

### Pathologic vs. Physiologic Hypertrophy

- The physiologic changes that occur in response to training can be difficult to differentiate from the pathologic processes that occur in hypertrophic cardiomyopathy (HCM) (Table 35.1).
- Magnetic resonance imaging (MRI) can detect atypical patterns of hypertrophy and late gadolinium enhancement, which may be suggestive of HCM.
- If the distinction between pathologic and physiologic hypertrophy cannot be established, a period of deconditioning should be considered.

### Participation Recommendations

- Athlete's heart describes normal physiologic adaptations to regular intense exercise, and thus, no treatment and no limits on sports participation are required.

## SUDDEN CARDIAC DEATH

### Epidemiology

- Sudden cardiac death (SCD) is the leading medical cause of death in young athletes.
- The actual incidence of SCD in athletes is difficult to estimate because of the lack of a mandatory national reporting system.
- New research suggest the incidence of SCD is around 1 in 50,000 athlete-years (AY) in college athletes and 1 in 80,000 AY in high school athletes with some higher-risk populations (Table 35.2).
- Males and African Americans are at a higher risk, with men's basketball appearing to be at a disproportionately higher risk: 1 in 9,000 AY.

### Presentation

- The prevalence of cardiovascular disorders in young people that can potentially lead to SCD is approximately 1 in 300.
- Most individuals with cardiovascular disorders will not go on to experience sudden cardiac arrest (SCA) or SCD; however, athletes may be at a higher risk because of their increased level of physical activity, which can be a trigger for arrhythmias.
- SCD is the presenting symptom of underlying cardiovascular pathology in 50%–90% of athletes limiting the usefulness of a history-based screen.
- Warning symptoms of underlying cardiovascular disease include a history of exertional chest pain, exertional syncope or presyncope, dyspnea or fatigue disproportionate to the degree of exertion, and palpitations or irregular heartbeats. Athletes with any of these symptoms require a careful workup before returning to exercise.
- A family history of sudden unexplained death or SCD before the age of 50 years or a history of familial cardiac disorders known to cause SCD in young athletes also warrant further diagnostic investigation before participation.

### Etiology of SCD in Athletes

- Older studies in US athletes suggest HCM as the leading cause of SCD in athletes; however, more recent studies and studies in the US military suggest that a pathologically normal heart is the most common finding at autopsy.
- Studies in other countries and a recent meta-analysis also suggest HCM may be less common as a cause of SCD than previously thought (Table 35.3).
- Specialized autopsy by a cardiovascular pathologist and molecular autopsy should help clarify the etiologies in the future.

## CAUSES OF SUDDEN CARDIAC DEATH

### Structural

#### Hypertrophic Cardiomyopathy

**Epidemiology:** HCM has a reported prevalence of 1 per 500 in the general US population; however, in larger-scale screening studies of athletes, the HCM prevalence ranges between 1 in 1,000 and 1 in 5,000. It is reported to cause 2%–36% of SCD in athletes (Table 35.3).

**Pathologic features:** Characteristic morphologic features of HCM include asymmetric LV hypertrophy (usually involving the ventricular septum), LV wall thickness of ≥16 mm (normal, ≤12 mm; borderline, 13–15 mm), a ratio between the septum and free wall of ≥1.3, and a nondilated LV. Histologic analysis shows disorganized cellular architecture. Intramural tunneling (myocardial bridging), wherein a segment of coronary artery is completely surrounded by the myocardium, is present in approximately one third of cases (Fig. 35.1).

**Development:** Morphologic features may appear in childhood but usually develop in early adolescence or young adulthood.

**Symptoms:** The presenting symptom of HCM is SCA in 80% of cases. Symptoms may include exertional chest pain, dyspnea, lightheadedness, or syncope.

**Physical examination findings:** HCM should be suspected in any athlete in whom a harsh systolic ejection murmur is heard on auscultation that increases with Valsalva and diminishes with maneuvers that increase venous return. A fourth heart sound may also be present.

**Diagnostic tests:** ECG is abnormal in up to 95% of athletes with HCM. Echocardiography remains the standard to confirm the diagnosis of HCM by identifying pathologic LV wall thickness (≥16 mm) and LV diastolic dysfunction. MRI has additional value in identifying segmental hypertrophy in the anterolateral LV free wall or at the apex and identifying late gadolinium enhancement.

**Return to play:** Exercise increases the risk of ventricular tachycardia/fibrillation and is a modifiable risk factor. Estimation of risk levels based on phenotype is not reliable because

of the morphologic diversity of HCM and the unpredictable instability of the myocardial substrate. Exercise is a modifiable risk factor; therefore, it is recommended that athletes diagnosed with HCM should not participate in strenuous physical activity.

**TABLE 35.1 DISTINGUISHING HCM FROM ATHLETE'S HEART**

| HCM | Athlete's Heart |
|---|---|
| Unusual pattern of LVH, may be heterogeneous | Symmetric LVH or uniform distribution of hypertrophy |
| Wall thickness > 16 mm | Wall thickness < 12 mm |
| LV cavity < 45 mm (small) | LV cavity > 55 mm (not small) |
| Left atrial enlargement | No left atrial enlargement |
| Abnormal LV filling | Normal LV filling |
| EKG abnormalities (see Chapter 30) | EKG with high voltage, but no Q wave changes (see Chapter 30) |
| Thickness does not decrease with deconditioning | LVH decreases with deconditioning |
| Family history of HCM | No family history of HCM |
| Positive genetic testing for HCM | Negative genetic testing for HCM |

### Congenital Coronary Anomalies

**Epidemiology:** Congenital coronary anomalies (CCAs) account for 6%–17% of cases of SCD in athletes (Table 35.3).

**Pathologic features:** The most common CCA is an abnormal origin of the left coronary artery arising from the right sinus of Valsalva. Other features that may contribute to ischemia during exercise include an acute angled take-off, hypoplastic ostium, or impingement of the anomalous artery as it traverses between the expanding great vessels during exercise (Fig. 35.2).

**Symptoms:** Chest pain, exertional syncope, and SCA

**Physical examination findings:** None

**Diagnostic tests:** ECG does not typically identify CCAs. Echocardiogram can identify CCAs of coronary artery origin in 80%–97% of patients. Advanced cardiac imaging such as computed tomography (CT) angiography, cardiac MRI, or coronary angiography may be necessary.

**Return to play:** Detection should result in exclusion from all competitive sports. However, participation may be considered 3 months after correction of the defect if patient remains symptom free and has a normal maximal exercise test.

### Myocarditis

**Epidemiology:** Acute inflammatory process involving the myocardium; Coxsackie B virus is implicated in more than 50% of cases, but echovirus, adenovirus, influenza, and *Chlamydia pneumoniae* have also been associated with myocarditis. It has been reported as the cause of death in 3%–13% in studies on SCD in athletes (Table 35.3).

**Pathologic features:** Lymphocytic infiltrate of myocardium with necrosis or degeneration of adjacent myocytes. Acute phase

**TABLE 35.2 INCIDENCE OF SUDDEN CARDIAC DEATH AND ARREST IN ATHLETES**

| Author | Year | Country | Method | Population | Incidence* | Number of Years | Age Range (Years) |
|---|---|---|---|---|---|---|---|
| Boden | 2013 | US | Retrospective cohort | College/high school football | 1:112,359 college<br>1: 312,500 high school | | |
| Van Camp | 1996 | US | Retrospective cohort | College/high school athletes | 1:300,000 | 10 | 17–24 |
| Maron | 2009 | US | Retrospective cohort | Athletes | 1:163,934 | 27 | 8–39 |
| Steinville | 2011 | Israel | Retrospective cohort | Athletes | 1st – 1:39,370<br>2nd – 1:37,593 | 24 | 12–44 |
| Corrado | 2003 | Italy | Prospective cohort study | Athletes/young people | 1:47,600 athlete<br>1:142,900 young people | 20 | 12–35 |
| Holst | 2010 | Denmark | Retrospective cohort | Athletes/young people | 1:82,645 athlete<br>1:26,595 general pop | 7 | 12–35 |
| Drezner | 2005 | US | Retrospective cohort | College athletes | 1:67,000 | 3.3 | |
| Harmon | 2011 | US | Retrospective cohort | College athletes | 1:43,000 | 5 | 18–26 |
| Harmon | 2015 | US | Retrospective cohort | College athletes | 1:53,000 | 10 | 17–26 |
| Maron | 2014 | US | Retrospective cohort | College athletes | 1:83,000 - confirmed<br>1:62,000 - presumed | 10 | 17–26 |
| Drezner | 2009 | US | Cross-sectional survey | High school athletes | 1:23,000 SCA + SCD<br>1:46,000 SCD | | |
| Toresdahl | 2014 | US | Prospective observational | High school athletes | 1:87,719 SCA + SCD | 3 | 14–18 |
| Maron | 2012 | US | Retrospective cohort | High school athletes | 1:150,000 | 26 | 12–18 |
| Roberts | 2013 | US | Retrospective cohort | High school athletes | 1:416,666 last decade<br>1:917,000 | | |
| Maron | 1998 | US | Retrospective cohort | High school athletes | 1:217,000 overall | 11 | |
| Marjion | 2011 | France | Prospective | Competitive athletes | 1:102,00 | | 10–35 |

*Incidence numbers are in athlete-years.

**TABLE 35.3 STUDIES OF THE ETIOLOGIES OF SUDDEN CARDIAC DEATH IN YOUNG PEOPLE**

| Author | Year | Country | Exertional Death vs. All Deaths | Population | Age Range (Years) | Number of Deaths | HCM | Idiopathic LVH/ Possible HCM | Coronary Artery Abnormalities | ARVC | DCM | AN-SUD | CAD | Myocarditis Related | Aortic Dissection | Other |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Corrado | 2003 | Italy | all | Competitive athletes | 12–35 | 55 | 2% | 0% | 13% | 22% | 0% | 7% | 20% | 13% | 2% | 22% |
| De Noronha | 2009 | UK | all | Athletes | 1–35 | 89 | 12% | 25% | 8% | 10% | 0% | 19% | 8% | 3% | 0% | 4% |
| Maron | 2009 | US | all | Athletes | 8–39 | 690 | 36% | 8% | 17% | 4% | 2% | – | 3% | 6% | 3% | 20% |
| Holst | 2010 | Denmark | exertional | Competitive athletes | 12–35 | 15 | 0% | 7% | 7% | 27% | 0% | 27% | 13% | 7% | 0% | 13% |
| Suarez-Mier | 2011 | Spain | exertional | Recreational athletes | 9–35 | 81 | 10% | 9% | 6% | 15% | % | 23% | 14% | 5% | 0% | 19% |
| Harmon | 2014 | US | all | Competitive athletes | 18–26 | 36 | 3% | 8% | 14% | 3% | 8% | 32% | 5% | 8% | 8% | 5% |

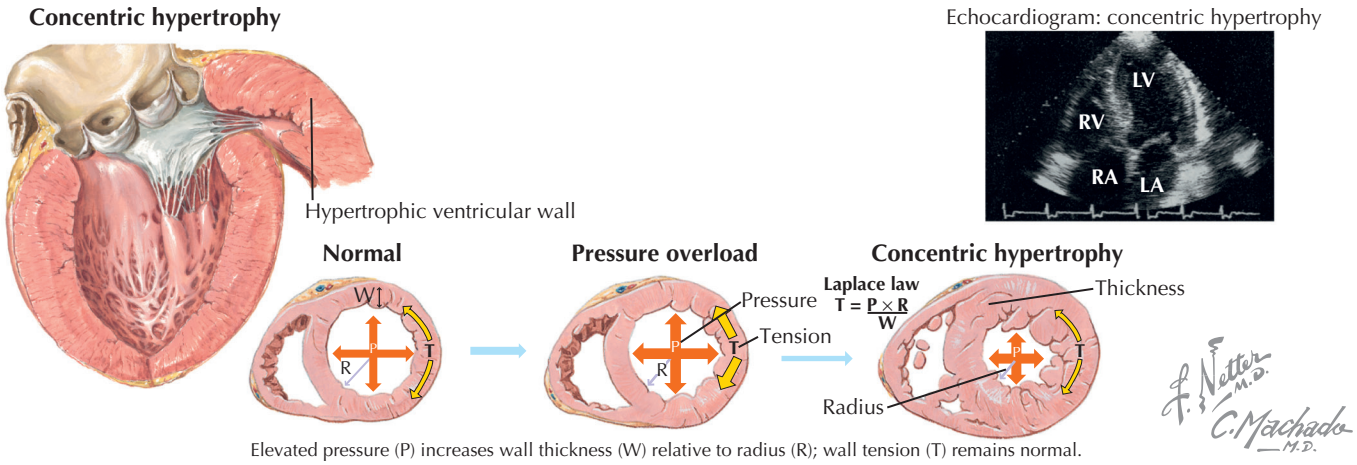


**Figure 35.1.** Left ventricular hypertrophy.

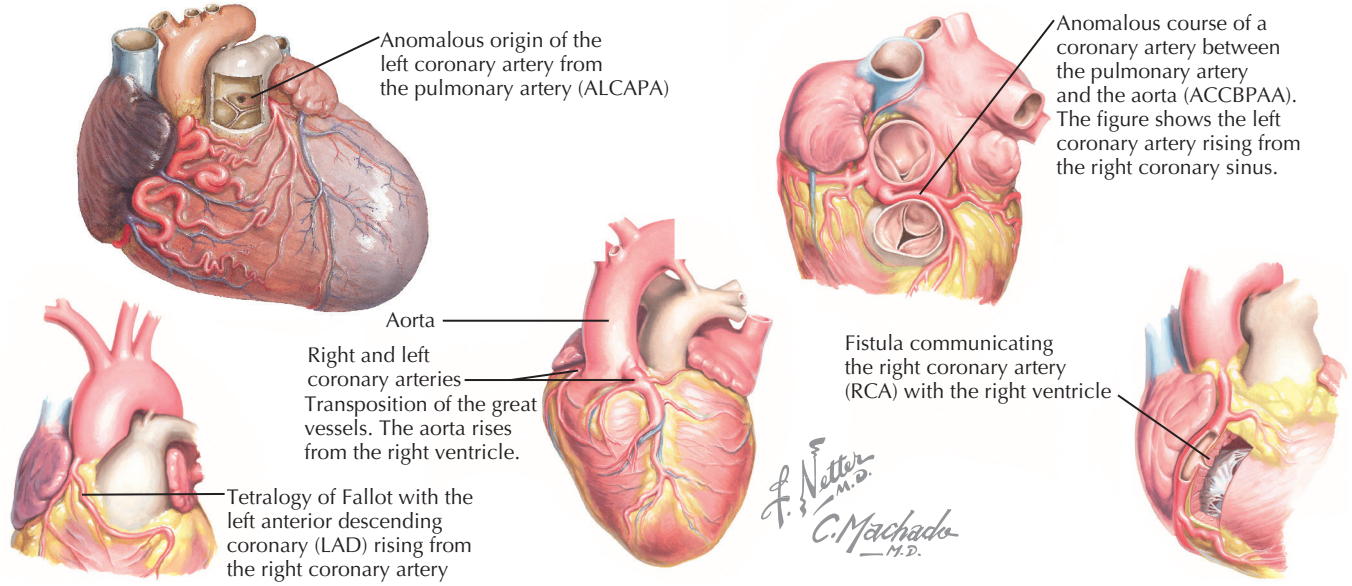


**Figure 35.2.** Congenital coronary artery anomalies.

presents with flu-like illness, which may lead to dilated cardiomyopathy. SCD may occur in the presence of either active or healed myocarditis (Fig. 35.3).

**Symptoms:** Characteristic symptoms include a prodromal viral illness, followed by progressive exercise intolerance and congestive symptoms of dyspnea, cough, and orthopnea.

**Physical examination findings:** S3 gallop and signs of heart failure (e.g., edema and pulmonary rales)

**Diagnostic tests:** ECG may show diffuse low voltage, ST- and T-wave changes, heart block, or ventricular arrhythmias (see Chapter 30). Laboratory tests include leukocytosis, eosinophilia, elevated sedimentation rate or C-reactive protein, and increased myocardial enzymes. Echocardiography may show dilated LV, global hypokinesis, segmental wall abnormalities, and decreased LV ejection fraction. Cardiac MRI can show a regional increase in water content on T2-weighted images and epicardial or midmyocardial late gadolinium enhancement.

**Return to play:** Athletes with definitive or probable myocarditis should not compete. Resolution is variable, but return to sport should not be considered before 3–6 months after the onset of the illness. The athlete may return to competitive sports if ventricular systolic function has returned to normal, serum markers of myocardial injury have normalized, and clinically relevant arrhythmias are absent.

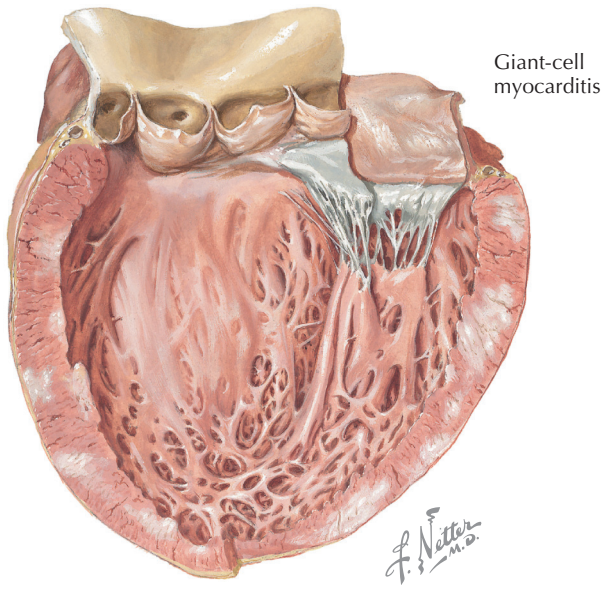


**Figure 35.3.** Idiopathic myocarditis.

### Arrhythmogenic Right Ventricular Cardiomyopathy

**Epidemiology:** Estimated prevalence is 1 case per 5000 population; based on previous studies, arrhythmogenic right ventricular cardiomyopathy (ARVC) accounts for 3%–27% of SCD in athletes (Table 35.3).
**Pathologic features:** Characterized by a progressive fibrofatty replacement of the right ventricular myocardium causing wall thinning and right ventricular dilatation
**Symptoms:** ARVC may present with syncope, chest pain, palpitations, or SCA.
**Physical examination findings:** Normal
**Diagnostic tests:** ARVC can be diagnosed using ECG although echocardiogram, cardiac MRI, or CT may demonstrate right ventricular dilatation and wall thinning, reduced right ventricular ejection fraction, or right ventricular aneurysms
**Return to play:** No competitive athletics because physical activity contributes to disease progression

### Dilated Cardiomyopathy

**Epidemiology:** Estimated prevalence is 1 case per 2500 population. Dilated cardiomyopathy (DCM) is a less common cause of SCD in athletes, attributed to 0–8% of cases (Table 35.3).
**Pathologic features:** Characterized by LV dilatation and systolic dysfunction with normal LV wall thickness; secondary DCM results from prolonged untreated systemic hypertension, ischemic heart disease, viral myocarditis, infiltrative diseases (e.g., sarcoidosis, amyloidosis, hemochromatosis), autoimmune illnesses, or toxins (e.g., ethanol).
**Symptoms:** Progressive exertional intolerance, dyspnea, orthopnea, fatigue, and edema
**Physical examination findings:** S3 or S4 may occur in DCM; holosystolic murmur suggestive of mitral regurgitation
**Diagnostic tests:** ECG and Holter monitoring may show supraventricular and ventricular tachyarrhythmias as well as major conduction delays (e.g., bundle branch block and atrioventricular block). Echocardiogram is diagnostic, demonstrating a dilated LV with reduced ejection fraction.
**Return to play:** It is recommended that athletes with DCM be excluded from all competitive sports, with the possible exception of low-intensity sports.

### Aortic Rupture/Marfan Syndrome

**Epidemiology:** Reported incidence of Marfan syndrome is 1 in 5,000–10,000 individuals.
**Pathologic features:** Progressive dilatation and weakness (cystic medial necrosis) of proximal aorta and myxomatous degeneration of mitral and aortic valves leading to valvular dysfunction
**Symptoms:** Symptoms related to aortic root dissection include chest and thoracic pain. Heart failure may also occur secondary to aortic valve incompetence.
**Physical examination findings:** Highly variable clinical features usually manifested in adolescence and young adulthood; diagnosis of Marfan syndrome is based on the Ghent nosology. These criteria rely on the recognition of both "major" and "minor" clinical manifestations involving the skeletal, cardiovascular, and ocular systems as well as the dura (Table 35.4).
**Diagnostic tests:** Echocardiogram is diagnostic for aortic root dilatation and aortic regurgitation. Slit-light ophthalmologic examination for ectopia lentis and lumbosacral MRI or CT for dural ectasia can also be diagnostic.
**Return to play:** Return to play (RTP) is based on aortic root dimension and pathologic features of the disease. RTP recommendations should be guided by risk assessment performed by a specialist familiar with aortic root pathology.

### Coronary Artery Disease

**Epidemiology:** Atherosclerotic coronary artery disease is the most frequent cause of SCD in athletes over the age of 30 years. The incidence of exercise-related sudden death in adults is 1 in 15,000–18,000.
**Pathologic features:** Most often caused by atherosclerotic plaque druption; exercise may be a stimulus for plaque disruption. Development is progressive and related to coronary risk factors (hypertension, diabetes, dyslipidemia, tobacco use, illicit drug use, and a family history of premature atherosclerotic disease).
**Symptoms:** Exertional chest pain, angina, lightheadedness, palpitations, or SCD
**Physical examination findings:** No specific cardiac findings
**Diagnostic tests:** ECG may show evidence of prior ischemia. Exercise stress testing may show ST-segment depression or U-wave inversion. Stress echo may show wall motion abnormality, and cardiac CT, MRI, or angiography will show extent of coronary artery narrowing.
**Return to play:** Dependent on extent of disease, symptoms, and stability after medical management, percutaneous coronary interventions, or surgery; RTP recommendations often guided by risk assessment and management by a specialist.

**TABLE 35.4 MAJOR AND MINOR CRITERIA FOR THE DIAGNOSIS OF MARFAN SYNDROME**

| Major | Minor |
|---|---|
| **Skeletal** | |
| **Need 4** | |
| Reduced upper to lower segment ratio (0.85) | Pectus excavatum (moderate) |
| Arm span exceeding height | Joint hypermobility |
| Arachnodactyly of fingers and toes | High-arched palate |
| Scoliosis > 20 degrees or spondylolisthesis | |
| Pectus carinatum | |
| Pectus excavatum | |
| Reduced extension of elbows | |
| Pes planus | |
| **Cardiovascular** | |
| **Need 1** | |
| Dilatation of the ascending aorta involving the sinuses of Valsalva, with or without aortic regurgitation | Mitral valve prolapse<br>Mitral regurgitation<br>Dilatation of the pulmonary artery |
| Dissection of the ascending aorta | Calcification of the mitral annulus |
| **Ocular** | |
| Ectopic lens | Flat cornea<br>Increased axial globe length<br>Hypoplastic iris<br>Myopia<br>Retinal detachment |
| **Other Findings** | |
| Dural ectasia affecting the lumbosacral spinal canal | Spontaneous pneumothorax<br>Apical blebs<br>Stretch marks |

## Primary Electrical Disease

**Channelopathies:** Diseases predisposing to potentially lethal ventricular tachyarrhythmias that are characterized by mutations in ion-channel proteins leading to dysfunctional sodium, potassium, calcium, and other ion transport across cell membranes.

It is likely that many deaths with a pathologically normal heart are due to channelopathies.

### Long QT Syndrome

**Epidemiology:** Long QT syndrome (LQTS) is the most common ion channelopathy.
**Pathologic features:** Characterized by prolongation of ventricular repolarization and QT interval corrected for heart rate ($QT_c$); there are six defined types, with three of them (LQTS-1, LQTS-2, and LQTS-3) occurring more frequently
**Symptoms:** Syncope or presyncope related to physical exertion or emotional stress; family history of sudden unexplained death, unexplained drowning or motor vehicle accident, or sudden infant death
**Diagnostic tests:** In an asymptomatic athlete without a family history of SCD, current cutoff values for a prolonged QTc is 470 ms in males and 480 ms in females.
**Return to play and treatment:** RTP decisions should be guided by a heart rhythm specialist or a genetic cardiologist experienced in the care of athletes. Competitive sports participation may be considered after implementation of treatment and appropriate precautionary measures with informed decision making.

### Short QT Syndrome

**Epidemiology:** Short QT syndrome (SQTS) has only recently been described (in 2000).
**Pathologic features:** Hyperfunctioning of potassium channel
**Symptoms:** Palpitations, syncope, atrial fibrillation, and SCA/SCD
**Diagnostic tests:** ECG changes include a $QT_c$ interval <340 ms and tall peaked T waves.
**Return to play and treatment:** Sports participation may be considered assuming appropriate precautionary measure and disease-specific treatments are in place and the athlete has been asymptomatic on treatment for at least 3 months.

### Brugada Syndrome

**Epidemiology:** Brugada syndrome was first described in 1992. It is a rare channelopathy that is more prevalent in males from Southeast Asia.
**Pathologic features:** Abnormalities of sodium channels
**Symptoms:** Syncope, SCA/SCD, and sudden death during sleep
**Diagnostic tests:** ECG shows a pattern of right bundle branch block and ST-segment elevation in leads $V_1$–$V_3$. ECG patterns may not be present unless "unmasked" with the administration of sodium-channel blockers (e.g., flecainide or procainamide) (see Chapter 30).
**Return to play and treatment:** Sports participation may be considered assuming appropriate precautionary measures and disease-specific treatments are in place and the athlete has been asymptomatic on treatment for at least 3 months.

### Catecholaminegic Polymorphic Ventricular Tachycardia

**Epidemiology:** Catecholaminergic polymorphic ventricular tachycardia (CPVT) is a familial disorder characterized by stress-induced ventricular arrhythmias that result in SCD in children and young adults.
**Pathologic features:** Abnormalities in calcium-channel function
**Symptoms:** Syncope, sudden death, and polymorphic ventricular tachycardia triggered by vigorous physical exertion or acute emotion
**Diagnostic tests:** Resting ECG is normal although certain patients will have prominent U waves. Exertion or epinephrine challenge can induce ventricular tachycardia.
**Return to play:** It is recommended that athletes with CPVT should not participate in competitive sports.

### Wolff–Parkinson–White Syndrome (WPW)

**Epidemiology:** Approximately 1% of ECGs will show changes consistent with Wolff–Parkinson–White (WPW) syndrome; however, only a small subset will go on to develop the associated arrhythmias that define the syndrome. Risk of SCD in WPW is estimated as 1 per 1000 patient years.
**Pathologic features:** A tachyarrhythmia caused by an accessory pathway (the Bundle of Kent) that directly connects the atria and ventricles and bypasses the AV node; the arrhythmia can be atrioventricular tachycardia (80%), atrial fibrillation (15%–20%), or atrial flutter (5%) (Fig. 35.4).
**Symptoms:** Palpitations, syncope, and near-syncope
**Diagnostic tests:** ECG shows characteristic changes (see Chapter 30); once diagnosed, risk stratification should be performed using exercise stress testing
**Return to play and treatment:** In athletes with low-risk pathways, RTP without treatment is reasonable. In athletes with high-risk pathways, catheter ablation with subsequent RTP should be considered.

## Traumatic

### Commotio Cordis

**Epidemiology:** Commotio cordis occurs in a **structurally normal heart** after the chest wall is struck with a blunt object. If the blow is sustained at a vulnerable phase of ventricular repolarization (just before peak of T wave), it can lead to ventricular fibrillation. More than 80% of cases involve sports with a firm projectile such as baseball, softball, hockey, and lacrosse. Attempts to prevent commotio cordis include the use of chest protectors and "safety balls." In animal model studies, chest protectors have not decreased the incidence of arrhythmia, whereas the use of "safety balls" have been associated with a lower incidence of arrhythmia. Other preventive measures include educating athletes, coaches, and caregivers regarding the presentation of commotio cordis, avoiding "shot blocking" in sports such as lacrosse and ice hockey, and an emergency action plan (EAP) that includes early access to defibrillation.
**Pathologic features:** Structurally normal heart
**Symptoms:** Instantaneous collapse following a blow to the chest. In approximately half of cases, a brief period (10 s) of consciousness occurs before the collapse.
**Treatment and return to play:** Treatment is expedient defibrillation. Survival is possible with early defibrillation, with survival rates of 25% if resuscitation is initiated within 3 minutes from collapse, and only a 3% survival if resuscitation is initiated later than 3 minutes after collapse.

## ATHLETES WITH SUSPECTED OR KNOWN CARDIOVASCULAR DISEASE

### Recognition and Evaluation of Worrisome Symptoms

- 20%–50% of athletes with SCD have had prior symptoms. Many had consulted providers and been reassured.
- Exertional syncope is *always* alarming and should be thoroughly investigated.
  - ECG will detect HCM 95% of times and also screens for LQTS, SQTS, Brugada syndrome, and pre-excitation syndromes. Other diseases that may cause changes in the ECG (myocarditis) can also be detected.
  - Echocardiogram is useful in evaluation of HCM, structural heart disease, and aortic root dilatation.
  - Exercise stress testing may uncover exertional arrhythmias, channelopathies, or ischemia secondary to coronary artery disease or anomalies.
  - Holter monitor or event monitor may also identify arrhythmias.

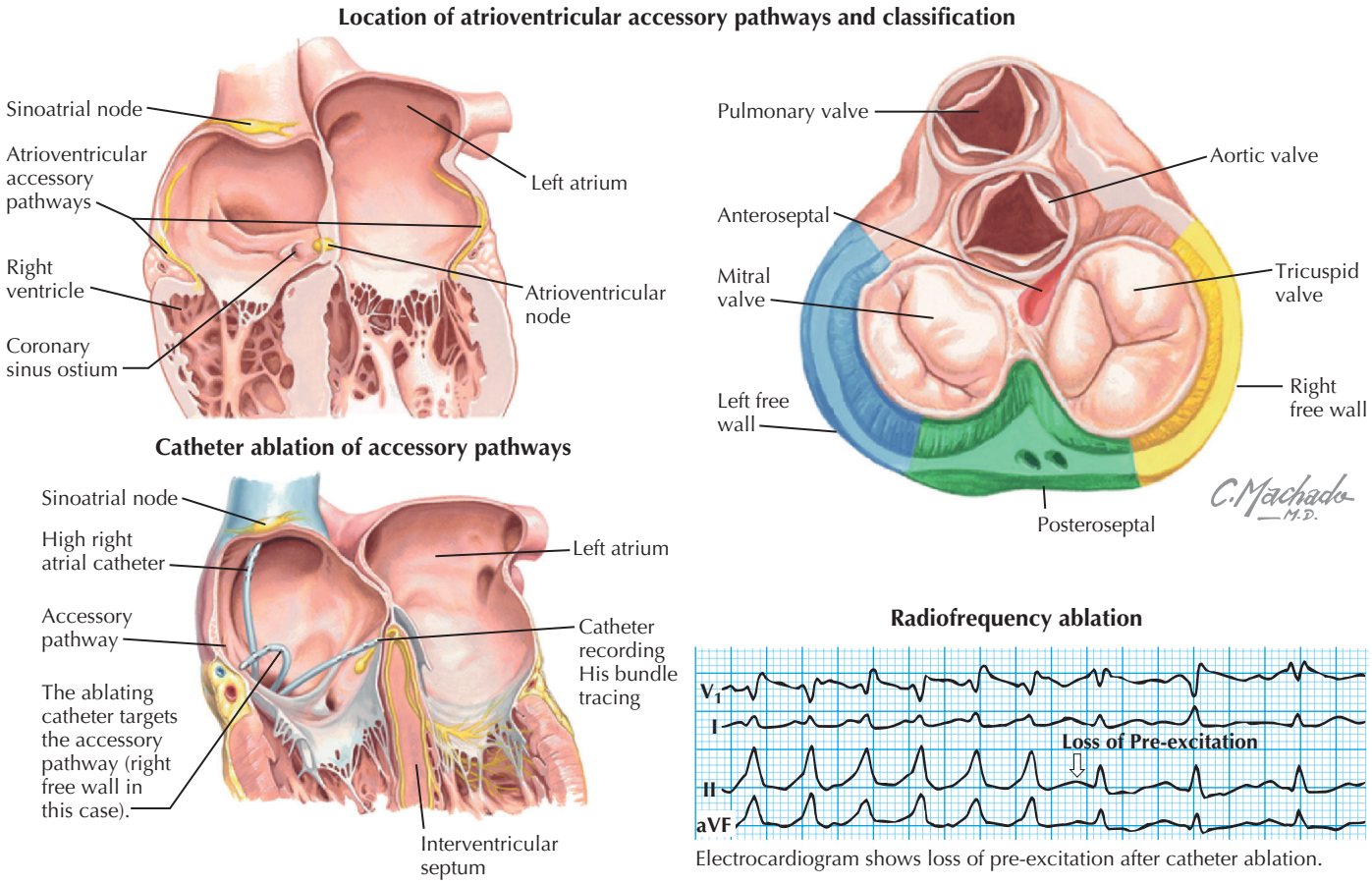


**Figure 35.4.** Accessory pathways and the Wolff–Parkinson–White syndrome.

- Cardiac MRI or CT may show structural abnormalities such as coronary anomalies, ARVC, or apical-variant HCM.
- Electrophysiology (EP) study may identify inducible arrhythmias or re-entrant pathways.
- Tilt table testing may provide supporting evidence that syncope occurred because of hemodynamic changes (neurocardiogenic syncope).

- Consultation with a specialist is recommended if an abnormality is suspected or identified.
- Athletes should be restricted from sports participation until the diagnostic workup is complete.

## Athletes With Cardiovascular Disease

- When athletes are identified with cardiac diseases that place them at a risk for SCA or disease progression, the question of advisable athletic activities is complex. Exercise is a known trigger for SCD because of increased catecholamine levels. Activity recommendations have evolved from strict disqualification decisions made by the treating physician to a model of shared decision making by considering a myriad of factors.
- Detailed eligibility and disqualification recommendations have been recently published in a Scientific Statement from the American Heart Association and American College of Cardiology.

## Legal Considerations

**Restriction of participation:** An institution has a right to restrict an individual from participation as long as that restriction is based on objective medical evidence and supported by the team physician and institutional medical consultants. Decision regarding medical eligibility to play may differ between institutions.

## EMERGENCY PREPAREDNESS AND MANAGEMENT OF SCA

### Emergency Action Plan (EAP)

- Every school or institution that sponsors athletic activities should have a written and structured EAP.
- The EAP should be developed in concert with local emergency medical services, school safety officials, likely first responders, and school administrators.
- The EAP should be specific to each individual athletic venue and provide plans for:
  - Communication: A communication system should be in place to activate the emergency medical services (EMS) system and to alert local/school responders and expedite transfer of emergency equipment (e.g., automated external defibrillator [AED]) to the scene.
  - Personnel: An identified team of targeted first responders (e.g., coaches and school health officials) should receive training in the recognition of SCA, cardiopulmonary resuscitation (CPR), and AED use.
  - Equipment: On-site AED programs are strongly encouraged and are likely the only means of achieving early defibrillation in the athletic setting.
  - Transportation: Transportation routes for arriving EMS should be defined as well as transport to an identified hospital with advanced cardiac life support services.

- The EAP should be reviewed and practiced at least annually by all potential responders to an SCA.
- The target time interval from collapse to first shock should be *less than 3 minutes*.

## Management of SCA

- Initial management of SCA should include early activation of EMS, early CPR, early defibrillation, and early transition to advance cardiac life support.
- Approximately 50% of athletes with SCA have brief myoclonic or seizure-like activity, and SCA should not be mistaken for a seizure.
- Agonal respirations or gasping and inaccurate rescuer assessment of pulse can also delay recognition of SCA.
- **SCA should be suspected in any collapsed and unresponsive athlete,** and an AED should be applied as soon as possible for rhythm analysis and defibrillation, if indicated.
- Commotio cordis should be suspected in any collapsed athlete who has been struck in the chest.
- CPR should be performed while waiting for an AED. "Hands-off" CPR is currently recommended for lay responders.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE HYPERTENSIVE ATHLETE

# 36

Mark W. Niedfeldt • Leon Y. Cheng

## GENERAL PRINCIPLES

- An estimated 76.4 million Americans over the age of 20 years have hypertension (HTN).
- HTN is the most common cardiovascular condition observed in competitive athletes.
- Athletes are usually considered to be free from cardiovascular disease because of their apparent high level of fitness.
  - The overall incidence of HTN in athletes is approximately 50% less than that in the general population.
  - Most cases are in the mild-to-moderate range.
- HTN begins in young adulthood.
  - Incidence increases with age.
    - 5%–10% in adults aged 20–30 years
    - 20%–35% in middle-aged adults
    - >50% in adults aged over 60 years
    - Residual lifetime risk of 90%
  - Almost 80% of adolescents with an elevated blood pressure (BP) (>142/92 mmHg) during preparticipation physical examinations have HTN.

## CLASSIFICATION OF HYPERTENSION (JNC-7)

- Progresses through three classifications (Fig. 36.1, Table 36.1)
- Normal systolic BP is <120 mmHg and diastolic BP is <80 mmHg
- **Prehypertension systolic BP is 120–139 mmHg and diastolic BP is 80–89 mmHg.**
  - Associated with increased cardiac output (CO), which primarily increases systolic BP, along with "normal" vascular total peripheral resistance (TPR)
  - TPR is normal compared with resting levels in normotensives but inappropriately high when CO is elevated.
    - In a nonhypertensive patient, TPR falls to compensate for a rise in CO, thereby maintaining normal BP.
    - Lack of decrease in TPR is a result of impaired baroreceptor function.
    - Baroreceptors are "reset" to maintain elevated rather than normal BP over time.
  - People with prehypertension are hypersensitive to catecholamine secretion and mental stress and have a hyperkinetic circulatory state.
- **Stage 1: systolic BP 140–159 mmHg and diastolic BP 90–99 mmHg**
  - Earliest stage and most common form detected in clinical settings
  - Increased heart rate (HR) and CO and decreased TPR
  - Decreased arterial lumen and disturbed autoregulation of blood flow in the periphery
- **Stage 2: systolic BP >160 mmHg and diastolic BP >100 mmHg**
  - Normal HR and CO
  - Increased TPR
    - Increased afterload leads to left ventricular hypertrophy (LVH) and increased diastolic BP. Severe and/or uncontrolled HTN may lead to the development of diastolic dysfunction and congestive heart failure.
    - CO can no longer increase in response to exercise or other physiologic demands.
    - Loss of contractility and congestive heart failure may develop.
- Most active individuals with HTN will fall into stage 1 or lower stage 2 (160s/100s).
- Those with comorbidities, such as diabetes or renal disease, should be treated at prehypertensive levels.
- Values for pediatric athletes are adjusted for age, gender, and height (Table 36.2).
- Higher stages are associated with a higher risk of nonfatal and fatal cardiovascular disease as well as progressive renal disease (Fig. 36.2).

## CLINICAL PATHOPHYSIOLOGY OF HYPERTENSION

### Primary Hypertension

- 95% of cases
- Abnormal neuroreflexes and sympathetic control of peripheral resistance
- Abnormal renal and metabolic control of vascular volume and compliance
- Abnormal local smooth muscle and endothelial control of vascular resistance
- Sustained increases in systemic vascular resistance (SVR)

### Secondary Hypertension

- 5% of cases
- Younger patients or adults with rapid onset of HTN and no prior history of HTN
- BP is often poorly responsive to routine therapy.

#### Causes

- **Renal (most common)**
  - Renovascular disease
    - Increased renin stimulates conversion of angiotensin I to angiotensin II, which is a vasoconstrictor, as well as release of aldosterone
    - Renal retention of sodium and water
    - Fibromuscular dysplasia in younger patients and atherosclerosis in older patients
  - Renal parenchymal disease
    - Inability of damaged kidneys to excrete sodium and water
- **Endocrine**
  - Adrenal
    - Pheochromocytoma
    - Cushing syndrome
    - Primary aldosteronism
  - Thyroid
    - Hyperthyroidism
    - Hypothyroidism
  - Acromegaly
  - Hyperparathyroidism
  - Estrogen
    - Oral contraceptive pills (OCPs)
    - 5% will develop HTN over 5 years
- **Other**
  - Coarctation of the aorta
  - Obstructive sleep apnea

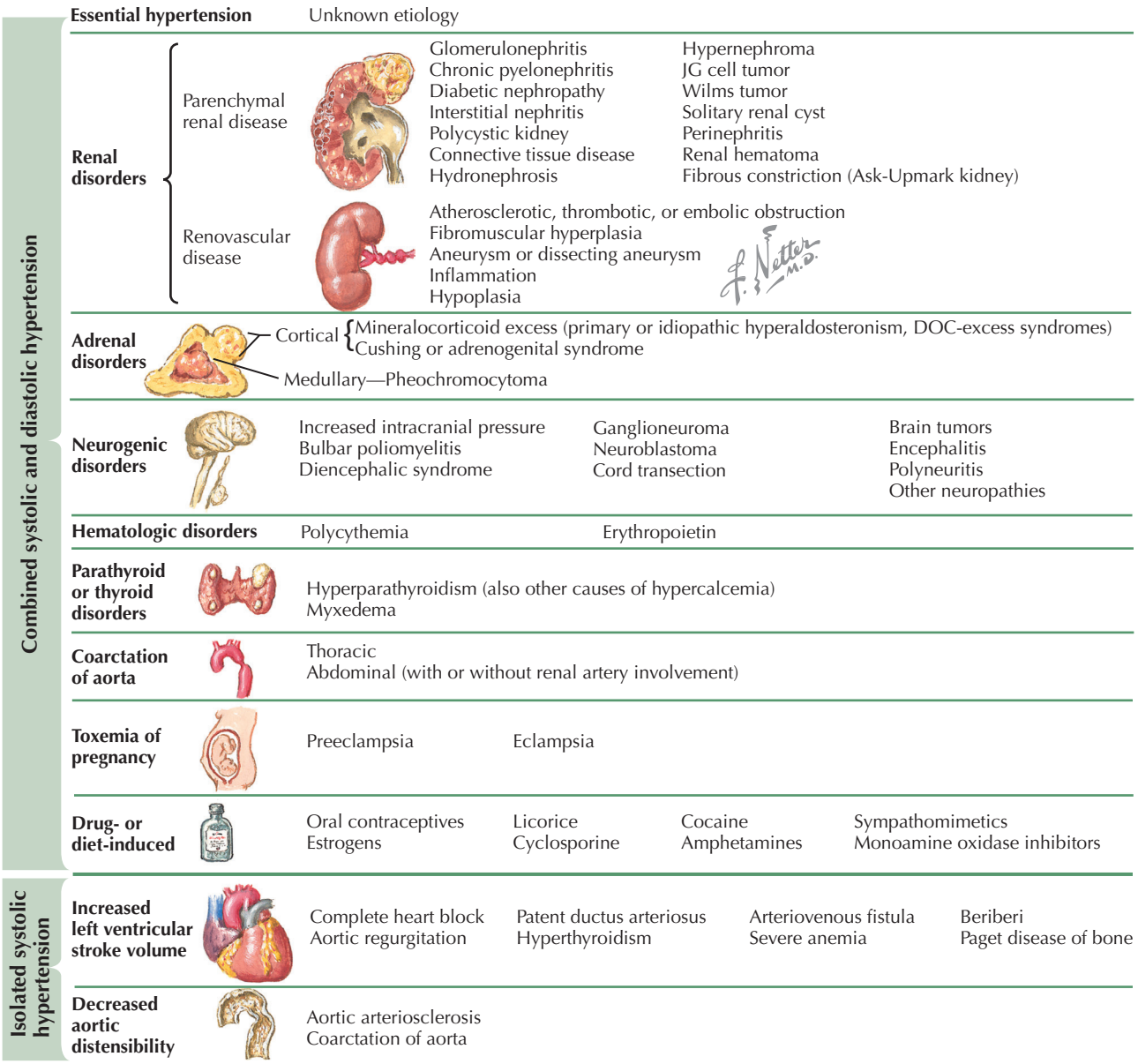



**Figure 36.1.** Causes of hypertension.

## RISK FACTORS FOR HYPERTENSION

- **Genetic factors**
  - Males more than females
  - African-Americans more than Caucasians (2 : 1) with Asians at the lowest risk
  - Family history (HTN twice as common if one or both parents have HTN)
- **Metabolic factors**
  - Obesity
  - Glucose intolerance
  - Endocrine disorders (see Causes)
- **Stress**
  - Environmental
  - Social
  - Leads to chronic neurogenic activation of the sympathetic nervous system
- **Behavioral factors**
  - High sodium intake
  - Excessive alcohol consumption
  - Drug abuse
    - Recreational: cocaine or tobacco (chew)
    - Ergogenic: stimulants or anabolic steroids

## DIAGNOSIS OF HYPERTENSION

### Resting Blood Pressure (BP)

- Diagnosis of HTN is based on an average of two or more appropriately measured, seated BP readings >140/90 mmHg at each of two or more office visits (see Table 36.1).
- In children and adolescents, HTN is defined as average systolic or diastolic BP ≥95th percentile for age, gender, and height, measured on three separate occasions (see Table 36.2).

### Environment During Measurement

- Measurement of BP should be performed in a standard measurement situation, preferably a quiet area.
- Let the athlete sit for a few minutes if possible.

- Repeat BP measurements if elevated.
- Choose the appropriate-size BP cuff. Several athletes will need a large cuff, and a thigh cuff should be available for very large athletes.
  - The inflatable bag of the BP cuff should cover approximately 80% of the arm's circumference.
  - BP may be overestimated if the BP cuff is too small, while BP may be underestimated if the BP cuff is too big.
- Avoid rapid deflation of the cuff.

## TABLE 36.1 CLASSIFICATION OF HYPERTENSION

| | Systolic (mmHg) | Diastolic (mmHg) |
|---|---|---|
| Normal | <120 | <80 |
| Prehypertension | 120–139 | 80–89 |
| Stage 1 hypertension | 140–159 | 90–99 |
| Stage 2 hypertension | ≥160 | ≥100 |

Classification of blood pressure (BP) for adults aged ≥18 years. The classification is based on the mean of two or more appropriately measured seated BP readings on each of two or more office visits. In contrast with the classification provided in the JNC VI report, a new category designated prehypertension has been added, and stage 2 and 3 hypertension have been combined. Patients with prehypertension are at an increased risk of progression to hypertension; those in the 130/80 to 139/89 mmHg BP range are at twice the risk of developing hypertension as those with lower values.
Adapted from the Seventh Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC-7). *JAMA.* 2003;289(19):2560-2575.

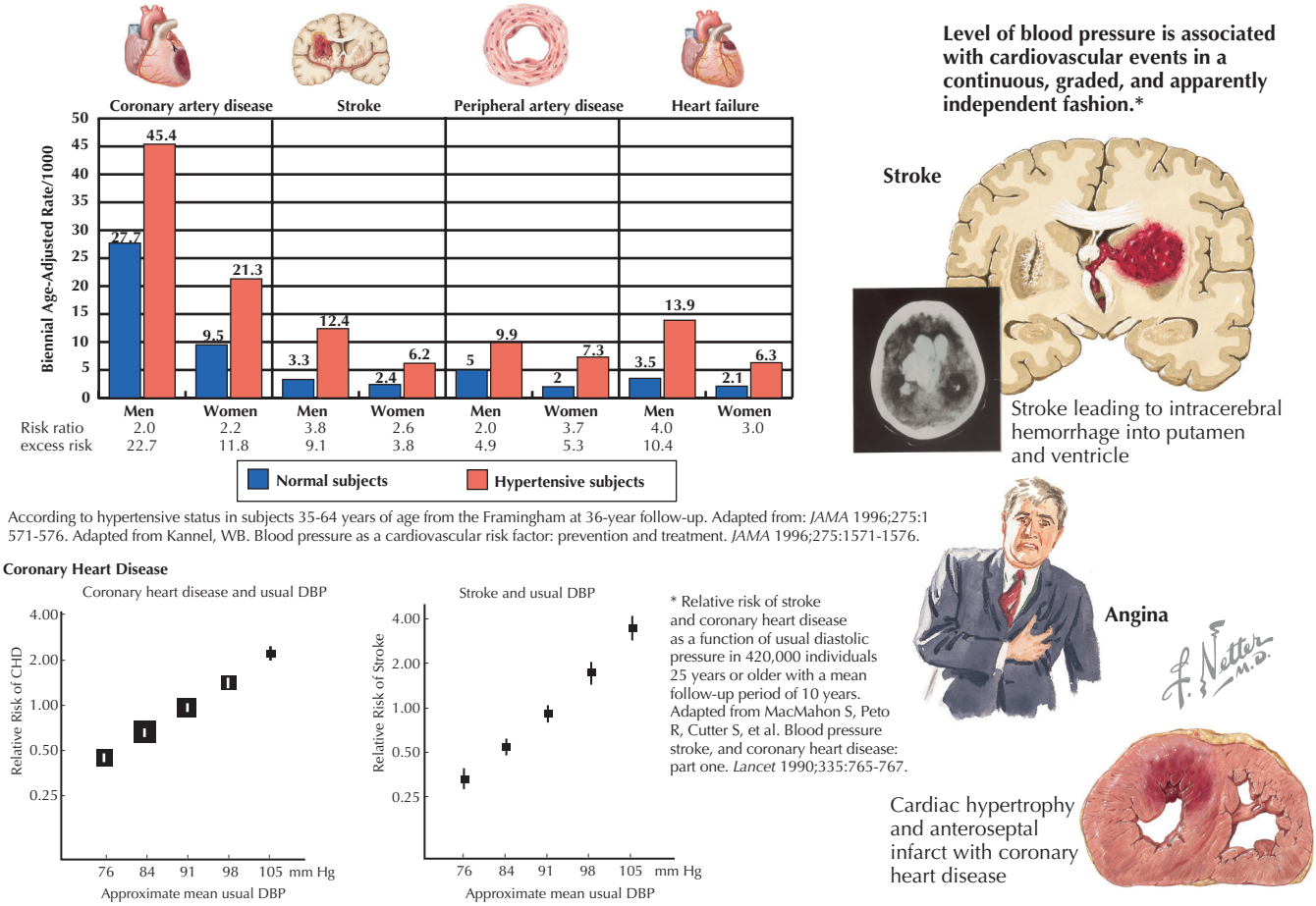
## "White Coat" Hypertension and Other Stress Phenomena

- Anxiety provoked by medical examination or other sources of mental stress can lead to artificially elevated BP, known as "white coat" HTN.

## TABLE 36.2 CLASSIFICATION OF BLOOD PRESSURE FOR PEDIATRIC ATHLETES

| | |
|---|---|
| Normal | Systolic and diastolic BP <90th percentile |
| Prehypertension | Systolic and/or diastolic BP ≥90th percentile but <95th percentile, or if BP exceeds 120/80 mmHg even if <90th percentile* |
| Stage I hypertension | Systolic and/or diastolic BP between the 95th percentile and 5 mmHg above the 99th percentile |
| Stage 2 hypertension | Systolic and/or diastolic BP, 99th percentile plus 5 mmHg |

*A systolic pressure of 120 mmHg may typically occur at 12 years of age, whereas a diastolic pressure of 80 mmHg typically occurs at 16 years of age. In children, the aforementioned definitions, based on the 2004 National High Blood Pressure Education Program Working Group (NHBPEP), are used to classify BP measurements. BP percentiles are based on gender, age, and height and on measurements on three separate occasions. The systolic and diastolic BP are of equal importance; if there is a disparity in category, the higher value determines severity of HTN.
From The National Heart, Lung, and Blood Institute. The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents. *Pediatrics.* 2004;114(2):555.



According to hypertensive status in subjects 35-64 years of age from the Framingham at 36-year follow-up. Adapted from: *JAMA* 1996;275:1 571-576. Adapted from Kannel, WB. Blood pressure as a cardiovascular risk factor: prevention and treatment. *JAMA* 1996;275:1571-1576.

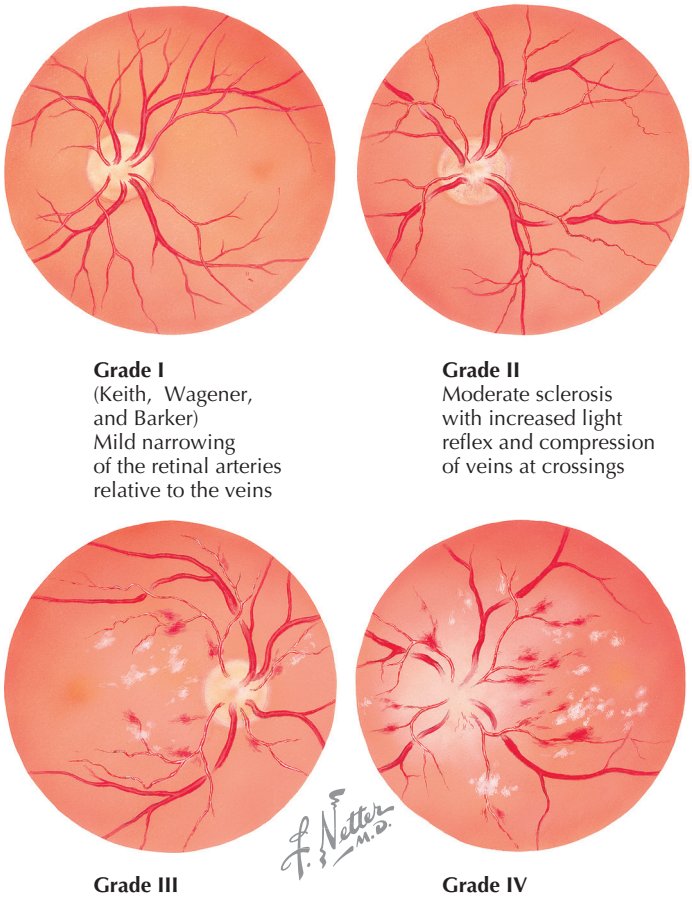
**Figure 36.2.** Hypertension as risk factor for cardiovascular disease.

- Average of several readings is a better estimate of true BP.
- If initial BP is high, have the athlete rest for 5 minutes and repeat BP measurement.
- If BP remains elevated, check BP at least once per week for at least two additional visits.
- Averaged daily BP is a better predictor of later end-organ damage than random office BP.
- Ambulatory 24-hour BP monitoring (ABPM) may more accurately assess BP in people with variable readings in the office or at home.
- Home BP monitoring is readily available and correlates well with ABPM.
- Importance of *accurate* readings should be emphasized.

## CLINICAL EVALUATION

### History

- **Cardiovascular risk factors:**
  - Smoking
  - Family history of cardiac disease in men younger than 55 years and women younger than 65 years
  - Obesity
  - Physical inactivity
  - Diabetes
  - Dyslipidemia
- **Diet and behavior:**
  - High sodium and saturated fat intake
  - Alcohol consumption
  - Herbs and supplements (particularly those for energy or weight loss)
  - Drug use
    - Over the counter (nonsteroidal anti-inflammatory drugs [NSAIDs], decongestants, caffeine, and diet pills)
    - Prescription (glucocorticosteroids, erythropoietin, cyclosporine, methylphenidates, and amphetamines)
    - Illicit (ergogenic aids, cocaine, and phencyclidine [PCP])
- Stress
- Review of systems to rule out secondary causes of HTN




**Grade I**
(Keith, Wagener, and Barker)
Mild narrowing of the retinal arteries relative to the veins

**Grade II**
Moderate sclerosis with increased light reflex and compression of veins at crossings

**Grade III**
Edema, exudates, and hemorrhages; sclerotic and markedly spastic ("silver-wire") arteries

**Grade IV**
Papilledema or choked disc, extensive hemorrhages, and exudates

**Figure 36.3.** Eye grounds in hypertension.

### Physical Examination

- Evaluate for secondary causes of HTN and end-organ damage
- Body mass index (BMI) often not useful in athletes because of higher levels of muscle mass
- Fundoscopic examination for retinopathy, as indicated by retinal hemorrhages or exudates, with or without papilledema (Fig. 36.3)
- Thyroid
- Cardiovascular (pulses, murmurs, and bruits)
- Abdominal masses
- Peripheral edema

### Laboratory Studies

- Hematocrit, Na+, K+, BUN, creatinine, glucose, lipid panel, urinalysis, EKG
- Further workup if suspicious of secondary causes
  - Renal ultrasound recommended for pediatric athletes with established HTN
  - Echocardiogram recommended in pediatric athletes with diabetes or renal disease associated with a BP between the 90$^{th}$ and 94$^{th}$ percentiles and all children with stage 2 HTN and BP in the ≥95$^{th}$ percentile

### Exercise Stress Testing

- **Although not routinely performed, exercise stress testing can be used to predict and differentiate types of HTN.**
- Differentiating stages of HTN
  - People with prehypertension start at higher resting levels than normotensives but do not show abnormally high BP levels during maximal exercise.
  - Rapid elevation in systolic BP indicates established HTN.
  - Hypertensives tend to have increased diastolic BP during and after exercise.
- Predicting future HTN
  - Exaggerated diastolic BP response to exercise (>95$^{th}$ percentile) predicts the risk of new-onset HTN in men and women (2–4 times higher risk).
  - Delayed recovery of systolic BP response is predictive of future HTN in men.
  - Exercise BP may be a significant predictor of adverse cardiovascular events in high-risk patients.

## APPROACHES TO MANAGEMENT OF HYPERTENSION

- Management of hypertensive athletes (or exercising adults with HTN) typically involves a combination of nonpharmacologic therapies, often with one or more antihypertensive medications.
- Goals
  - Goal BP varies according to patients' age and comorbidities
  - BP <140/90 mmHg for most athletes
  - BP <130/80 mmHg if coexisting disease (chronic kidney disease [CKD] with proteinuria)

  - BP <120/90 mmHg for nondiabetic adults aged ≥50 years with risk factors for cardiovascular disease or with CKD
- Special considerations in athletes
  - Potential influence of exercise and training on HTN
  - Potential side effects of antihypertensive medications on athletic performance
  - Certain therapies are banned by various sports governing bodies, including the International Olympic Committee (IOC) and the National Collegiate Athletic Association (NCAA).

## Nonpharmacologic Therapy (See Table 36.3)

- Nonpharmacologic strategies provide safe and effective foundation for any good antihypertensive regimen with low to nonexistent risk of side effects.
- Dependent on long-term compliance and lifestyle changes, which can be difficult because of deeply ingrained lifestyle behaviors
- Most appropriate for prehypertension and stage 1 HTN

### Dietary Interventions

WEIGHT REDUCTION

- Obesity is defined as BMI >30 kg/m$^2$.
- Obesity increases preload and afterload, resulting in hypertensive effect, but mechanism not well understood

**TABLE 36.3 NONPHARMACOLOGIC STRATEGIES FOR MANAGING HYPERTENSION: SORT KEY RECOMMENDATIONS FOR PRACTICE**

| Clinical Recommendations | Evidence Rating |
|---|---|
| A diet that emphasizes vegetables, fruits, and whole grains is recommended to lower blood pressure. | A |
| Limiting sodium intake to 2,400 mg per day is recommended to lower blood pressure. Additional benefit occurs with a limit of 1,500 mg per day. | B |
| To lower blood pressure, patients should engage in moderate to vigorous aerobic physical activity three or four times per week for an average of 40 minutes per session. | A |
| Clinicians should ask all adults about tobacco use and provide tobacco cessation interventions for those who use tobacco products. | A |
| To lower blood pressure, alcohol consumption should be limited to no more than two drinks per day for most men and one drink per day for women. | C |
| Self-measured blood pressure monitoring, with or without additional support (e.g., education, counseling, telemedicine, home visits, Web-based logging), lowers blood pressure compared with usual care, although the benefits beyond 12 months are not clear. | A |
| Patients with hypertension and obstructive sleep apnea should use continuous positive airway pressure to lower blood pressure. | C |

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go tohttp://www.aafp.org/afpsort.
From Wexler R, Aukerman G. Nonpharmacologic Management of Hypertension: What Works? *Am Fam Physician.* 2015 Jun 1;91(11):772-776.

- Hyperinsulinemia and insulin resistance may play a central role because obesity, glucose intolerance, and HTN are components of the metabolic syndrome.
- Weight reduction is the most effective nonpharmacologic measure of BP reduction.
  - For each 1 kg of weight loss, the systolic and diastolic BP each fall by approximately 1 mmHg.
  - Weight reduction of 10 pounds reduces BP in overweight athletes with HTN and enhances BP-lowering effects of medications, possibly because of reductions in both preload and afterload.
- Dietary approach to stop hypertension (DASH) diet
  - Reduced levels of total and saturated fat and cholesterol and sweets
  - Increased potassium, calcium, magnesium, fiber, and protein
  - Emphasizes fruits, vegetables, low-fat dairy, whole grains, fish, poultry, and nuts
  - Showed decreases in BP equivalent to medications (11.4/5.5 mmHg in hypertensive patients and 5.5/3.0 mmHg decrease in those with prehypertension) and is particularly effective in African-Americans
- Fish oil supplementation
  - High doses may decrease BP by 6/4 mmHg, but long-term safety of fish oil in doses high enough to lower BP is unknown.

ELECTROLYTES

- **Sodium ($Na^+$)**
  - A reduction in sodium can result in a significant decrease in BP.
  - 2–4 g/day can reduce systolic BP by 2–8 mmHg.
  - Recommended intake is 2.4 g/day (6 g NaCl).
  - Salt-sensitive groups benefit most:
    - Two-thirds of African-Americans
    - Diabetics
    - Older people
- **Potassium ($K^+$)**
  - Increased potassium intake in hypertensives can lower BP by a mean of 5.3/3.1 mmHg.
  - Increasing $K^+$ decreases BP in hypokalemic patients (endurance athletes) and may protect from ventricular ectopy.
  - Effects of potassium supplementation greatest in those with higher levels of sodium in diet
  - Goal is 90 mmol/day.
- **Calcium ($Ca^{2+}$)**
  - 800–1200 mg/day of elemental calcium may reduce BP in calcium-deficient (particularly women), but the overall effect is minimal.
  - Supplementation not routinely recommended for treatment of HTN
- **Magnesium ($Mg^{2+}$)**
  - Controversial, possible benefits
  - Important in patients who are deficient, particularly from diuretics
  - Supplementation not routinely recommended

### Exercise

- Vigorous exercise correlates with a lower risk of developing HTN.
  - Decrease in BP dependent more on intensity of exercise than frequency
  - Both aerobic exercise and resistance training (e.g., weight-lifting) lower resting BP; therefore, combination of aerobic activity and resistance training may have additive effects on lowering BP.
- Regular aerobic exercise regimen in people with essential HTN can lower BP by as much as 5–15 mmHg within 4 weeks.

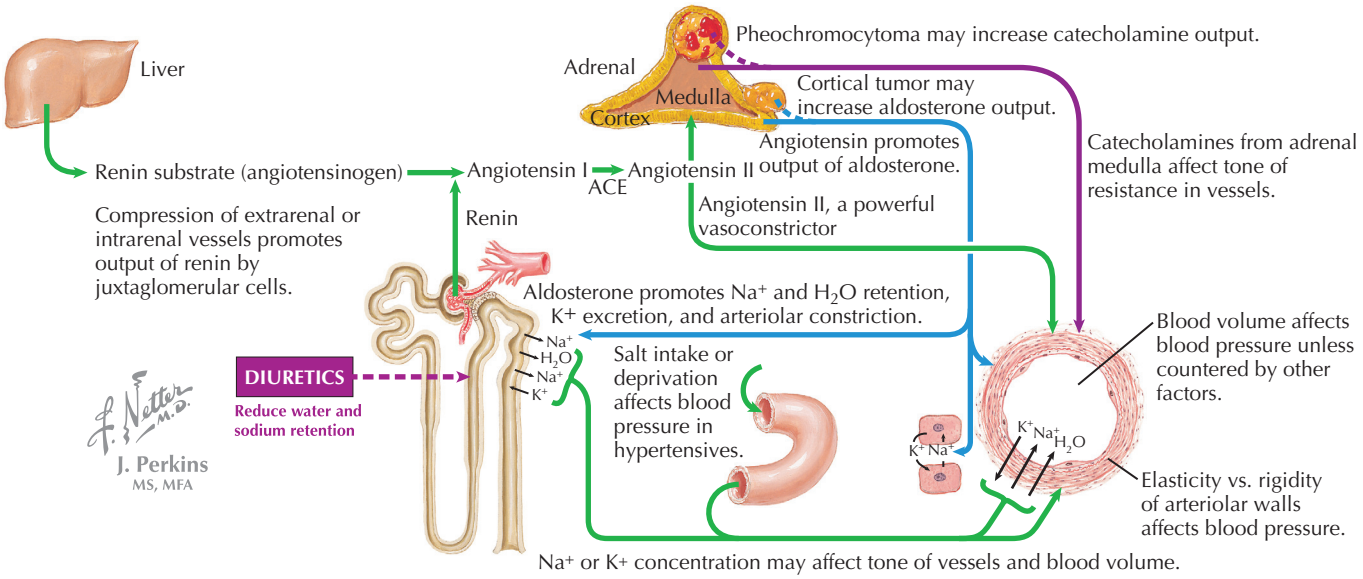



**Figure 36.4.** Hypertension treatment: diuretics.

- Certain clinical trials show that aerobic exercise conditioning lowers resting systolic BP by an average of 11 mmHg and diastolic BP by an average of 6 mmHg in hypertensives.
  - Benefits more marked in prehypertension and stage 1 HTN
  - Difficult to determine if benefit is a direct effect of exercise or secondary to weight loss
- Moderate-intensity resistance training lowers resting BP by 4/4 mmHg.

### Lifestyle Changes

- Alcohol consumption should be limited to 1 ounce (2 beers) of alcohol per day.
- Stimulants—caffeine acutely raises BP, but avoidance is unnecessary because of the rapid development of tolerance
- Cocaine
- Anabolic steroids
- Other medications such as sympathomimetics (decongestants), appetite suppressants, and OCPs may affect BP.

### Relaxation Techniques

- Biofeedback, stress management, muscle relaxation techniques, meditation, and yoga may be used as adjunctive therapies.

## Pharmacologic Therapy

### Basic Concepts

- Must be individualized and carefully monitored for potential side effects
- Most individuals will require two or more medications to achieve goal BP.
- If initial BP is >20/10 mmHg over goal, consider initiating therapy with two agents.
- Monitor effects of therapy on performance.
- NSAIDs may decrease the activity of several antihypertensive medications.
  - Beta-blockers, ACE inhibitors, and diuretics may have their therapeutic activity inhibited.
  - Limited reports suggest possibility of acute increase in BP with uses of cyclooxygenase-2 (COX-2) inhibitors.
- IOC and NCAA regulations ban the use of certain medications such as diuretics and beta-blockers (in certain events).

### Diuretics (Thiazides and Loop Inhibitors)

- Decrease plasma volume, CO, and SVR (Fig. 36.4)
  - Short-term use reduces maximal exercise capacity and submaximal endurance.
  - Attenuated increase in BP, decreased SV, but maintenance of normal HR response during exercise.
- Decreased mortality and morbidity in the elderly and are superior in preventing one or more forms of cardiovascular disease
- Inexpensive
- Side effects
  - Hypovolemia, orthostatic hypotension—not recommended for athletes prone to dehydration because of further reductions in intravascular volume.
  - Urinary loss of $K^+$ and $Mg^{2+}$, which can lead to muscle cramps, arrhythmias, and rhabdomyolysis, particularly when competing or exercising vigorously in warm weather. Cramping may occur despite normal serum potassium.
  - Increases in plasma cholesterol, glucose, and uric acid at higher doses
  - Higher incidence of sexual dysfunction in males
- Thiazides are the preferred first step in therapy for casual exercisers, active elderly, and African-Americans.
- Loop diuretics are inappropriate for use in athletes.
- All diuretics are banned by athletic associations and cannot be used by athletes subject to drug testing because they dilute the concentration of steroids and drugs in the urine.

### Angiotensin-Converting Enzyme (ACE) Inhibitors

- Captopril, enalapril, lisinopril, benazepril, fosinopril, quinapril, and ramipril (Fig. 36.5)
- Competitive inhibition of conversion of angiotensin I to angiotensin II by ACE in plasma and vascular smooth muscles
- Blocks vasoconstriction and $Na^+$ retention from angiotensin II
- Increases stroke volume, slight decrease in HR, and decreases TPR
- Beneficial effects in patients with heart failure, systolic dysfunction, or nephropathy; reverses ventricular hypertrophy and microalbuminuria and preserves renal function
- In exercise
  - No major effect on energy metabolism
  - No impairment of $\dot{V}O_2$ max, training, or competition

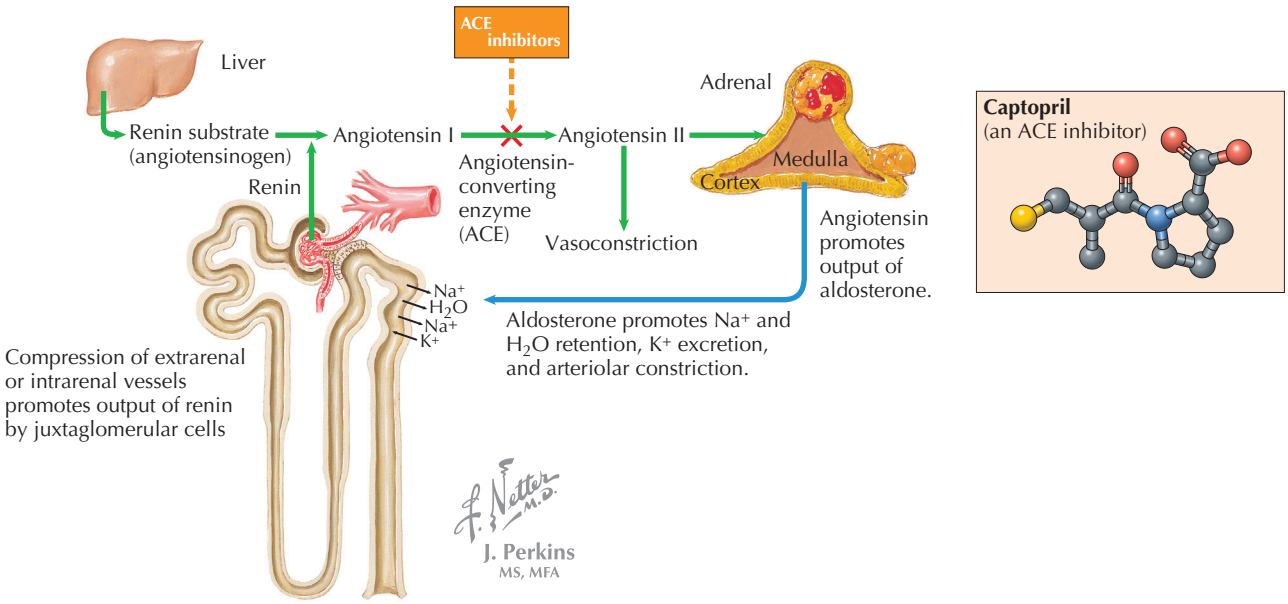



**Figure 36.5.** Hypertension treatment: angiotensin-converting enzyme inhibitors.

- Decreased "exaggerated" BP effects during exercise, including isometric exercise
- Major side effect is dry, nonproductive cough
- Anecdotal reports of postural hypotension when stopping abruptly after intense exercise (adequate cool down needed)

- Excellent for mild to moderate HTN, and effectiveness may be improved with addition of low-dose thiazide diuretics
- Often first-line agent for HTN in active athletes
- Concomitant use of NSAIDs may increase potassium-sparing effects and potentially cause hyperkalemia.
- Women of childbearing age need contraception because this class is contraindicated in pregnancy.

### Angiotensin II Receptor Blockers

- Irbesartan, losartan, valsartan, and candesartan
- Block renin-angiotensin system by preventing angiotensin II from binding to its subtype 1 receptor
- Selective blockade prevents vasoconstriction and aldosterone secretion
- Effects similar to ACE inhibitors but avoids the most common side effect of dry cough
- May improve exercise tolerance in hypertensives because angiotensin II levels increase during exercise
- May be beneficial in athletes with early diastolic dysfunction because of blocking effects during LV relaxation
- Generally recommended only for those who cannot tolerate ACE
- Women of child-bearing age need contraception because this class is contraindicated in pregnancy.

### Calcium Channel Blockers

- Include dihydropyridines (e.g., amlodipine, nifedipine, nicardipine, felodipine, and nisoldipine) and nonhydropyridines (e.g., verapamil and diltiazem)
- Inhibit calcium slow-channel conduction, reducing calcium concentration in vascular smooth muscle cells, and leading to decreased SVR (generalized vasodilatation)
- Effective in reversing ventricular hypertrophy
- Dihydropyridines (e.g., nifedipine and amlodipine)
  - Reflex tachycardia
  - Fluid retention (pedal edema)
  - Vascular headaches
- Nondihydropyridines (e.g., verapamil and diltiazem)
  - HR suppression
  - Minor impairment of maximal HR
  - Decreased LV contractility
  - Verapamil decreases pressor response during isometric exercise
  - Women show greater BP responsiveness to exercise with diltiazem than men
  - Constipation (verapamil)
- In exercise
  - No major effect on energy metabolism
  - $\dot{V}O_2$ max generally preserved
  - Potential for competitive "steal" of muscle blood flow because of vasodilatation
  - Earlier onset of lactate threshold
  - Verapamil preferred over dihydropyridines for high-intensity exercise because of improved BP control
- Generally well tolerated and effective in active patients, particularly young African-Americans
- Several physicians have found this class of drugs useful as the first-line therapy in athletes
  - May be particularly effective in hypertensive patients who are noncompliant with dietary salt restriction
  - May also be preferred in patients concurrently taking NSAIDs because antihypertensive efficacy is not blunted

### Alpha$_1$-Receptor Blockers

- Prazosin, terazosin, and doxazosin
- Competitively block postsynaptic alpha$_1$ arteriolar smooth muscle receptors
- Decrease SVR (no reflex increase in HR or CO)
- First-dose effect of syncope; hence, first dose must be administered at night
- During exercise
  - No major changes in energy metabolism
  - $\dot{V}O_2$ max preserved
  - No major effect on training or sports performance
- Useful for diabetic athletes with HTN and hypercholesterolemia
- Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT)

- Discontinued doxazosin arm because of increased congestive heart failure compared with diuretics
- Use this class with caution in master athletes aged over 55 years.

### Central Alpha Antagonists

- Clonidine, guanabenz, guanfacine, and methyldopa
- Act on alpha$_2$ receptors in brainstem to block central sympathetic stimulation with decreased HR and TPR at rest
- Also block sympathetically mediated sodium retention
- During exercise
  - No major changes in energy metabolism
  - $\dot{V}O_2$ max preserved
  - No major effect on training or sports performance
- Side effects
  - Mild to moderate drowsiness and dry mouth
  - Impotence
  - Rebound HTN with abrupt discontinuation of oral clonidine
- Clonidine available in transdermal system (patch replaced once per week)
- Rarely used because of side effects

### Beta-Blockers (See Table 36.4)

- Noncardioselective
  - Decreases HR by 20%–30%
  - Decreases contractility
  - Increases SVR (muscle and skin)
  - Inhibition of lipolysis and glycogenolysis
  - Increased cholesterol (decreased HDL)
  - Increased perception of exertion
  - Bronchoconstriction in predisposed athletes
- Cardioselective
  - Less effects on beta-2 vasodilatation, lipolysis, and glycogenolysis
  - Impairment of CO and $\dot{V}O_2$ max is generally similar but may be less because of compensatory increase in SV
- During exercise
  - Significant loss of $\dot{V}O_2$ max
    - Decreased CO and skeletal muscle flow
    - Well-trained athletes have a greater decrease in $\dot{V}O_2$ max than untrained subjects
  - Beta-blockers increase perceived exertion in working muscles, thus causing reduced endurance, probably as a result of metabolic effects with no increase in perceived cardiovascular exertion.
  - Beta-blockers decrease performance more in people with a high percentage of slow-twitch muscle fibers. This effect is more pronounced with propranolol (noncardioselective) than with atenolol (cardioselective).
  - Impairment of substrate mobilization results in earlier fatigue and lower lactate threshold.
    - Blocks lipolysis and glycogenolysis
    - Symptoms of hypoglycemia during or after intense exercise may be masked in diabetics.
  - Exercise bronchospasm may be increased; thus, use with caution in athletes with asthma.
  - Side effects less with cardioselective beta-blockers
- Not recommended in athletes unless underlying condition requires their use
  - Intermittent exertion sports
  - Untrained or partially trained athletes
  - Patients with arteriosclerotic heart disease where increased exercise tolerance may be noted
  - Hypertensives with excessive rise in systolic BP during exercise
  - Performance anxiety in minimal-exertion sports
- Banned in precision sports (e.g., shooting sports and golf)

**TABLE 36.4 BETA-BLOCKING AGENTS ARRANGED BY CARDIOSELECTIVITY AND INTRINSIC SYMPATHOMIMETIC ACTIVITY (ISA)**

| | No ISA | ISA |
|---|---|---|
| **Cardioselective** | Atenolol (Tenormin)<br>Metoprolol (Lopressor)<br>Betaxolol (Kerlone)<br>Bisoprolol (Zebeta) | Acebutolol (Sectral) |
| **Noncardioselective** | Nadolol (Corgard)<br>Propranolol (Inderal)<br>Timolol (Biocadren) | Pindolol (Visken)<br>Carteolol (Cartrol)<br>Penbutolol (Levatol) |

### Combined Alpha- and Beta-Blockers

- Labetalol and carvedilol
- Three effects (Fig. 36.6)
  - Beta-blockade (decreased HR leading to decreased CO and renin)
  - Alpha$_1$-blockade (decreased vasoconstriction leading to decreased TPR)
  - Beta$_2$-agonist (decreased TPR)
- Beta effects greater than alpha effects (3 : 1 for oral formulations)
- Decreased SVR
- Less impairment of muscle blood flow and $\dot{V}O_2$ max
- May be the best choice if beta-blockade is necessary
  - CO decreased 10%–14% at rest and during exercise after 1 year of therapy
  - CO gradually returns to baseline over next 5 years because of increased SV
  - TPR remains decreased 15%–20%
  - Exercise hemodynamics return to normal

### Other

- Direct vasodilators (e.g., hydralazine and minoxidil) directly cause smooth muscle vasodilation in arterioles. These should be used in conjunction with diuretics and beta-blockers because of reflex tachycardia and excess fluid retention.
- Peripheral-acting adrenergic antagonists (e.g., guanadrel, guanethidine, reserpine, and mecamylamine) inhibit catecholamine release and may cause serious orthostatic and exercise-induced hypotension.

## APPROACH TO ACTIVE PATIENTS

### Prehypertension

- Modify lifestyle

### Stage 1 Hypertension

- Nonpharmacologic intervention
  - 3–6 months
  - Monitor frequently
- Pharmacologic therapy
  - Low dose
  - Observe for 6–8 weeks
- Initial therapy
  - ACE, ARB, or calcium channel blocker
  - Thiazide diuretic initially or added if $Na^+$ sensitive
  - Combined beta/alpha if beta-blockade needed
- Reduce or withdraw in 6 months
  - A small proportion of individuals will remain normotensive after BP is controlled.

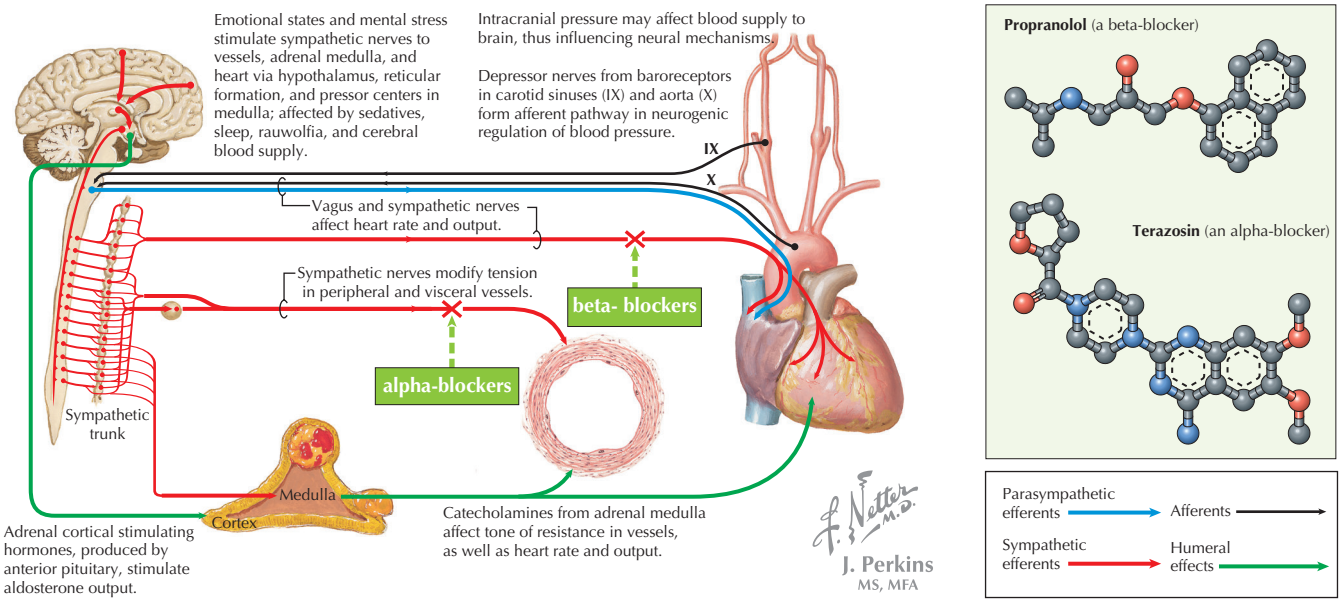
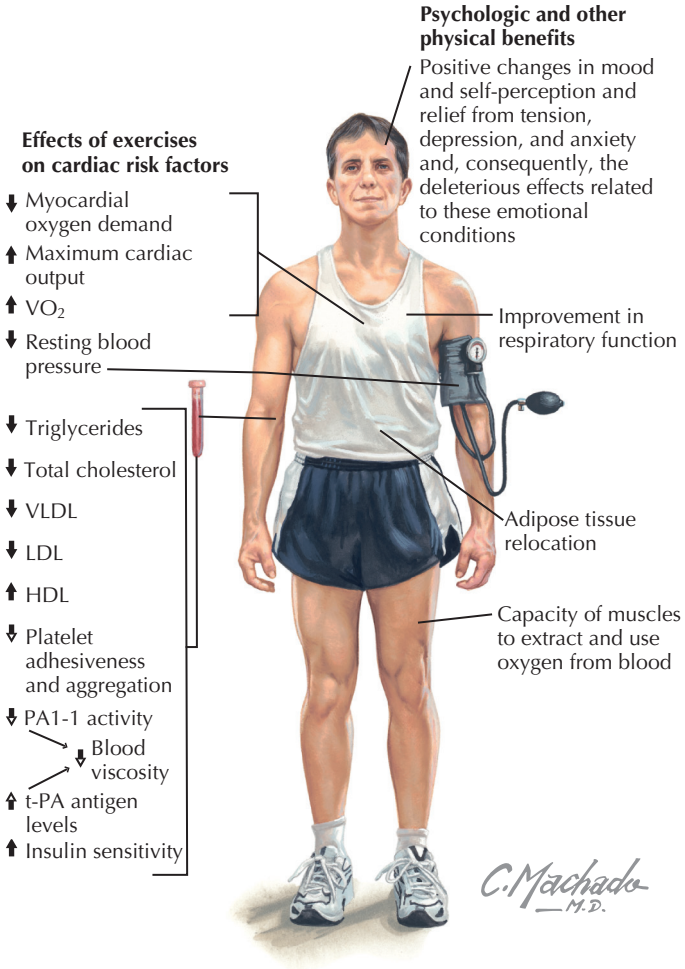



**Figure 36.6.** Hypertension treatment: beta-blockers and alpha-blockers.

- May involve reestablishing baroreceptor control mechanisms
- Emphasize need for long-term follow-up care and management

## EXERCISE PRESCRIPTION FOR HYPERTENSIVE PATIENTS

- The recommended mode, frequency, duration, and intensity of exercise is generally the same as those for nonhypertensive individuals (Fig. 36.7).
- Repetitive performance of both aerobic and resistance exercise lowers systolic and diastolic BP.
  - Each 30-minute period of aerobic exercise at 50% of maximal oxygen uptake lowers BP for 24 hours.
  - An even greater reduction is seen with exercise at 75% of maximal oxygen uptake.
- Monitor BP every 2–4 months to monitor the impact of exercise.
- Prescribing exercise—"FITT"
  - Frequency: 5–6 sessions/week
  - Intensity: 55%–70% of predicted maximum HR (MHR)
    - Moderate-intensity exercise (55%–70% MHR; defined as 40%–70% $\dot{V}O_2$ max by some) lowers BP more effectively than higher-intensity exercise (80%–85% MHR). Higher-intensity exercise may actually increase resting BP.
    - Training at 20% and 60% of maximum work capacity results in similar resting BP reductions.
    - In older or less fit patients, start at 55%–60% MHR; in relatively more fit patients, start at 65%–70% MHR
    - Predicted MHR: 220 minus age
  - Time (duration): initially 15–20 minutes/session; eventually 30–40 minutes/session
  - Type of exercise: "dynamic isotonic exercise" or moving body through space is most effective; examples include walking, jogging, swimming, cycling, cross-country skiing, and aerobic dance
- Resistance training
  - Circuit weight training may reduce BP when performed at 30%–50% maximum resistance with intervals of 15–30 seconds between stations.




The physical activity guidelines are targeted to increase physical activity to promote health but will not necessarily result in physical fitness and should not diminish the importance of achieving physical fitness.

**Figure 36.7.** Effects of exercise on cardiovascular health.

  - Progressive resistance exercise results in small reductions in resting BP.
  - Regular resistance training leads to attenuated BP and HR responses to any given load.
- Hypertensive athletes should have their BP controlled before returning to participation in vigorous sports.
- Recommendations regarding athletic participation are based on the 36th Bethesda guidelines (Table 36.5).
  - Presence of stage 1 HTN in the absence of target organ damage, including LVH or concomitant heart disease, should not limit the eligibility for any competitive sport.
  - Athletes with stage 2 HTN should be restricted from high-static sports (activities that generate large intramuscular forces with little change in muscle length) until BP is controlled because they do not shunt blood to skin as effectively, resulting in increased core temperatures and increased fluid and $K^+$.
    - Low-intensity sports are recommended.
    - Should avoid "collision" sports that could lead to kidney damage

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

**TABLE 36.5 EXERCISE RECOMMENDATIONS FOR HYPERTENSIVE ATHLETES**

| | |
|---|---|
| Prehypertension (120/80 mm Hg to 139/89 mm Hg) | No restrictions |
| Stage 1 hypertension (140/90 mm Hg to 159/99 mm Hg with no target organ damage) | No restrictions, monitor BP |
| Stage 2 hypertension (systolic BP >160 mm Hg and diastolic BP >100 mm Hg) | Restricted from high static sports until BP controlled |
| Controlled with end-organ damage | Low intensity sports recommended |
| Controlled severe hypertension | Should avoid "collision" sports that could lead to kidney damage |

Data from 36th Bethesda Conference: Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. *JACC* 2005;45(8):1318-1375.

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# EXERCISE-INDUCED BRONCHOSPASM, ANAPHYLAXIS, AND URTICARIA

37

Marc A. Molis • Whitney E. Molis

## EXERCISE-INDUCED BRONCHOSPASM

**Definitions:** Exercise-induced bronchospasm (EIB) is defined as a decline in forced expiratory volume in 1 second ($FEV_1$) or peak expiratory flow rate shortly after the onset or cessation of exercise. The terms *exercise-induced asthma* (EIA) and *EIB* are often used interchangeably; however, *EIA* should be reserved for athletes who have a known diagnosis of asthma or asthma symptoms at rest; *EIB* should refer to athletes who experience symptoms only while exercising. The rationale for the differentiation between the two terms is the difference in treatment with the mainstay of treatment for asthma and EIA being inhaled corticosteroids, which have little to no benefit in EIB. EIB treatment is aimed more at reducing symptoms while the athlete is exercising.

**Pulmonary function criteria:** Table 37.1 lists the different testing procedures approved by the World Anti-Doping Agency (WADA) and the resulting decrease in $FEV_1$ that can be used to diagnose EIB. Maximal decrease in $FEV_1$ usually occurs after 5–8 minutes of vigorous exercise. Pulmonary function tests (PFTs) usually return to baseline 60 minutes after exercise.

**Epidemiology:** Approximately 12% of the total population experiences EIB. EIB can be detected in approximately 41% of people with a history of allergic rhinitis; 40%–90% of asthmatics have EIA. EIB and EIA occur equally between both genders and can develop at any age. The rate of EIB is highest among cold weather athletes (skiing) and indoor sports athletes (ice hockey and swimming); increased prevalence in endurance athletes as well

**Risk factors:** A history of asthma is the biggest risk factor for EIA. For EIB, risk factors include a family history of asthma, allergic rhinitis, personal or family history of atopy, cold weather sports, and sports that require a high ventilation rate (e.g., Nordic skiing, soccer, and distance running).

**Mechanism:** At present, the exact mechanism of EIB remains unknown, but current understanding of the pathophysiology is that hyperventilation during exercise causes loss of heat and drying of the airways, leading to dehydration of airway cells and increased intracellular osmolarity. This creates an osmotic gradient that stimulates the release of inflammatory mediators including histamines, leukotrienes, and cytokines. These mediators, along with airway drying, cause an exaggerated response that results in EIB. After the completion of exercise, airway cooling reverses as smaller bronchial vessels warm, creating reactive hyperemia; this warming establishes another osmotic gradient that releases more mediators, causing bronchospasm and airway edema, further contributing to EIB.

**Triggers:** Oral breathing (unlike nasal breathing, which warms the air, oral breathing does not and is more likely to cause cooling of the airways and trigger EIB), dry air, cold air (essentially, colder, drier air will cause more severe symptoms), pollution, and allergens in the air; intense exercise is more likely to trigger EIB (e.g., cross-country skiing, basketball, and running); chemicals such as chlorine in pools and insecticides, pesticides, herbicides, and fertilizers used to maintain fields can trigger EIB.

**Clinical signs and symptoms:** Can include wheezing, shortness of breath, coughing (usually after exercise), chest tightness, chest pain (usually reported in children), poor athletic performance, and early fatigue compared with peers; symptoms are similar to those that occur in an acute asthma attack. They often resolve when the activity is discontinued. There can be a late-phase response that occurs 4–8 hours after exercise.

**Physical examination:** May include the following on examination:

- Lungs—wheezing (particularly expiratory phase); may hear rales or rhonchi; wheezing at rest should raise concerns for true asthma
- Skin—signs of atopic disease such as eczema
- Nose—enlarged and boggy turbinates
- Throat—cobblestoning and enlarged tonsils
- Sinus—tenderness on pressure

**Testing:** EIB used to be a disease classically diagnosed and treated by self-reported symptoms. Recent studies have shown a lack of specificity and sensitivity based on symptoms, emphasizing the need for objective testing. Following are the different diagnostic tests that are commonly used to diagnose EIB.

**Types of testing:**

- PFT can be performed using exercise challenge tests or bronchial provocation testing (where the patient inhales a substance designed to induce bronchoconstriction). Spirometry should initially be performed at rest and then immediately after exercise to establish baselines and compare results. Testing should be ideally performed for at least 8 minutes, allowing the athlete to reach >90% of peak heart rate by 2 minutes and maintaining the same for another 6 minutes.
  - The International Olympic Committee (IOC) recommends that $FEV_1$ be measured at least 3 minutes after the challenge and accepts a 10% decline in $FEV_1$ as an indication for use of a beta-2 agonist. At present, the IOC requires prior notification for the use of a beta-2 agonist; the notification must be accompanied by objective evidence that justifies the need for medication. The IOC accepts the following tests and the associated decrease in $FEV_1$ for diagnosing EIB and allowing an athlete to use a beta-2 agonist: eucapnic voluntary hyperpnea (EVH) test (10%), exercise challenge test (10%), or hyperosmolar aerosis test (15%). EVH testing is performed using dry air containing 5% carbon dioxide. Hyperosmolar aerosis can be performed using either hypertonic saline or inhaled powdered mannitol (not available in the United States).
  - Sport-specific field testing (e.g., having a hockey player skate on ice till symptoms develop) is ideal when resources are allow. Following are examples of clinical/laboratory testing that can replace field testing:
  - Free running
    - The patient runs either indoors or outdoors. This can also be performed in a stairwell if space is limited.
    - Advantages—Most likely to induce symptoms, requires minimal cardiovascular (CV) monitoring
    - Disadvantages—Depending on the season, difficult to control environmental factors such as temperature and humidity and may not trigger EIB in all patients
  - Treadmill
    - Advantages—CV and pulmonary monitoring can be performed during the workout. Workload can be standardized.

**TABLE 37.1 WADA-APPROVED BRONCHIAL PROVOCATION TESTS FOR DIAGNOSING EIB**

| Bronchial Provocation Test | Decrease in $FEV_1$ for Position Test Results |
|---|---|
| Eucapnic Voluntary Hyperpnea | >10% |
| Methacholine Aerosol Challenge | >20% |
| Mannitol Inhalation | >15% |
| Hypertonic Saline Aerosol Challenge | >15% |
| Exercise Challenge (field or laboratory) | >10% |
| Histamine Challenge | >20% |

*EIB,* Exercise-induced brochospasm; *$FEV_1$,* forced expiratory volume in 1 second; *WADA,* World Anti-Doping Agency.

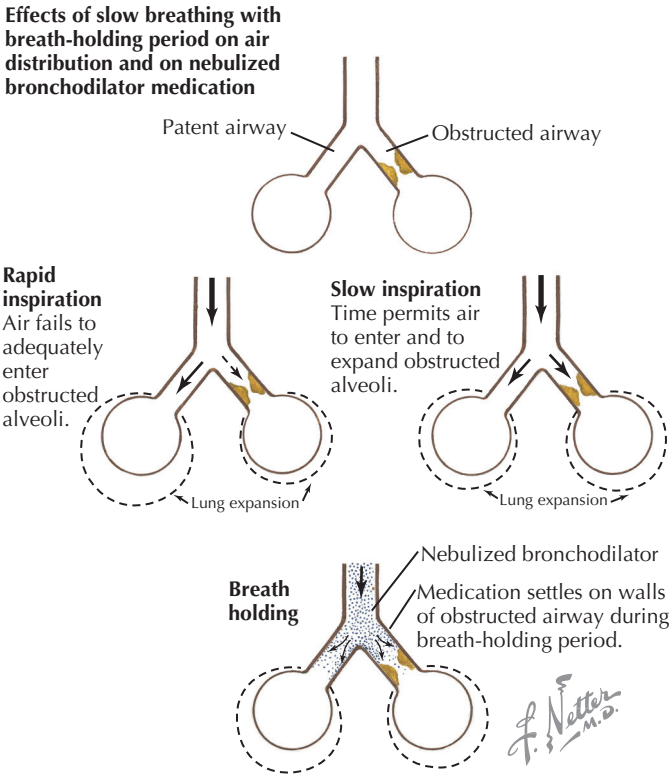


**Figure 37.1.** Bronchospasm.

- Disadvantages—Expensive equipment needed; less likely to induce EIB because it is indoors and factors such as temperature, humidity, and pollutants are more controlled.
- Bicycle ergometer
  - Advantages—Workload can be easily maintained. CV monitoring is the easiest.
  - Disadvantages—Least likely to produce EIB symptoms; need for expensive equipment
- Bronchial provocation testing
  - The patient inhales a substance designed to induce bronchoconstriction. It may be used as primary diagnostic test or when exercise challenge is equivocal. See Table 37.1 for types of testing and criteria for positive test.
- Other testing
  - Skin testing—probably the most useful because there is a strong correlation between allergies and EIB/EIA
  - Computed tomography (CT) of sinuses if there is a concern for chronic sinusitis causing the symptoms
  - Chest radiograph—may show signs of underlying lung disease
  - Echocardiogram—if a CV abnormality is a possible cause of the symptoms

**Medications and testing:** When performing PFT, certain medications must be avoided or stopped so as to not confound the testing and produce false-negative results.

- Inhalers—Albuterol and other beta-agonists should not be used 24 hours before testing.
- Inhaled steroids—stop 1 week before testing
- Theophylline—stop 24 hours before testing
- Leukotriene receptor antagonist—stop 24 hours before testing
- Cromolyn sodium/nedocromil sodium—stop 24 hours before testing
- Antihistamines—do not need to be stopped for PFT but need to be stopped 72 hours before skin testing

**Differential diagnosis:**

- **Vocal cord dysfunction (VCD)**
  - VCD can produce respiratory symptoms when the ventilatory rate rises; usually have inspiratory wheezing and/or stridor as opposed to EIB and asthma, which primarily produces expiratory wheezing. The stridor in VCD occurs because of paradoxical closure of the vocal cords. Patients complain of difficulty "getting air in" and difficulty in breathing. VCD is frequently misdiagnosed as asthma and warrants special consideration in the diagnosis of EIB.
  - The diagnosis of VCD is often made based on clinical presentation. Flow-volume loop is performed while the patient is symptomatic; flattening of the inspiratory loop can be seen. Laryngoscopy, which allows visualization of vocal cord adduction on inspiration (classically seen as adduction of the anterior two thirds of the vocal cords with a posterior diamond-shaped chink remaining open), is considered to be the gold standard of diagnostic testing for VCD. Treatment is reassurance, education, and speech therapy.
- **Gastroesophageal Reflux Disease (GERD)**
  - May present with atypical symptoms such as chronic cough and wheezing
  - Consider in athletes who have symptoms of GERD or worsening symptoms of EIB associated with regurgitation, large meals, or alcohol
- **Swimming-Induced Pulmonary Edema (SIPE)**
  - SIPE presents with shortness of breath and cough during or immediately after swimming with associated evidence of pulmonary edema. Spirometry reveals an acute restrictive pattern, with changes lasting up to 1 week.
- **Other**
  - General deconditioning
  - Chronic lung disease including asthma
  - CV disorders

**Prevention and treatment:**

- **Pharmacologic**
  - **Inhaled beta-adrenergic agonists:** Prevention and treatment can be achieved by using a short acting beta-2 agonist (e.g., albuterol) inspired approximately 15–30 minutes before exercise; this can often prevent or reduce the symptoms of EIB. Should ideally be performed using a spacer with the inhaler to maximize the concentration of the medicine; only albuterol in an aerosolized form has been shown to be effective in EIB and asthma (Fig. 37.1).
  - **Leukotriene antagonists:** Can be added as a second-line medication or occasionally as monotherapy; not as

effective in individuals with isolated EIB but may aid in athletes who have underlying asthma
  - **Inhaled corticosteroids:** Not effective in isolated EIB, but the mainstay of treatment of persistent asthma; all athletes who have persistent asthma and have trouble with exercise should be on an inhaled steroids. The exception to this may be athletes who would benefit from long-acting beta-agonists (athletes who work out multiple times a day, long-distance running where short-acting beta-agonists may not be as effective). Long-acting beta-agonists are not currently available as a solitary medication because of a black box warning for use and sudden cardiac death. However, they can be found in conjunction with inhaled steroids as so-called combination therapy, and this can be of use in certain athletes.
  - **Antihistamines/intranasal steroids:** Both will help athletes who have underlying allergic rhinitis.
  - **Other medications:** Cromolyn can be used as an adjunctive treatment for asthma. Not used as much because of the effectiveness of inhaled corticosteroids; for allergic rhinitis, nasal steroid sprays as well as oral antihistamines can help resolve symptoms
  - **See Table 37.2 for a list of governing bodies and their rules for pharmacologic treatment of asthma.**
- **Nonpharmacologic treatment and prevention**
  - Conditioning—may help to reduce symptoms and severity of EIB
  - Patients can be coached to "run through" their EIB; this means that the athlete continues to exercise while having symptoms, and certain patients can push through the transitory symptoms and continue to perform their sport.
  - Short burst of vigorous exercise (e.g., wind sprints) may be used to treat EIB and induce short-term refractoriness. Attributed to endogenous release of prostaglandins
  - Warming up before activity induces bronchodilation and refractoriness to EIB.
  - Cooling down after strenuous exercise decreases EIB symptoms after exercise.
  - Avoid hyperventilation.
  - Cold weather may exacerbate EIB; hence, dressing appropriately may help. In addition, in cold weather, a scarf may help retain warmer and more humid air, thereby reducing EIB symptoms.
  - Avoid exercising in areas that have high pollen counts or heavy pollution.

**TABLE 37.2 GOVERNING BODIES FOR PHARMACOLOGIC TREATMENT OF ASTHMA**

| Governing Body | Policy on EIB/EIA | Medications |
|---|---|---|
| NCAA | Need a written prescription from doctor for the medication | Albuterol is allowed in aerosolized form only |
| International Olympic Committee | Need a positive exercise challenge or bronchial provocation test (BPV) test<br>Need a therapeutic use exemption (TUE) form for medications | All beta-2 agonists and their D- and L-isomers are prohibited<br>Exceptions are formoterol, albuterol/salbutamol, salmeterol, and terbutaline |

*EIA,* Exercise-induced asthma; *EIB,* Exercise-induced bronchospasm; *EVH,* eucapnic voluntary hyperpnea.

  - Avoid vigorous activity when the patient has a cold or when allergies are not well controlled.
  - Use nasal breathing (as opposed to mouth breathing) to help warm the air when exercising.

## CHOLINERGIC URTICARIA

**Definition:** Cholinergic urticaria is the name given to hives that are precipitated by an increase in core body temperature; also referred to as *generalized heat urticaria*

**Epidemiology:** Cholinergic urticaria is believed to account for 5% of all cases of chronic urticaria and approximately 30% of all physical urticaria. Approximately 15% of the general population will experience at least one episode in their lifetime. Typical onset is 2nd or 3rd decade of life. Familial cases have been reported but are rare (all affected patients in these cases were males with father-to-son transmission). There may be a slight predominance in male gender.

**Triggers:** Include exercise, strong emotions, and bathing in hot water (essentially any trigger that causes a rise in core body temperature may provoke an onset of cholinergic urticaria); ingestion of spicy or hot foods can also be a trigger. All these factors lead to increased sweat production, which may have certain bearing on the pathogenesis of the condition.

**Pathogenesis:** Exact mechanism not known, but thought to be due to an abnormal cutaneous response to the presence of cholinergic agents, releasing mediators that cause an urticarial response. Studies have shown elevated plasma histamine in such patients.

**Clinical signs and symptoms:** Typical appearance is that of numerous punctate wheals (1–3 mm) surrounded by large flares. As the response progresses, the flares may coalesce to form large areas of erythema. The wheals typically begin on the trunk and neck and spread distally to involve the face and extremities (Fig. 37.2). Patients will often experience various sensations including itching, burning, or tingling; can progress to systemic symptoms such as hypotension, angioedema, and bronchospasm (this is rare); can also have signs of cholinergic stimulation such as lacrimation, salivation, and diarrhea (also rare)

**Testing:** The presence of classic lesions in the context of a typical inciting trigger is often enough to suggest a diagnosis, but confirmatory testing can be attempted. Provocation testing:
  - Typically, a methacholine injection should be positive in patients with cholinergic urticaria; this is accomplished by an intradermal injection of methacholine in saline, and a positive test produces hives around the injection site. However, this test is only positive in about one third of patients who do have cholinergic urticaria.
  - Can also conduct specific provocation testing to provoke a response; would include having the patient mimic the activity (e.g., exercising or eating certain foods) to try and produce a reaction, including exercising in a warm room
  - Can also try and raise the core body temperature by having the patient partially submerged in a hot water bath at 40°C until the body temperature has been raised by 0.7°C; the appearance of generalized urticaria confirms the diagnosis. Note: Aquagenic urticaria may be precipitated by this procedure, but the wheals should only occur in submerged portions of the skin.

**Treatment: Nonpharmacologic:** Identification and avoidance of known triggers is the first step in controlling cholinergic urticaria. Avoid strenuous exercise in hot weather. Avoid bathing in overly hot water. **Pharmacologic:** Antihistamines are the treatment of choice, and among these, hydroxyzine is the agent of choice. A low dose should be initiated and gradually increased until the urticaria is controlled (typically, 100–200 mg divided over 24 hours).

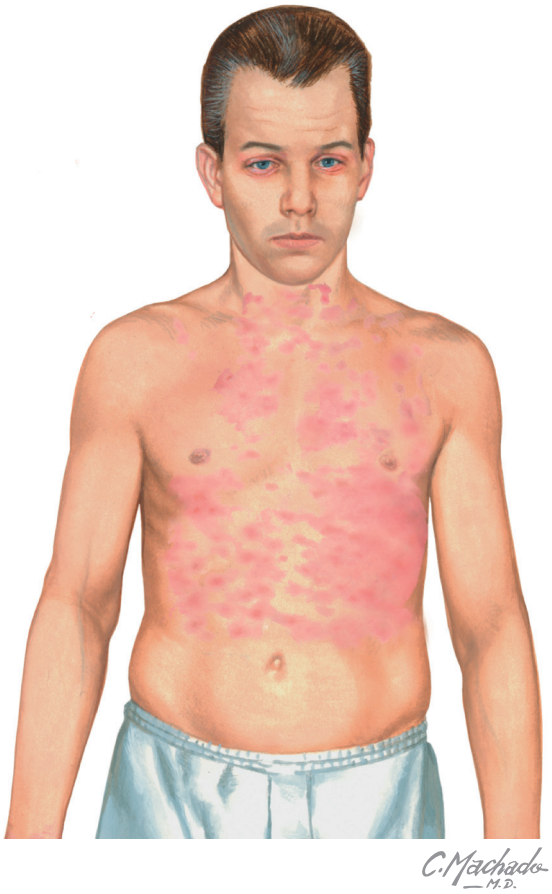


Figure 37.2. Urticaria.

## EXERCISE-INDUCED ANAPHYLAXIS

**Definition:** Exercise-induced anaphylaxis is a distinct form of physical allergy wherein exercise can cause a spectrum of symptoms, including pruritus, urticaria, angioedema, wheezing, hypotension, syncope, and even death. In such patients, urticaria is an early sign of true anaphylaxis.

**Epidemiology:** Age of onset ranges from early childhood through adulthood and seems to be more prevalent in females. Approximately 50% of patients have a history of atopy.

**Triggers:** Can be triggered by any physical activity but most commonly seen with aerobic sports and jogging; factors that have been associated with exercise-induced anaphylaxis include menstruation, use of aspirin and nonsteroidal anti-inflammatory drugs (NSAIDs), and exposure to cold weather. Certain patients have a food-dependent variant where they need to ingest a certain food item and then exercise to provoke an anaphylactic reaction. Common foods associated with these reactions are celery, shellfish, cheese, eggs, chicken, hazelnuts, oranges, apples, peaches, grapes, wheat, and cabbage.

**Pathogenesis:** The exact mechanism remains unknown, but mast cells are known to be a crucial factor because of studies that have demonstrated elevation of serum histamine and tryptase levels during attacks. In addition, studies have shown mast cell degeneration on skin biopsies performed after attacks; may be a priming phenomenon at work where one stimulus acts as a cofactor for the reaction to occur. The food and medication may act as the primer, and exercise then triggers the event.

**Clinical signs and symptoms:** Within several minutes of exercising, patients experience a prodromal phase that consists of fatigue, warmth, pruritus, and erythema. These symptoms then progress to large hives that become confluent and eventually appear as angioedema. If the patient continues to exercise, the attack progresses to systemic anaphylaxis with CV (hypotension or syncope), respiratory (wheezing or stridor), and gastrointestinal (nausea, vomiting, abdominal pain, and colic) symptoms. Once an attack occurs, it lasts from 30 minutes to 4 hours. A late phase has been described, which can cause headache, fatigue, and warmth that lasts for 24–72 hours.

**Testing:** Exercise testing is the preferred diagnostic method. Testing must be performed in a controlled environment, with the appropriate medical personnel, epinephrine, and resuscitative equipment present. Vital signs and spirometry should be monitored. It is recommended that an IV be in place to both draw serum markers and administer medications. False-negative challenges are common, so the testing may need to be repeated on multiple occasions.

**Treatment:**

- **Nonpharmacologic:** The first step in effective management is to identify and avoid and specific foods, medicines, or other triggers; may need to limit the intensity and/or frequency of exercise. Avoid exercising 4–6 hours after eating or during extremely hot, humid, or cold weather.
- **Pharmacologic:** Patients should always carry self-injectable epinephrine at all times. They also need to exercise/train with a partner who can inject them with the epinephrine, if necessary. Antihistamine therapy has demonstrated only partial benefits in the prevention of anaphylaxis.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# NEUROLOGIC PROBLEMS IN THE ATHLETE

# 38

David J. Petron • Steven A. Makovitch

## EPILEPSY AND SEIZURE ACTIVITY IN ATHLETES

### Definition

**Seizure:** A transient disruption of brain function from abnormal, excessive, synchronous neuronal activity in the brain; its clinical manifestation depends on the specific region and extent of the brain involved, which may include altered motor function, sensation, alertness, perception, and autonomic function.

**Epilepsy:** An enduring predisposition to generate recurrent, unprovoked epileptic seizures; Worldwide prevalence is approximately 8.2 per 1000 individuals; 75% of epileptics experience their first seizure before the third decade of their life.

### Classification

Seizures can be systematically classified as focal or generalized.

#### Generalized

**Overview:** Seizures involve both cerebral hemispheres, are of abrupt onset, and involve alteration in consciousness.

**Tonic–clonic (grand mal):** Occurs at all ages; may have a prodromal phase lasting hours to days

Typically starts with a tonic phase (rigid extension) lasting up to 20 seconds, followed by a clonic phase (synchronous muscle jerking) lasting 1–2 minutes; most last for <1 minute. Characteristics include loss of consciousness, convulsions, muscle rigidity, and urinary but not fecal incontinence; may progress to generalized status epilepticus; a postictal phase (confusion, headache, and fatigue) is common. May be followed by focal weakness or paralysis, including vision and speech function, reflecting postictal depression of the epileptogenic cortical area; this phenomenon, known as *Todd's paralysis*, is reversible and usually resolves within 48 hours.

**Absence (petit mal):** Occurs generally in children; no prodromal or postictal phase reported. **Typical:** Onset is acute, as is recovery, with seizure activity lasting <10 seconds. **Atypical:** Lasts for >10 seconds, with more gradual onset and recovery; sudden loss of awareness with associated staring, rhythmic blinking, and possibly clonic jerks

**Generalized status epilepticus:** Defined as a continuous seizure activity lasting 30 minutes or as two or more discrete seizures between which consciousness is not fully regained. Seizures lasting >5 minutes have a high likelihood of progressing to status epilepticus and should be treated aggressively. **This is a medical emergency. Activate emergency medical services (EMS).** Ensure adequate airway, breathing, and circulation. Attempt to obtain intravenous (IV) access and to prevent aspiration. Benzodiazepines are the drugs of choice for initial treatment because they are fast acting and effective. Lorazepam at a dose of 0.1 mg/kg IV over 2 minutes is considered as first-line treatment. Consider metabolic etiologies. Complications include dysrhythmias, metabolic abnormalities, hyperthermia, pulmonary edema, rhabdomyolysis, and pulmonary aspiration.

#### Focal

**Overview:** Originate in a localized region of the brain and cause symptoms specific to the part of the cortex wherein they originate; partial seizures may become secondarily generalized.

BROAD TYPES

**Simple focal:** May include motor, sensory, autonomic, or psychic symptoms; no alteration of consciousness occurs; may be isolated or progress to complex focal or generalized seizures; associated with Todd's paralysis (see Tonic Clonic Seizures).

**Complex focal:** Most common type of seizure in epileptic adults; characterized by focal repetitive, purposeless, and complex movements (e.g., chewing, gesturing, lip smacking, and finger snapping) with alteration of consciousness; associated with auras that represent sensory and psychoillusory phenomena; most originate in the temporal lobe; typically last for <90 seconds and are associated with the postictal phase; amnesia specific to the event is common.

#### Posttraumatic Seizures

**Description:** Provoked seizures following traumatic brain injury (TBI); classified by timing of the seizure activity

**Immediate (concussive convulsions):** Occurs within the first 24 hours after TBI; about one half of seizures occur during the first 24 hours and one quarter during the first hour; controversial classification as they are not felt to represent a true seizure but rather a fairly benign phenomenon that occurs immediately after a concussion; usually do not require anticonvulsant therapy

**Early:** Occurs within 1 week of TBI; risk factors include young age, severity of injury, alcoholism, and intracerebral or subdural hematoma. Incidence is approximately 6%–10% but higher in patients with marked head injury. Seizure activity is usually tonic–clonic within the first 24 hours, progressing to more focal symptoms thereafter. A computed tomography (CT) scan of the head indicated considering the higher incidence of intracranial bleeding; not felt to represent epilepsy but often treated with prophylactic antiepileptic medication to minimize the risk of status epilepticus and secondary injury; treatment with antiepileptic medications does not affect the risk of posttraumatic epilepsy.

**Late:** Occurs over 1 week after TBI, with most occurring before 2 years after injury

Risk factors: early posttraumatic seizures, severity of injury, age > 65 years, alcoholism, brain contusion, and intracerebral or subdural hematoma. Overall incidence is approximately 2% but is strongly correlated with severity of TBI. Seizures are also associated with underlying brain pathology. They may recur in up to 70% of cases and often require long-term anticonvulsant medication.

### Precipitating Factors for Seizure

- Idiopathic, new-onset epilepsy, stress, sleep deprivation, fatigue, prenatal or perinatal brain injury, hyperthermia, metabolic (dehydration, hypoglycemia, hyponatremia, etc.), infectious (e.g., meningitis), trauma, drugs/alcohol (intoxication and withdrawal), febrile (usually occur between 6 months and 5 years of age), and intracranial lesions (mass, hematoma, etc.)

### Evaluation of Epileptic Athletes

- Before participation, the physician should be familiar with certain aspects of seizure history including seizure types (frequency, duration, and manifestations), precipitating factors,

postictal recovery, any history of status epilepticus, current anticonvulsant use, including side effects and medication adherence, and head trauma history.

### On-Field Treatment

- Standard guidelines for management of airway, breathing, and circulation should be followed.
- Assist the patient to the ground, and clear the area of any potential hazards.
  - Do not restrain the athlete.
  - Rolling the athlete to his or her side while he or she is convulsing may lead to injury; wait until the seizure is over before attempting this.
- Do not place anything in the athlete's mouth, particularly fingers. The mouth guard may be removed if it can be performed safely.
- If there is any concern for status epilepticus, activate EMS for transport to a medical facility.
- If this was the patient's first onset of a seizure or if clinical presentation is different from baseline seizure history, additional workup including imaging is indicated.

### Epilepsy and Sports

- Points to consider:
  - Does the specific type of seizure disorder place the athlete at an increased risk of injury (e.g., absence vs. simple focal)?
  - Will the athlete's condition place others at a risk of injury?
  - How intent is the athlete on playing the sport?
- Sports participation is associated with factors that may alter the seizure threshold.
  - Stress: Known risk factor for seizures, and this should be addressed on an individual basis
  - Hyperventilation: Resting hyperventilation may predispose to a decreased seizure threshold. Exercise-induced hyperventilation, however, is a physiologic response and does not seem to have a negative effect on the seizure threshold.
  - Alterations in drug metabolism: Studies have found no significant difference in metabolism during pre-exercise, exercise, and postexercise periods. However, if seizure activity increases, this should be considered.
  - Exercise-induced seizures:
    - Although there are certain cases of exercise lowering the seizure threshold, this seems to be the exception. Overall, the frequency of seizure is decreased by aerobic activity and is supported by electroencephalogram (EEG) findings. Aerobic exercise should be encouraged in epileptic patients.
    - Prolonged exercise, as seen in endurance athletes, may alter physiologic parameters associated with seizure activity (hyponatremia, hypoglycemia, etc.).
    - Consider diagnosis of seizures with episodes of exercise-induced syncope.
- People with epilepsy are less active, less physically fit, less likely to participate in sports, and are at a risk of social isolation.
- There is currently no evidence that contact sports are harmful to most athletes with epilepsy.
  - There is evidence to suggest that severe head injury causes or exacerbates preexisting epilepsy, but this does not apply to repeated mild TBI.
- Contact sports should be evaluated on an individual basis based on the presumed danger an athlete may present to themselves or others.

### Sport-Specific Guidelines

**Contact sports:** No restriction unless newly diagnosed or unclear course

**Water sports:** Generally permitted with appropriate precautions (avoid open water, wear flotation device, and supervised by qualified personnel); scuba diving, competitive underwater swimming, and diving prohibited with active epilepsy although may be considered after a prolonged seizure-free period (e.g., the UK Sport Diving Medical Committee suggests 5 years free of seizures without medication).

**Motor sports:** Discouraged with active epilepsy

**Aerobic sports:** No restrictions; wear appropriate protective gear

**Sports at heights:** Equestrian sports should be avoided with active epilepsy. Certain gymnastic events (e.g., high bar) should be discouraged. Skydiving, hang gliding, and free climbing should be discouraged. Pilot's license is prohibited.

**Shooting sports:** Specific consideration for type and frequency of seizures, pattern of occurrence, and type of weapon fired

## CERVICAL CORD NEURAPRAXIA (TRANSIENT QUADRIPLEGIA)

**Overview:** Transient neurologic episodes characterized by a temporary loss of motor or sensory function, or both. This is likely secondary to central cord contusion with or without associated ischemia. Most common with contact sports. Prevalence is estimated at 7 per 10,000 football participants.

**Mechanism of injury:** Varied mechanisms (usually hyperflexion, hyperextension, or axial loading of the neck) resulting in cervical cord contusion and decreased blood flow; suspected to be associated with spinal stenosis and the shape of the spinal canal. Degree of stenosis may be estimated using the Torg ratio (ratio of spinal canal width to vertebral body diameter). A ratio of <0.80 is considered indicative of "significant cervical stenosis"; this ratio has been shown to be an unreliable measure in larger athletes because of the relative vertebral body width yielding poor positive predictive value, and it has been replaced by magnetic resonance imaging (MRI) evaluation.

**Symptoms:** Bilateral sensory changes, motor changes, or combined sensorimotor deficits. Total body numbness may occur. Neck pain is usually absent.

**Physical examination:**

- **On the field**
  - Assume cervical spine injury until proven otherwise. Follow cervical spine precautions and use cervical collar and spine board.
  - Athlete should be stabilized and transported to the nearest appropriate facility by EMS for further evaluation.
  - For football, remove the face mask, but helmet and shoulder pads should remain in place. According to the most recent position statement by the American College of Sports Medicine (ACSM), it is strongly advised to not remove an American football helmet and shoulder pads from an unconscious athlete or an athlete who has sustained a neck injury. If any of the criteria below are met, the helmet and the shoulder pads should be removed maintaining cervical spine precautions:
    - If the helmet and chin strap do not securely hold the head, such that immobilization of the helmet does not also immobilize the head
    - If the design of the helmet and chin strap is such that even after removal of the face mask, the airway cannot be controlled or ventilation provided
    - If after a reasonable period of time, the face mask cannot be removed to gain access to the airway
    - If the helmet prevents immobilization for transportation in an appropriate position
    - Standard guidelines for management of airway, breathing, and circulation must be followed.

**TABLE 38.1 PERIPHERAL NERVE INJURY CLASSIFICATION**

| Seddon | Sunderland | Pathology | Electrodiagnostic Findings | Prognosis |
|---|---|---|---|---|
| Neuropraxia | I | Demyelination | Conduction block | Excellent, recovery typically 2–3 months |
| Axonotmesis | II | Demyelination and axon loss | Axon loss | Good, but limited by distance to muscle |
| Axonotmesis | III | II + endoneurium involvement | Axon loss | Fair, surgery may be required |
| Axonotmesis | IV | III + perineurium involvement | Axon loss | Poor, surgery usually required |
| Neurotmesis | V | IV + epineurium involvement | Axon loss | Poor, no spontaneous recovery and surgery required |

- **Off the field**
  - Thorough cervical spine and neurologic evaluation is necessary.
  - Pain and tenderness over the cervical spine is not associated with transient quadriplegia and should prompt consideration of other injuries such as fracture.

**Diagnostics:** Plain cervical spine trauma radiograph series should be obtained to rule out fracture or dislocation. Once a fracture is definitively ruled out, flexion and extension views may be obtained to check for ligamentous instability. CT to rule out occult facture. MRI should be obtained to evaluate spinal stenosis, spinal cord, and associated injuries.

**Treatment:** Initial treatment should focus on cervical spine immobilization, medical stabilization, and transport via EMS. The most recent Cochrane review recommends high-dose methylprednisolone within 8 hours of spinal cord injury because it has been shown to improve recovery of motor functions. However, controversy still exists, and additional research is needed. There is no role for steroids in athletes with transient quadriparesis who show resolution of symptoms. Systemic hypothermia, which is thought to decrease tissue metabolism and limit secondary hypoxic injury, has also been used but is still experimental and not part of routine care.

**Prognosis and return to play (RTP):** Temporary condition by definition, but both extent and duration of neurologic dysfunction factor in the overall prognosis; average rate of recurrence for players who return to football is 56%. Repeat episodes may be associated with cervical radiculopathy and myelopathy. Predisposition to permanent neurologic injury after an episode of cervical cord neurapraxia is controversial, but there are published case reports regarding the same; RTP decisions are made on an individual basis taking into account history of injuries, anatomical predisposition, and future risk of injury. Although discussing all possible scenarios is beyond the purpose of this text, we have enlisted a few examples here: If an athlete is asymptomatic without documented stenosis, it is generally accepted that he/she be allowed to RTP. If an athlete is asymptomatic with documented stenosis, significant controversy exists regarding RTP. Stenosis is more accurately defined by adequate cerebrospinal fluid around the cord as opposed to by absolute canal diameter. Opinions range from considering this as a relative contraindication to an absolute contraindication. Multiple episodes without documented stenosis should prompt serious consideration of discontinuing contact sports.

## PERIPHERAL NERVE INJURY IN ATHLETES

**Overview of anatomy:** The peripheral nervous system includes cranial nerves, spinal nerves and their roots and branches, peripheral nerves, and neuromuscular junctions.

**Classification of injury:** There are two primary classification systems: Seddon and Sunderland. Descriptions of each type, prognosis, and associated electrodiagnostic findings are compared in Table 38.1 (see also Fig. 38.1). Neuropraxia (grade I) is typically a demyelinating injury with good recovery. Axonotmesis (grades II–IV) involves axon disruption but with preservation of some degree of supporting structures (endoneurium, perineurium, and epineurium). The more supporting structures involved, the worse the outcomes. Neurotmesis (grade V) is complete disruption of both axons and supporting structures, with limited scope of recovery without surgical intervention.

**Mechanism of injury:** Injuries can be subcategorized into acute, subacute, or chronic. Acute injuries may result from nerve compression, stretch/traction, or laceration and usually result from an unexpected fall or extrinsic force (e.g., tackle). Stretch/traction injuries are most common; most nerves can tolerate 10%–20% lengthening before structural damage occurs. Subacute or chronic injuries are best classified as overuse injuries resulting from repetitive microtrauma or compression.

**Evaluation:** History should elicit a specific time course and mechanism of injury. A detailed physical examination is essential and includes muscle bulk inspection, strength testing, deep tendon reflexes, and sensory examination. Provocative maneuvers should be performed, including neural tension testing (i.e., straight leg raise) and direct nerve percussion (i.e., Tinel's sign). Equipment should be inspected for appropriate use and fit. In addition, a kinetic chain evaluation must be performed in search of a root cause for overuse injuries (i.e., a volleyball athlete with knee pain leading to shoulder overuse and subsequent suprascapular nerve injury).

**Diagnostics:**

- **Electrodiagnostic testing:** Used to assess the neurophysiologic function of the peripheral nervous system, including nerve roots, plexus, and peripheral nerves; testing includes nerve conduction studies (NCS) and needle electromyography (EMG). Used as an extension of physical examination and can help clarify or confirm a suspected diagnosis; more specifically, it can help to define location, duration, severity, and prognosis of neuromuscular disorders. NCS are performed by placing electrodes on the skin and stimulating the nerve of interest with electrical impulses. NCS are divided into motor and sensory nerve testing and help to determine if there is dysfunction of the myelin sheath (slowed electrical impulse), axons (reduced amplitude), or a combination of both. EMG involves a needle electrode that is inserted into a muscle of interest and is able to analyze motor units and their recruitment. Moreover, EMG can detect muscle denervation and whether or not any re-innervation has occurred.
- **MRI:** Useful as an adjunct in diagnosis and prognosis; may help with acute injuries in assessing soft tissue damage and deciding on surgical intervention. Normal peripheral nerves appear isointense compared with surrounding muscles, reveal a fascicular pattern, and are usually surrounded by a small rim of fat tissue. Nerve hyperintensity can be detected

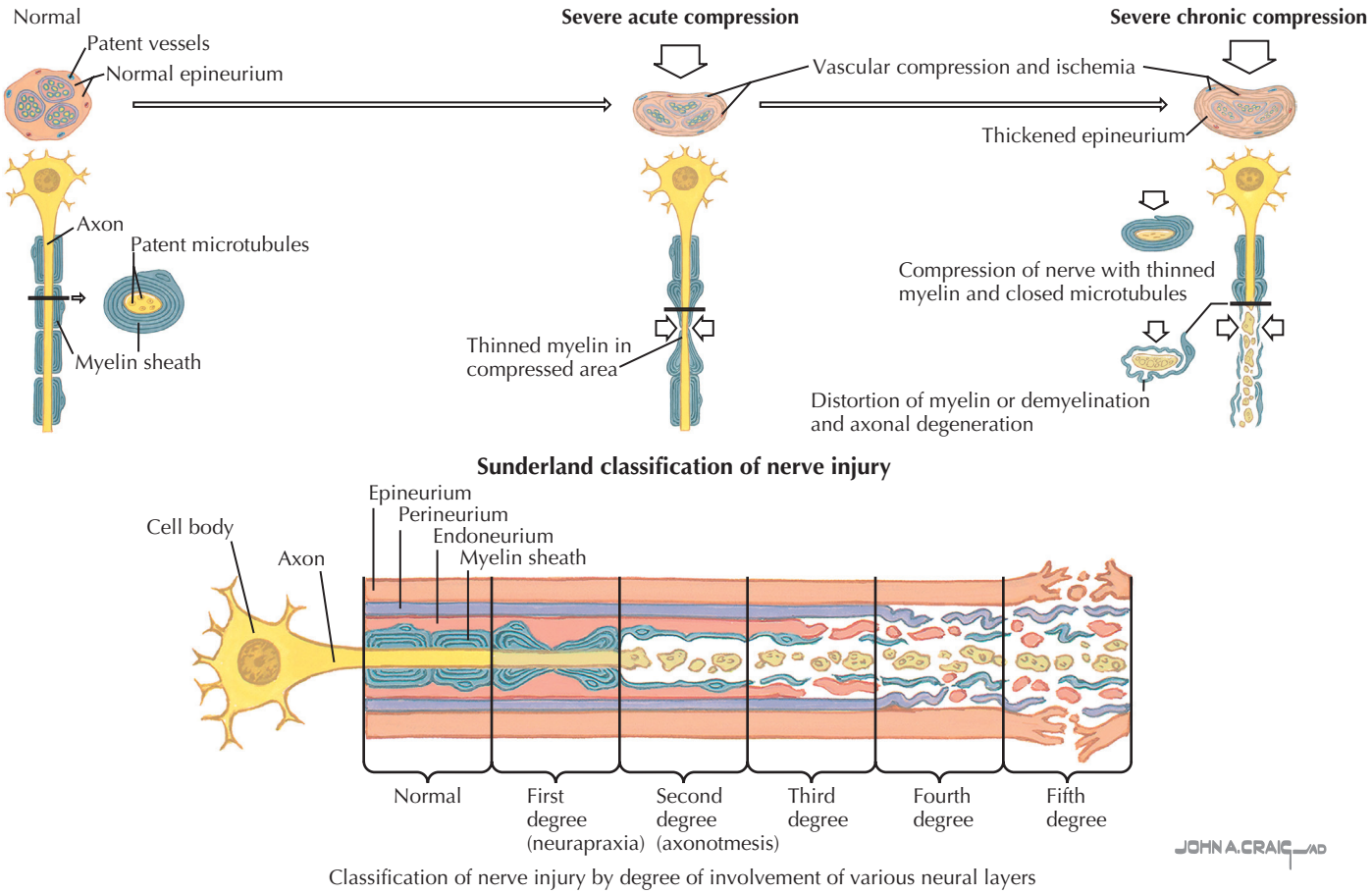



**Figure 38.1.** Peripheral nerve injury classification.

on T2-weighted MRI within 24 hours of a nerve injury. Focal demyelination reveals hyperintensity at the lesion site, whereas axonal injury with Wallerian degeneration reveals hyperintensity in the entire nerve segment distal to the lesion. Magnetic resonance neurography (MRN) refers to nerve-specific imaging using pulse sequences for optimal visualization. Axial T1-weighted and fluid-sensitive fat-suppressed T2-weighted images serve as the mainstay for interpreting peripheral nerve signal intensity, course, caliber, fascicular pattern, size, perineural fibrosis, and mass lesions. MRI can also identify masses that cause nerve compression.

- **Ultrasound:** Provides immediate, nonionizing, high-resolution, bedside assessment of peripheral nerves and surrounding tissues with ease of contralateral comparison; functional assessment is possible and can detect abnormal nerve motion and nerve entrapment with joint movement (subluxing ulnar nerve at the elbow). In addition, sonopalpation (transducer pressure) to the area of nerve dysfunction is useful for confirming typical symptoms. In long-axis view, peripheral nerves appear fascicular with hypoechoic fascicles surrounded by hyperechoic connective tissue. In short-axis view, peripheral nerves display a honeycomb or speckled appearance. With regard to nerve entrapment, ultrasound can demonstrate sudden flattening (notch sign) or gradual narrowing (dumbbell sign) at the compression site with proximal hypoechoic nerve swelling.

**Treatment:**

- **Surgical:** Open, sharp traumatic injuries should be immediately repaired through surgical intervention. For open, blunt injuries, delayed repair after 2–3 weeks is recommended to allow scar formation and demarcation of healthy nerve ends. Closed injuries are usually initially treated conservatively. Axonotmetic versus neurotmetic lesions should be differentiated using serial examinations and electrodiagnostic studies after 3 weeks. Surgical exploration using intraoperative electrophysiologic monitoring is suggested if no recovery is seen after 3–4 months. At that time, neurotmetic lesions are repaired and axonotmetic lesions are treated conservatively.
  - "**Rule of three":** Immediate surgery within 3 days for clean and sharp injuries; early surgery within 3 weeks for blunt injuries; delayed surgery at 3 months for closed injuries
- **Conservative management:** Includes a period of rest and avoidance of aggravating activities along with bracing and padding for protection of the injured region; a full kinetic chain evaluation should be performed to identify underlying faulty biomechanics predisposing to an overuse neuropathy. Physical therapy can aid in neuromuscular retraining and progressing back to appropriate sport-specific technique. Medications for symptomatic relief, including anti-inflammatory and neuromodulating agents (i.e., gabapentin), can be used for unpleasant dysesthesias.

**Prognosis and recovery:** Largely dependent on the type of nerve injury; most traumatic injuries are a combination of axonal and demyelinating pathologies, and prognosis depends on the predominant injury. Mechanisms of recovery include resolution of conduction block (failure of nerve propagation beyond the demyelinated segment), distal axonal sprouting, and axonal regeneration. Resolution of conduction block is typically the first mechanism to promote strength recovery. Recovery after

an ischemic injury is quick, whereas demyelinating injuries take up to several months. With an incomplete axonotmesis injury, distal sprouting of intact motor axons starts within 4 days after an injury. In minor axonotmesis injuries, axons can traverse the segment of injury in 8–15 days and then regenerate at a rate of 1–5 mm/day. The regeneration rate is faster for more proximal lesions and after a crush injury, whereas it is slower for distal lesions and after a laceration injury. Even if axons are unable to grow across the injury site, sprouting of motor axons can re-innervate about 5 times their original muscle fiber territory. Moreover, recovery depends on muscle integrity when the nerve reaches it. Muscle remains viable for re-innervation until about 18–24 months after an injury, following which fibrosis develops. Therefore, recovery, including surgical repair, is poor if denervation persists beyond 1–2 years.

**Optimal timing of electrodiagnostic testing:** NCS can help immediately localize a lesion as conduction is absent across the injury site and is normal distally until Wallerian degeneration is complete as early as day 7. EMG in the first week can help determine whether an injury is complete or incomplete. Presence of even a single motor unit firing on EMG signifies at least partial axonal continuity of a nerve. Using NCS to achieve stimulation above and below an injury site can differentiate between neuropraxia and more severe axonotmesis and neurotmesis as early as day 7 for motor and day 11 for sensory nerves owing to completion of Wallerian degeneration. However, the most useful diagnostic information can be obtained 3–4 weeks after an injury when needle EMG reveals denervation abnormalities. Findings of re-innervation are first seen proximally, progressing distally. Paraspinal re-innervation can be seen as early as 6–9 weeks after an injury, followed by proximal and then distal muscles between 2 and 6 months.

## BRACHIAL PLEXUS AND CERVICAL ROOT INJURIES ("STINGERS/BURNERS")

**Overview:** Transient neurologic injury accompanied by burning pain in the upper extremity believed to be the result of unilateral nerve traction or compression, generally involving C5 or C6 levels or the superior trunk of the brachial plexus (Fig. 38.2); unclear if this results from brachial plexus or cervical root injury. One of the most common injuries in sports medicine; up to 65% of college football athletes have reported such an injury in their career

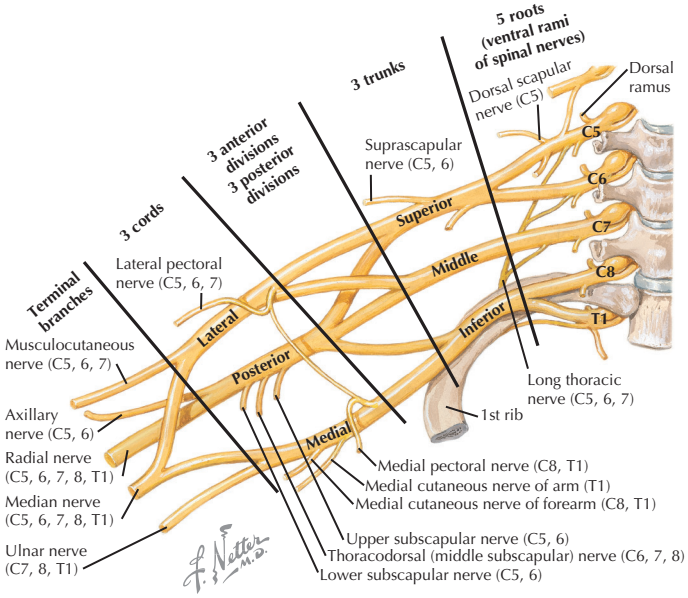



**Figure 38.2.** Brachial plexus schema.

**Proposed mechanisms of injury:** Neck extension with lateral bending, resulting in a compression injury; distraction of the shoulder from the head and neck causing nerve traction; or direct blow to the supraclavicular region at Erb's point (where the brachial plexus is most superficial), causing compressive injury

Cervical stenosis predisposes to stingers and may contribute to more complicated mechanisms.

**Presentation:** Symptoms include unilateral, transient burning pain or numbness with or without weakness starting from the neck/shoulder and commonly radiating in a C5 or C6 distribution. A circumferential rather than dermatomal pattern may be present. Symptoms in lower extremity or bilateral upper extremities should prompt consideration of central cord injury. Care should be taken to distinguish this condition from transient quadriplegia, which may present with bilateral sensory changes, motor changes, or combined sensorimotor deficits. Typically not associated with neck pain or limited range of motion (ROM); symptoms generally last from seconds to minutes; if symptoms last longer or if there is cervical pain or limited ROM, cervical spine MRI should be strongly considered before RTP. Up to 10% have a neurologic deficit lasting from hours to weeks.

**Physical examination:** Athletes frequently leave the field shaking their arm and hand and may elevate the arm to decrease the neural tension. Initial evaluation should focus on the cervical spine for any evidence of fracture (bony tenderness [particularly midline], deformity, or swelling). If initial evaluation is normal, assess ROM. Conduct a complete neurologic examination, including strength, sensory, and muscle stretch reflex testing. The time of onset and severity of weakness may vary. C5 and C6 are the most common levels affected. Most common sites of weakness are deltoid, biceps, supraspinatus, and infraspinatus, resulting in weak shoulder abduction, elbow flexion, and external rotation of upper arm. Percussion of Erb's point may elicit radiation of pain. A Spurling maneuver has been shown to recreate symptoms in 70% of cases—good specificity but variable sensitivity. Contralateral examination should be normal. If bilateral symptoms are present, full cervical precautions should be initiated, and the athlete must be transported to an appropriate medical facility for further evaluation and imaging. Shoulder examination must also be performed to rule out other injuries.

**Diagnostics:** Diagnosis based on physical examination. Radiographs usually normal; consider MRI for prolonged symptoms or recurrent stingers. EMG may be appropriate if symptoms persist beyond 3 weeks. The role of EMG in RTP decisions is controversial because EMG findings may remain abnormal long after clinical symptoms have resolved.

**Treatment:** Athletes should be removed from play until completely asymptomatic. Immediate supportive treatment includes rest and use of a sling. More chronic symptoms may necessitate anti-inflammatory medications, physical therapy, and occasionally, nerve root blocks. Prevention of recurrence is central to treatment. Rehabilitation should be instituted for neck and shoulder strengthening.

**Preventative equipment:** To limit excessive neck extension and lateral flexion, high-riding shoulder pads and neck rolls may be considered, but efficacy is debatable. Straps that connect the helmet and shoulder pads are not recommended. Evaluate tackling technique.

**Prognosis and RTP:** Decision is clinical. Athletes may return to full contact when they have pain-free, full ROM of the neck and shoulder, and normal neurologic examination, including normal symmetric strength. Longer rest periods are indicated for symptoms lasting >15 minutes and for recurrent stingers in

the same season. Continue to evaluate for delayed weakness during the same event and for the next 2 weeks. Repetitive or prolonged episodes warrant additional diagnostic testing. Chronic and recurrent stingers have been associated with cervical disk disease, spinal stenosis, and/or neural foraminal narrowing. Prolonged time to symptom resolution (particularly >3 weeks) and recurrent stingers over a short period of time have poorer prognosis. Some experts advocate that if cervical foraminal stenosis is discovered, the athlete should discontinue contact sports.

## AXILLARY NERVE INJURY

**Overview:** The axillary nerve is derived from the posterior cord of the brachial plexus at the C5 or C6 level with occasional contribution from C4. It travels across the anteroinferior aspect of the subscapularis muscle before entering the quadrilateral space and dividing into two branches. The anterior branch passes around the surgical neck of the humerus, and the posterior branch travels adjacent to the inferior aspect of the glenoid rim before dividing into the upper lateral brachial cutaneous nerve and the nerve to the teres minor (Fig. 38.3). The quadrilateral space is bordered by the long head of the triceps medially, the humerus laterally, teres minor superiorly, teres major and latissimus dorsi muscles inferiorly, and the subscapularis anteriorly. Branches:

- Anterior branch: Motor innervation of the deltoid
- Posterior branch: Motor innervation of the teres minor
  - Superior lateral cutaneous nerve: sensory innervation over the inferior portion of the deltoid (upper and lateral arm)

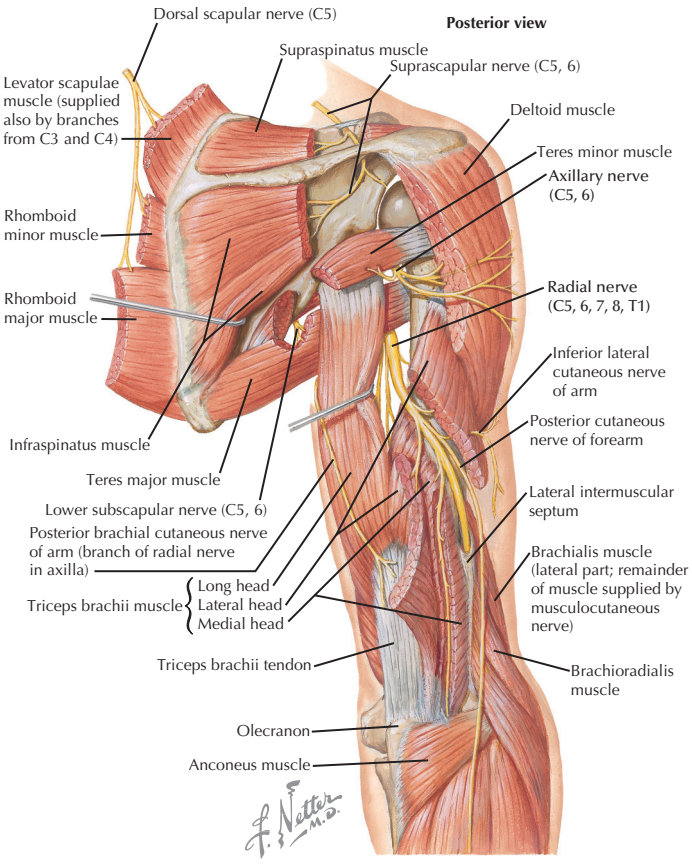



**Figure 38.3.** Scapular, axillary, and radial nerves.

- Uncommon nerve injury, representing <1% of all nerve injuries

**Common sports for injury:** Baseball, football, hiking, hockey, martial arts, rugby, volleyball, and wrestling

**Mechanism of injury:** Most commonly incurred with other brachial plexus injuries

- **Traumatic:** Direct (e.g., contusion to anterolateral deltoid or humeral fracture) or indirect (e.g., shoulder traction with dislocation)
- **Compression:** Quadrilateral space syndrome; this is a rare condition characterized by compression of the posterior humeral circumflex artery and axillary nerve as they pass though the quadrilateral space. Compression is believed to be caused by fibrotic bands within the space or by hypertrophy of the muscles that form the borders, but the exact etiology is unclear.
- **Iatrogenic** (e.g., rotator cuff surgery)

**Symptoms:** Variable

The athlete may not complain of frank weakness. Possible symptoms include early fatigue or weakness, particularly with overhead activities or abduction. The athlete may notice numbness in the lateral upper arm region. Night pain is frequently reported.

**Differential diagnosis:** Cervical pathology (typically C5/C6), rotator cuff injury, vascular compression, and suprascapular nerve injury

**Physical examination:** Visual inspection may reveal deltoid or teres minor atrophy with late presentation. Examine the neck, shoulder, and arm. ROM and strength in all planes should be tested. Palpate the deltoid for contraction during the initiation of abduction. Be sure to evaluate all three heads of the deltoid because nerve injury may not be uniform throughout. Weakness may occur with external rotation, abduction, and/or forward flexion. Evaluate sensation in the upper lateral arm region.

**Diagnostics:** Radiographs should be obtained to check for fracture of the humerus. Cervical spine films assess bony cervical pathology. MRI and electrodiagnostics should be considered.

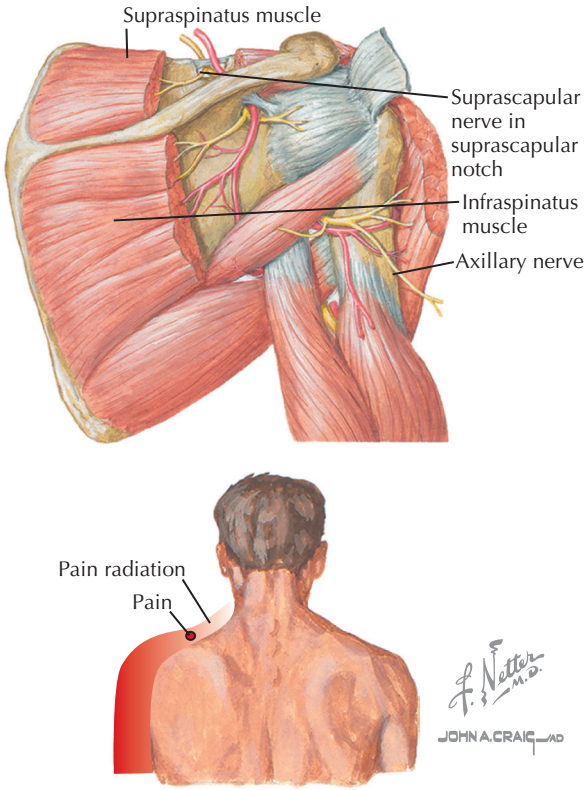
**Treatment:** Depends on the underlying etiology and injury to surrounding structures

Address underlying shoulder dislocation and/or fracture. Conservative treatment includes rest and physical therapy for active and passive ROM and strengthening. Electrical stimulation of the deltoid is an optional treatment.

**Prognosis and RTP:** Full recovery with nonoperative treatment is expected in 85%–100% of cases within 12 months if they are associated with dislocation or fracture. Various case series describing injuries from direct contusion have shown poorer prognosis with respect to recovery of deltoid function, but a substantial proportion of these patients returned to contact sports despite deltoid paralysis. RTP decisions depend on the associated injuries involved, but full shoulder ROM and good strength are recommended.

## SUPRASCAPULAR NERVE INJURY

**Overview:** The suprascapular nerve originates from the superior trunk of the brachial plexus (C5 and C6 with variable contribution from C4). It travels laterally across the posterior triangle of the neck through the suprascapular notch and under the superior transverse scapular ligament before sending off branches to the supraspinatus. The nerve then travels around the spinoglenoid notch to enter the infraspinatus fossa, where it divides to supply the infraspinatus (Fig. 38.4) and innervates the supraspinatus and infraspinatus with sensory branches to the acromioclavicular and glenohumeral joints. Sensory innervation of the proximal–lateral arm is reported in 15% of patients. The nerve is the most frequently injured peripheral branch of the brachial plexus in athletes.




Compression of suprascapular nerve may cause lateral shoulder pain and atrophy of supraspinatus and infraspinatus muscles.

**Figure 38.4.** Neuropathy around the shoulder: suprascapular nerve.

**Common sports for injury:** Volleyball, baseball, basketball, tennis/racquetball, football, weightlifting, hiking, wrestling, cycling, and ballet dancing

**Mechanism of injury:**

- **Trauma:** Direct (e.g., scapular or clavicular fracture, shoulder dislocations, and penetrating trauma) and indirect (e.g., traction or repetitive overuse)
- **Compression:** Usually via surrounding ligaments or mass lesions (e.g., cyst, lipoma, and fibrous band); overhead activities such as throwing lead to greater degrees of friction, traction, and compression; external rotation, cross-body adduction, and forward flexion are also implicated; proposed "sling effect" refers to friction of the nerve against surrounding structures of the suprascapular notch with depression and retraction or hyperabduction of the shoulder.
- **Iatrogenic**

**Symptoms:** Include dull pain at the posterior aspect of the shoulder exacerbated by overhead maneuvers and/or weakness of the affected shoulder, particularly during rotation and abduction. Atrophy present in up to 80% of patients (see Fig. 38.4); onset is usually insidious but may follow an acute event. Lesions at the spinoglenoid notch often present with asymptomatic atrophy of the infraspinatus alone, whereas lesions at the suprascapular notch often reveal atrophy of both infraspinatus and supraspinatus.

**Differential diagnosis:** Cervical spine pathology, brachial plexopathy, biceps tendonitis, adhesive capsulitis, impingement syndrome, rotator cuff, and intra-articular glenohumeral pathology

**Physical examination:** Visual inspection may reveal atrophy of the infraspinatus or supraspinatus. Atrophy of the infraspinatus is better visualized from behind and above, but supraspinatus atrophy may be difficult to visualize. Physical examination may reveal tenderness at the injury site, and cross-arm adduction may increase pain. Physical examination findings are similar to those of impingement syndrome. Include thorough neurologic evaluation, including neck and upper extremity, specifically during external rotation and abduction.

**Diagnostics:** Often considered a diagnosis of exclusion; may try injection of a local anesthetic into the suprascapular notch for diagnostic purposes, but this is nonspecific

An anteroposterior radiograph directed caudally at 30° or a Stryker notch view may be useful to assess the shape of the suprascapular notch. MRI is considered the optimal imaging modality for evaluating suprascapular nerve palsy because it should reveal both mass lesions and other rotator cuff or soft tissue pathology. Ganglion cysts causing suprascapular nerve compression are often associated with labral tears. Ultrasound may also be useful but is operator dependent. Electrodiagnostics may help localize the site of injury and rule out a C5 or C6 radiculopathy.

**Treatment:** Treatment and prognosis depend on the underlying etiology. Initial treatment is conservative and includes avoidance of aggravating activities, anti-inflammatory medications, and rehabilitation for stretching and strengthening of surrounding structures. Structural lesions (e.g., labral pathology) correlating with clinical symptoms should be surgically treated. Nonoperative treatment of ganglion cysts has a high failure rate, and cysts are frequently drained surgically. Occasionally, exploration and decompression of compressive lesions or bands may be required. If symptoms are not caused by known structural lesions or if atrophy continues to persist for >6 months, surgical exploration should be considered. Follow-up EMG may aid in the decision to proceed with surgical therapy.

**Prognosis and RTP:** Natural history of the injury varies greatly, but resolution of symptoms in the absence of a mass lesion is expected within 6–12 months of the diagnosis. The patient may continue his or her chosen sport at a competitive level despite muscle atrophy if appropriate strength permits safe participation. RTP decisions are individual and activity specific.

## LONG THORACIC NERVE INJURY

**Overview:** The long thoracic nerve arises from the anterior branches of C5 through C7, with 20% contributions from intercostal nerves. At 22–24 cm in length, it descends posteriorly to C8 and T1 rami, passes beneath the clavicle, and continues distally on the external surface of the serratus anterior (Fig. 38.5). This nerve innervates the serratus anterior, which protracts and rotates the scapula as well as stabilizes the scapula during abduction.

**Common sports for injury:** Tennis/racquetball, backpacking, archery, basketball, bodybuilding, football, golf, gymnastics, martial arts, hiking, shooting, volleyball, and wrestling

**Mechanism of injury:**

- **Traumatic:** Direct (e.g., contusion to shoulder or lateral thorax) and indirect (e.g., stretching of the nerve can occur with the neck turned to the contralateral direction and the arm raised overhead or shoulder depression in conjunction with contralateral neck bending)
- **Compression**: Multiple possible areas for compression by surrounding structures, crutches, or inflamed bursa
- **Iatrogenic** (e.g., postoperative)

**Symptoms:** Typical winging or popping of the scapula during movement secondary to compromised glenohumeral biomechanics; others may complain of pain in the shoulder, neck, and/or scapular area lasting up to a few weeks, followed by insidious weakness with overhead activities or forward elevation

**Differential diagnosis:** Cervical disc disease, rotator cuff pathology, brachial neuritis (Parsonage–Turner syndrome), adhesive

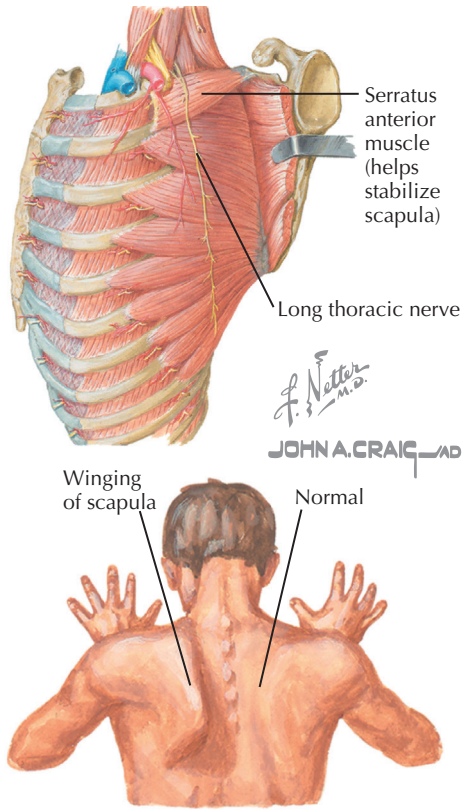



**Figure 38.5.** Neuropathy around the shoulder: long thoracic nerve.

capsulitis, glenohumeral instability or arthritis, acromioclavicular joint arthritis, thoracic outlet syndrome, and spinal accessory nerve injury

**Physical examination:** Visual inspection will reveal winging of the affected scapula exaggerated by forward elevation or pushing off a wall (see Fig. 38.5). Atrophy of the serratus anterior may be visible. Winging is associated with both long thoracic and spinal accessory nerve injuries. Winging associated with spinal accessory nerve injury is generally not exaggerated by forward elevation as is seen with long thoracic etiology. Physical examination reveals weakness with forward elevation, often limiting movement to 110°. Altered scapulohumeral rhythm may be found.

**Diagnostics:** Considered a clinical diagnosis; plain radiographs are generally normal; CT and MRI generally not useful unless used to rule out other pathologies; electrodiagnostics help distinguish from other causes of scapular winging (dorsal scapular and spinal accessory nerve injury)

**Treatment:** Conservative treatment includes rest, reassurance, anti-inflammatory medications or medications for neurogenic pain, maintaining ROM, and strengthening of surrounding structures. Bracing may be considered for improved function or pain control but has not been highly effective. Indications for surgical consultation include symptoms persisting beyond 1 year with no improvement on EMG and iatrogenic injury.

**Prognosis and RTP:** Isolated atraumatic long thoracic nerve palsy generally resolves in 1–2 years. RTP decisions should be individualized and based on the athlete's strength and the demands of the sport.

## SPINAL ACCESSORY NERVE INJURY

**Overview:** The spinal accessory nerve is a pure motor cranial nerve. It enters the foramen magnum and then exits the jugular foramen before passing through the upper third of the sternocleidomastoid (SCM). The nerve then assumes a subcutaneous course in the posterior cervical triangle to the trapezius. This nerve provides motor innervation to the trapezius and SCM.

**Common sports for injury:** Hockey, backpacking, lacrosse, wrestling, and martial arts

**Mechanism of injury:**

- **Traumatic**: Direct (e.g., penetrating or blunt trauma to the posterior neck) or indirect (e.g., traction)
- **Compression:** Mass lesions or external (e.g., backpack)
- **Iatrogenic:** Subject to insult during lymph node dissection, carotid endartectomy, etc.

**Symptoms:** Pain around the shoulder, weakness, difficulty with abduction and overhead activities, and sagging of the shoulder; radicular pain caused by traction from the drooping shoulder may also occur

**Differential diagnosis:** Long thoracic nerve injury or cervical pathology

**Physical examination:** Visual inspection may reveal asymmetry of the neckline, drooping of the affected shoulder, loss of normal scapulohumeral rhythm, and "lateral" scapular winging. Atrophy of the trapezius and SCM with associated spasm of the levator scapulae and rhomboids may be present. Physical examination may reveal weakness with abduction and forward elevation and inability to shrug the affected shoulder. Winging of the scapula may occur but is generally not as severe as that seen with long thoracic nerve injury. Winging associated with spinal accessory nerve injury is not exaggerated by forward elevation as is seen with long thoracic nerve injury. Scapular stabilization against the back by the physician may relieve symptoms.

**Diagnostics:** Radiographs of the cervical spine, chest, and shoulder are indicated but are usually negative. MRI is useful only for ruling out mass lesions. Electrodiagnostics are usually warranted.

**Treatment:** Treatment and prognosis depend on the underlying etiology. Conservative treatment comprises anti-inflammatory drugs, electrical stimulation, limitation of overhead activities, and physical therapy focusing on shoulder girdle and scapular rehabilitation. A sling may be used as needed but be wary of frozen shoulder. Braces to stabilize the scapula have not been highly effective. Indications for surgical consultation include symptoms and persistent atrophy for >6 months, penetrating trauma, and iatrogenic injury.

**Prognosis and RTP:** Injuries caused by blunt trauma typically recover in <1 year with conservative treatment. With injury caused by penetrating trauma or laceration, prognosis is better if surgical intervention is not delayed for >6 months. RTP decisions are individual and activity specific.

## RADIAL NERVE INJURY

**Overview:** The radial nerve originates from the posterior cord of the brachial plexus (C5–T1); it runs with the deep artery before passing into the cubital fossa and descending between the brachioradialis and brachialis. At the level of the lateral epicondyle, it branches into the superficial and deep branches (Fig. 38.6). The deep branch courses around the neck of the radius and enters the posterior compartment, terminating as the posterior interosseous, which continues under the supinator in the radial tunnel. The superficial branch passes anterior to the pronator teres, eventually entering the dorsum of the hand. Branches:

- **Forearm branches:** Motor innervation of triceps brachii, anconeus, brachioradialis, and extensor carpi radialis longus
- **Posterior interosseous nerve (PIN):** Motor innervation of the extensor carpi radialis brevis, extensor carpi ulnaris, extensor digiti minimi, extensor digitorum, supinator,

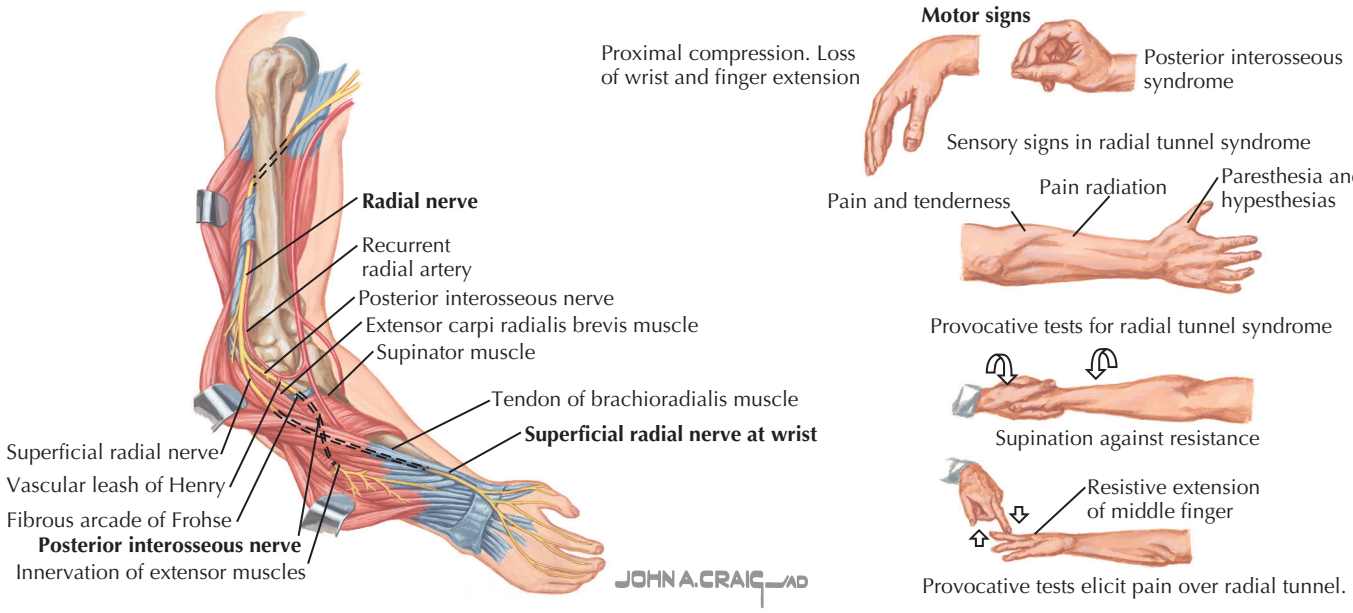



**Figure 38.6.** Radial nerve in forearm.

extensor indicis proprius, abductor pollicis longus, and extensor pollicis longus and brevis
- **Posterior cutaneous nerve:** sensory innervation of posterior arm and forearm
- **Inferior lateral cutaneous nerve:** sensory innervation of lateral arm
- **Superficial branch:** sensory innervation of proximal dorsal three and a half digits and dorsal hand

**Common sports for injury:** Arm wrestling, baseball, football, tennis/racquetball, weightlifting, frisbee/discus throw, rowing, and gymnastics

**Mechanism of injury:**
- **Traumatic**: Direct (e.g., humeral shaft fracture) or indirect (e.g., muscle strain)
- **Compression**: Multiple possible sites of compression; repetitive pronation/supination increases compressive forces. More proximal lesions may occur with tourniquet use, inappropriate use of axillary crutches, and "Saturday night compression palsy."
  - Radial tunnel syndrome (RTS)—compression at the elbow
  - Wartenberg syndrome—caused by direct trauma to or compression of the superficial branch in the forearm

**Symptoms:** Symptoms around the elbow may mimic lateral epicondylitis; of the patients with lateral epicondylitis, 5%–10% have associated radial nerve entrapment.
- RTS: Deep ache distal to the lateral epicondyle that is worse at night and after throwing; generally not associated with sensory loss or weakness. Syndrome is controversial; may occur with racquet sports because of repetitive supination/pronation.
  - The radial tunnel is the area between the lateral epicondyle and the supinator muscle, which exists as a potential space with numerous sites of compression.
- PIN compression syndrome: The same structures are compressed as with RTS, but symptoms are predominantly weakness of the purely motor PIN-supplied muscles after a prodrome of lateral forearm or elbow pain. Throwers may note decreased control and velocity.
- Wartenberg syndrome: May be caused by any tight-fitting strap or band around the wrist; symptoms include pain and decreased sensation over the dorsoradial hand, dorsal thumb, and index finger; not associated with throwing

**Differential diagnosis:** Varies depending on symptoms

**Physical examination:** Complete neurologic examination of the upper extremities is warranted. Specific tests (see Fig. 38.6):
- RTS: Resisted extension of the middle finger with the elbow extended, forearm pronation with the wrist flexed, elbow flexion and supination, or supination with the elbow extended may elicit pain. Certain tests will be positive with lateral epicondylitis. Certain studies support the use of diagnostic blocks.
- PIN: Same as above but with weakness on extension of the wrist, thumb, and index finger; forearm supination and grip is also weak. Symptoms may be reproduced by compression of three fingerbreadths below the lateral epicondyle and can also worsen if misdiagnosed as lateral epicondylitis with application of a counterforce brace over the site of entrapment.
- Wartenberg syndrome: Tinel's sign should be positive; not associated with weakness. Pseudo–Finkelstein test can be positive with forced pronation and ulnar flexion of the wrist.
- Check for cervical radiculopathy and thoracic outlet syndrome.

**Diagnostics:** Plain radiographs are generally negative in the absence of trauma but may reveal calcifications. MRI is helpful only if a mass lesion is suspected. Electrodiagnostics are usually not helpful with RTS but may be diagnostic in PIN. Negative test does not rule out entrapment; consider testing if associated with trauma.

**Treatment:**
- RTS: Standard conservative treatment includes rest (may be assisted with splinting), avoidance of provocative movements, anti-inflammatory medications, physical therapy, and pain management.
- PIN: All of the above for RTS plus splinting to maintain function
- Wartenberg syndrome: Standard conservative treatment includes rest (may be assisted with splinting in supination) and anti-inflammatory medications.
- Surgical intervention for RTS is rare.

**Prognosis and RTP:** Generally responds to conservative treatment; limited case series show a good prognosis with surgical decompression.

## MEDIAN NERVE INJURY

**Overview:** The median nerve arises from two cords of the brachial plexus, lateral (C6 and C7) and medial (C8 and T1), with occasional input from C5. It descends on the medial arm and enters the cubital fossa before passing between the heads of the pronator teres, where it gives off the anterior interosseous branch. It then descends between the flexor digitorum superficialis (FDS) and the flexor digitorum profundus (FDP), giving off the palmer cutaneous branch before passing within the carpal tunnel to reach the hand (Fig. 38.7). Branches:

- Forearm branches: motor innervation of the pronator teres, flexor carpi radialis, and palmaris longus
- Anterior interosseous nerve: motor innervation of the FDP (radial half), flexor pollicis longus (FPL), pronator quadratus
- Terminal motor branches: motor innervation of the FDS, abductor pollicis brevis, opponens pollicis, flexor pollicis brevis, lateral two lumbricals
- Palmer cutaneous nerve: sensory innervation of lateral palm
- Digital cutaneous branches: sensory innervation of volar and distal dorsal surfaces of the radial three and a half digits

**Common sports for injury:** Wrist: archery, basketball (wheelchair), bicycling, weightlifting, football, golf, and wrestling; palmer branch: cheerleading and golf; and pronator teres: archery and baseball

**Mechanism of injury:**

- **Traumatic:** Direct or indirect
- **Compression:** Most commonly within the carpal tunnel (carpal tunnel syndrome [CPS]) but occurs at multiple sites, including within the pronator teres (pronator syndrome [PS]) and along the anterior interosseous nerve (anterior interosseous syndrome [AIS]) (Fig. 38.8)

**Symptoms:**

- **CTS:** Paresthesias and weakness in the radial three and a half digits of the hand; increased symptoms with repetitive movements; pain may radiate proximally; nighttime symptoms are common
- **PS:** Pain in the proximal volar surface of the forearm often radiating distally, which generally increases with activity; often described as fatigue; more distal sensory symptoms may occur but are uncommon; not typically associated with nighttime symptoms
- **AIS**: Deep constant pain in the proximal volar forearm preceding gradual weakness of the FPL and FDP; no sensory loss noted

**Differential diagnosis:** Varies depending on symptoms

**Physical examination:** Thenar muscle wasting may be present in advanced cases, usually with CTS. Thorough neurologic examination including two-point discrimination should be performed. Specific tests:

- CTS: Tinel's sign (tapping over the median nerve at the wrist reproduces index symptoms) and Phalen's sign (wrist flexion for 60 seconds produces paresthesias in a median nerve distribution) are typical
- PS: Pronator compression test wherein compression over the pronator teres reproduces symptoms; tenderness over the pronator is also common. Tinel's sign over the cubital fossa and reproduction of symptoms with resisted pronation in the extended elbow support the diagnosis; may cause numbness in the thenar eminence in the distribution of

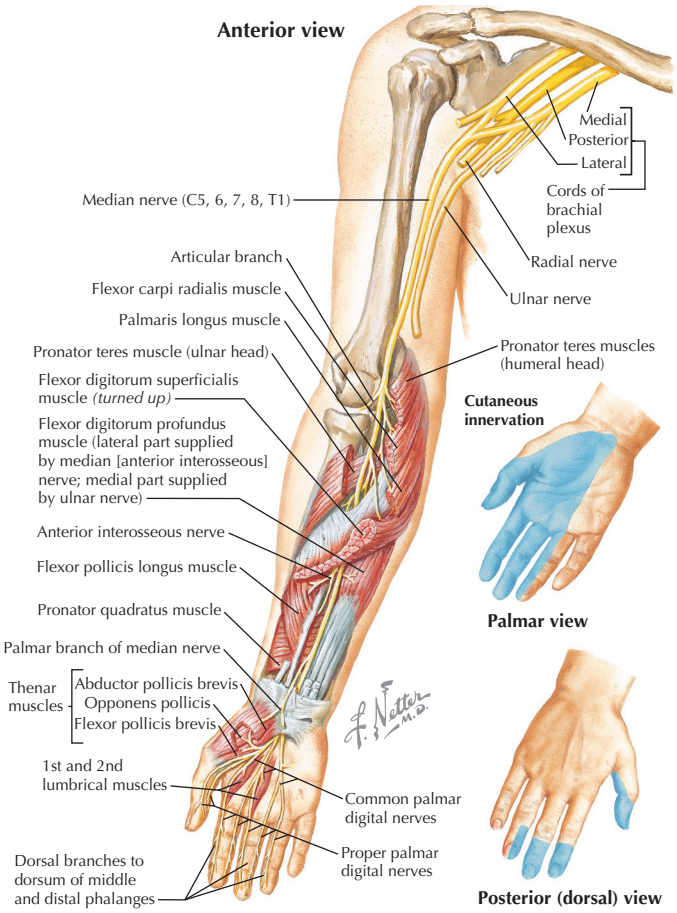



**Figure 38.7.** Median nerve.

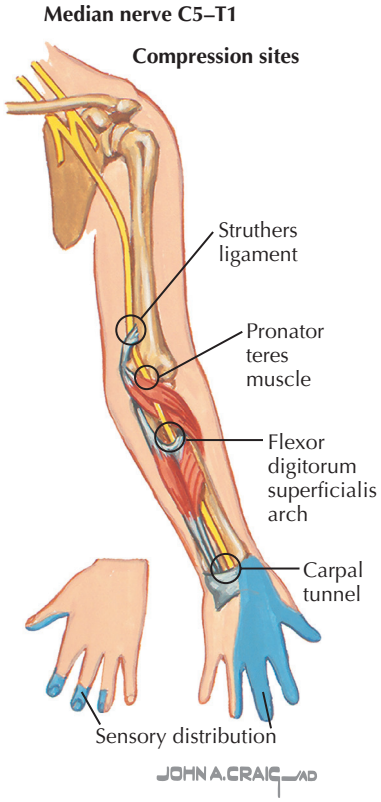



**Figure 38.8.** Common sites of upper extremity nerve entrapment.

the palmar cutaneous branch of the median nerve (spared with CTS)
- AIS: Weakness of the FDP and FPL, manifesting as inability to make an appropriate circle with the index finger and thumb
- Check for cervical radiculopathy and thoracic outlet syndrome.

**Diagnostics:** Plain radiographs may reveal osteophytes or bony abnormalities causing compression. MRI should be obtained if compressive masses are suspected. Electrodiagnostic studies are warranted with CTS and AIS but are unreliable with PS. Comparison studies on sensory nerve conduction in the hand using the Combined Sensory Index (CSI) improves the sensitivity of mild CTS detection. However, negative studies do not rule out CTS.

**Treatment:**
- CTS: Conservative treatment with splinting the wrist in neutral position including at night, rest, physical therapy, and anti-inflammatory medications. Corticosteroid injections into the carpal tunnel may relieve symptoms.
- PS: Rest, anti-inflammatory medications, and immobilization with elbow flexion at 90° and the forearm in neutral position for 3–6 weeks. Use of injectable corticosteroids is debatable.
- AIS: Same as PS
- Indications for surgical consultation include failed conservative management and progressive symptoms. Specific time periods for trial of conservative treatment are 6 months for CTS and 3–6 months for PS and AIS, but these vary according to available literature.

**Prognosis and RTP:**
- **CTS**: Prognosis is variable but generally responds well to conservative treatment within a few months with very good prognosis is expected if surgical treatment required
- **PS**: 50% resolution in 6–8 weeks with conservative treatment; very good prognosis if surgical decompression required
- **AIS:** Frequently resolves with conservative management; good prognosis if surgical intervention required

## ULNAR NERVE INJURY

**Overview:** The ulnar nerve arises from the C8 and T1 roots. Contribution from C7 is not uncommon. It descends distally, just medial to the axillary artery. After passing posterior to the medial epicondyle of the humerus within the cubital tunnel, it enters the anterior compartment of the forearm between the heads of the flexor carpi ulnaris (FCU). Descending between the FCU and FDP, the nerve gives off the palmar cutaneous branch and then the dorsal cutaneous nerve. After passing through Guyon's canal, the nerve splits into superficial sensory and deep motor branches (Fig. 38.9). The nerve may move as much as 7 mm anteromedially and lengthen as much as 4.7 mm during flexion. Branches:
- Forearm branches: motor innervation of FCU and FDP (ulnar half)
- Superficial motor branch: motor innervation of the palmaris brevis
- Deep motor branch: motor innervation of the hypothenar muscles, adductor pollicis, all interossei, medial two lumbricals, and the deep head of flexor pollicis brevis
- Dorsal and palmar sensory branch: sensory innervation of medial palm/dorsal hand and volar and distal dorsal surfaces of ulnar one and a half digits

**Common sports for injury:**

**Elbow:** baseball; tennis; bicycling; bodybuilding/weightlifting; judo, karate, and kickboxing; cross-country skiing; and wrestling

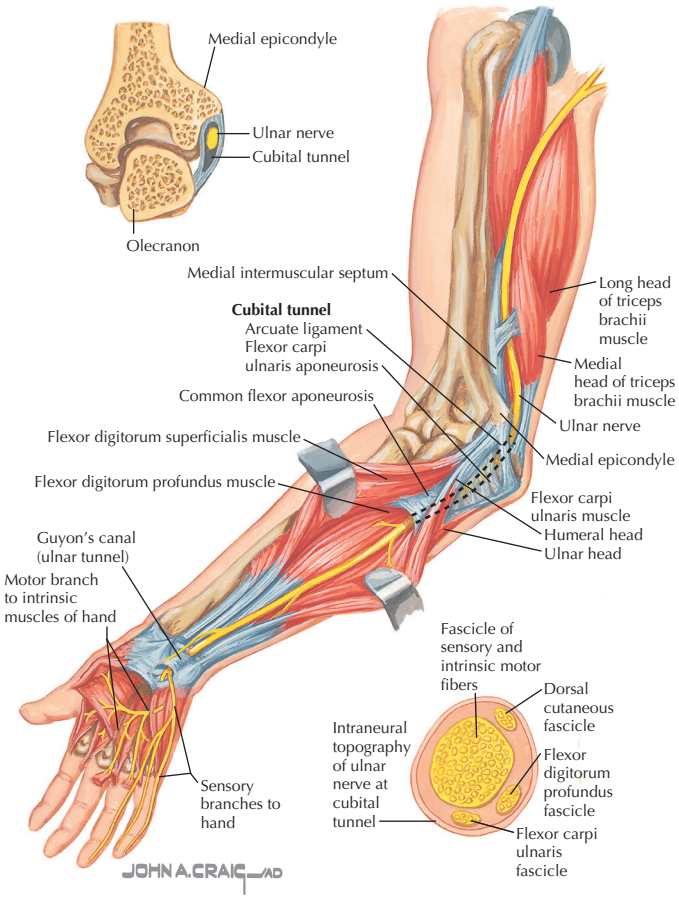



**Figure 38.9.** Compression of ulnar nerve.

**Wrist:** basketball (wheelchair), bicycling, football, cross-country skiing, and snowmobiling

**Flexor carpi ulnaris:** weightlifting and golf

**Deep motor branch:** weightlifting

**Mechanism of injury:**
- **Trauma:** Direct and indirect trauma (i.e., traction and friction)
- **Compression:** Common sites for compression at the elbow include the ulnar groove, below the medial epicondyle (also known as the *cubital tunnel*), and above the medial epicondyle (also known as the "arcade of Struthers"). Compression at the wrist usually occurs at Guyon's canal (see Fig. 38.9); may also occur with muscular hypertrophy of the FCU, ulnar artery aneurysm, lipoma, etc.

**Symptoms:**
- **Cubital tunnel syndrome:** Common in baseball pitchers because of the large valgus stress at the elbow and repetitive flexion/extension (valgus overload syndrome); pain may occur at the medial joint line or paresthesias during late cocking or early acceleration; radiation to the hand is common. Snapping of the nerve may occur with flexion/extension. Symptoms may also be focused more distally, mimicking ulnar tunnel syndrome. Sleeping with elbows fully flexed can aggravate the symptoms.
- **Ulnar tunnel syndrome (Guyon's canal syndrome):** Also known as handlebar or cyclist's palsy because of repetitive loads on the palmar aspect of the wrist during cycling; symptoms include paresthesias and pain in the fourth and fifth digits, decreased grip strength (40% of the grip strength is derived from ulnar nerve musculature), and pain at the

volar wrist ulnar aspect; fourth and fifth digit abduction and adduction weakness imply poorer prognosis.

**Differential diagnosis:** C8 and/or T1 radiculopathy, brachial plexopathy, and polyneuropathy; other causes of wrist pain include hypothenar hammer syndrome, fracture of the hook of the hamate, ganglion cysts, ulnar carpal instability, and ulnar artery aneurysm

**Physical examination:** Visual inspection for flexion contractures or valgus deformity at the elbow, muscle hypertrophy or atrophy, and claw hand deformity; palpate for a subluxing ulnar nerve as the elbow is flexed. Stress the ulnar collateral ligament (UCL) for laxity; thorough neurologic examination including intrinsic muscles of the hand. Dorsal symptoms rule out the possibility of ulnar nerve entrapment at the wrist. Specific tests:

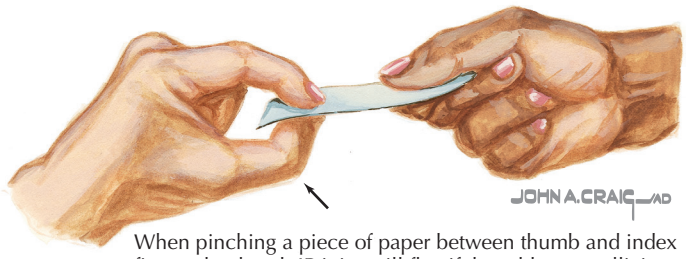
- Elbow flexion test: holding elbow in maximal flexion with full wrist extension for one minute to test for symptom reproduction
- Tinel's sign: palpation of the ulnar nerve reproducing symptoms at the cubital tunnel (particularly combined with elbow flexion) or Guyon's canal
- Phalen's sign: wrist flexion eliciting paresthesias in an ulnar distribution
- Froment's sign: weakness of adduction of the thumb (Fig. 38.10)
- Evaluate for cervical radiculopathy and thoracic outlet syndrome

**Diagnostics:** Choice of imaging is based on location of the lesion. Plain radiographs may reveal bony changes or osteophytes around the elbow (ulnar sulcus or cubital tunnel view) or hamate fracture at the wrist (carpal tunnel view). MRI studies and angiograms may reveal occult hamate fracture and/or ulnar artery thrombosis. Electrodiagnostics help in localization of the lesion.

**Treatment:** Treatment and prognosis depend on the underlying etiology and injury to surrounding structures.

- Cubital tunnel syndrome: Rest, ice, anti-inflammatory medications, and physical therapy; splinting in mild flexion (elbow pad worn in "reverse" or fiberglass volar flexion "block" splint) or use of an elbow pad to avoid pressure on the cubital tunnel may help.
- Ulnar tunnel syndrome: Similar to cubital tunnel syndrome treatment, plus wrist splinting in functional position with slight dorsiflexion; corticosteroid injection into Guyon's canal may be considered but often yields only transient relief.
  - In cases of cyclist's palsy, use of padded gloves, specialized grips, altering and frequently changing hand position, and appropriate bicycle fitting may decrease symptoms.
- Indications for surgical consultation include failure to respond to conservative treatment, persistent motor weakness, intrinsic paralysis, or progression of symptoms. Surgical intervention for cubital tunnel syndrome frequently includes decompression of the nerve and/or anterior transposition for neuropathy associated with elbow deformity or subluxation of the nerve.

**Prognosis and RTP:** RTP decisions depend on the associated injuries involved and treatment required.




When pinching a piece of paper between thumb and index finger, the thumb IP joint will flex if the adductor pollicis muscle is weak (ulnar nerve paralysis).

**Figure 38.10.** Positive Froment's sign.

## TIBIAL NERVE INJURY

**Overview:** The tibial nerve derives from the L4–S3 roots as part of the sciatic nerve. It branches from the sciatic nerve in the distal thigh and continues through the popliteal fossa, entering the calf between the two heads of the gastrocnemius muscle. At the medial ankle, the nerve passes into the foot through the tarsal tunnel. The tarsal tunnel is a fibro-osseous tunnel inferior and posterior to the medial malleolus, formed by the bony floor and the flexor retinaculum. Within the tunnel, it splits into the medial and lateral plantar nerves (LPNs) (Fig. 38.11). The medial plantar nerve (MPN) passes distally between the abductor hallucis and flexor digitorum brevis. The LPN passes between the quadratus plantae and flexor digitorum brevis. Branches:

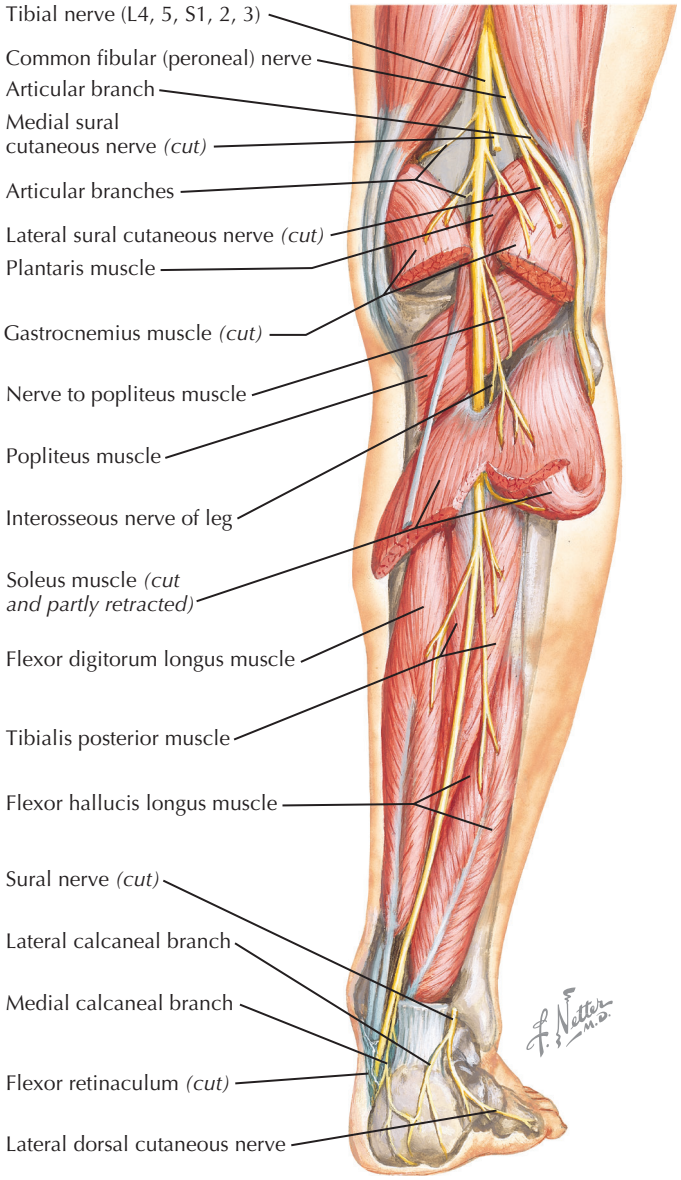



**Figure 38.11.** Tibial nerve.

- **Direct branches:** motor innervation of the semimembranosus, semitendinosus, biceps femoris (long head), plantaris, popliteus, gastrocnemius, soleus, tibialis posterior, flexor hallucis longus, and flexor digitorum longus; sensory innervation of posterolateral calf via sural branches
- **MPN:** motor innervation of the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and first lumbrical; sensory innervation of the medial sole and medial three and a half toes
- **LPN:** motor innervation of the quadratus plantae, flexor digiti minimi, adductor hallucis, interossei, abductor digiti minimi, and lateral three lumbricals; sensory innervation of the lateral sole and lateral one and a half toes
- **Medial calcaneal nerve:** sensory innervation of the plantar and medial heel

**Common sports for injury:** Running, hockey, hiking, skiing, ballet, and gymnastics

**Mechanism of injury:**

- Traumatic: direct or indirect trauma (most commonly to the distal tibia or ankle), overuse, and fractures
- Compression: mass lesion, exostosis, accessory flexor digitorum longus, varicose veins, overpronation, rearfoot valgus deformity, and muscular hypertrophy
- Systemic disorders: such as rheumatoid arthritis, diabetes, etc.

**Symptoms:** Posterior tibial nerve injury (tarsal tunnel syndrome) is the most common neuropathy involving this nerve. Compression occurs within the tarsal tunnel. Patients experience burning pain on the medial plantar foot, which is worse with prolonged standing or walking. Proximal radiation to the calf is not uncommon. Motor weakness is generally not reported, but weakness of toe flexion may occur. Occasional night pain may be reported.

**Differential diagnosis:** Varies depending on symptoms

**Physical examination:** Visual inspection in advanced cases may reveal atrophy of intrinsic foot muscles. Physical inspection should include thorough examination of soft tissues for evidence of mass lesion. Complete neurologic examination of bilateral lower extremities should be included. Inspect the lower back for signs of radiculopathy. Specific tests:

- Pain when the ankle is placed in extremes of dorsiflexion; Tinel's sign behind medial malleolus at the tarsal tunnel or at various entrapment sites distal to the tunnel reproducing symptoms; decreased sensation along the planter aspect of the foot; tenderness and/or mass or swelling at the tarsal tunnel; occasionally weakness with great toe plantarflexion
- A two-point discrimination test on medial and lateral sides of the foot may localize the lesions.

**Diagnostics:** Plain radiographs may reveal fracture or osteophytes. MRI rules out mass lesions. Nerve conduction studies of the medial and/or plantar nerves may be prolonged.

**Treatment:** Determined based on the underlying cause: conservative treatment includes rest, change in footwear or running posture, orthotic with medial support, splinting, anti-inflammatory medications, and corticosteroid injections. Indications for surgical intervention include mass lesions and failure of conservative treatment after 3–6 months.

**Prognosis and RTP:** Conservative treatment is successful in a majority of cases. If surgical intervention is required, approximately 80%–90% of patients will experience improvement or resolution of symptoms; this estimate drops to 75% if the specific cause is not known.

**Less common tibial nerve injuries:**

- MPN injury ("jogger's foot"): Symptoms include burning heel pain, aching in the arch, and decreased sensation in the plantar foot behind the great toe. Compression occurs distal to the tarsal tunnel, most commonly in the abductor tunnel behind the navicular tuberosity. There may also be tenderness of the MPN at the entrance of the abductor tunnel and weakness of intrinsic foot musculature; difficult to differentiate from plantar fasciitis
- LPN injury: Symptoms may include decreased sensation at the lateral one third of the plantar foot. There may be weakness of the abductor digiti quinti, but this is difficult to determine.

## COMMON FIBULAR (PERONEAL) NERVE INJURY

**Overview:** The common fibular (peroneal) nerve (CFN) derives from L4 to S2 roots as part of the sciatic nerve. The fibular nerve branches from the sciatic nerve in the upper popliteal fossa before giving off a lateral sural cutaneous branch, which becomes part of the sural nerve. Traveling posterior to the fibular head, the nerve enters the peroneal (fibular) tunnel and then splits into the superficial fibular nerve (SFN) and the deep fibular nerve (DFN) (Fig. 38.12). Traveling within the lateral compartment, the SFN continues distally along the anterior intermuscular septum, eventually piercing the deep fascia at the distal third of the leg to become subcutaneous. The DFN travels on the interosseous membrane in the anterior compartment before crossing the distal end of the tibia and continuing under the extensor retinaculum and through the anterior tarsal tunnel (a flattened space between the inferior

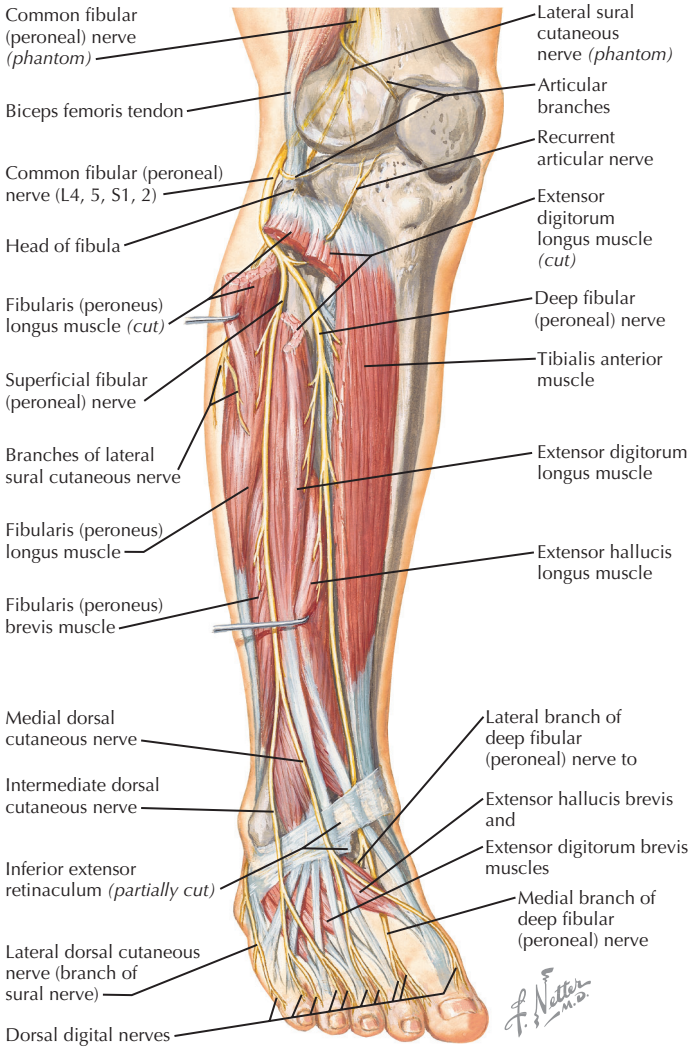



**Figure 38.12.** Common fibular (peroneal) nerve.

extensor retinaculum and the fascia overlying the talus and navicular) to enter the foot. Branches:
- Direct branches: motor innervation of biceps femoris (short head) and sensory innervation of lateral leg
- Lateral sural cutaneous: sensory innervation of the lateral and posterior leg
- DFN: motor innervation of tibialis anterior, extensor hallucis longus and brevis, peroneus (fibularis) tertius, and extensor digitorum longus and brevis; sensory innervation of the first web space
- SFN: motor innervation of peroneus longus and brevis and sensory innervation of dorsum of foot and toes except lateral fifth toe and first web space

**Common sports for injury:** Running, bicycling, soccer, auto racing, football, ballet dancing, hockey, martial arts, surfing, horse racing, and skiing

**Mechanism of injury:**
- Traumatic: Direct contusion, repetitive motion injury, or stretch injury; the latter mostly occurs where the nerve passes through the peroneus longus muscle; may occur with knee dislocations, fibular fracture, proximal tibiofibular instability, and severe ankle inversion
- Compression: Most common at the peroneal tunnel; also occurs with internal masses (e.g., fabella), casting, weight loss, and after prolonged bed rest or prolonged positions such as squatting, kneeling, or sitting cross-legged
- Iatrogenic: During a surgery (i.e., knee arthroscopy) or caused by positioning

**Symptoms:**
- **CFN injury:** Partial or complete foot drop; may be insidious or acute and may present as tripping or falls. Lateral lower leg and dorsal foot paresthesias are common; occurs with compression at the peroneal tunnel
- **SFN injury**: Pain or paresthesia over the lateral calf, lower leg, and/or dorsum of the foot reproduced by resisted ankle dorsiflexion and eversion; etiology usually undetermined but may be associated with fascial defect(s); associated with lateral compartment syndrome
- **DFN injury** (anterior tarsal tunnel syndrome): Pain over the dorsomedial aspect of the foot with radiation into or numbness within the first web space; occurs with compression of the nerve at the ankle as it passes the talonavicular joint; associated with anterior compartment syndrome

**Differential diagnosis:** Varies depending on symptoms

**Physical examination:** Visual inspection in chronic cases may display atrophy in the anterior and lateral compartments of the leg. Observation of the patient while ambulating may reveal steppage gait or hip hiking. Physical inspection should include thorough examination of soft tissues around the knee for evidence of mass lesion or fascial hernia. Consistent neurologic findings include difficulty in heel walking, weakness with dorsiflexion, great toe extension, and weakness with foot inversion while in dorsiflexion but not plantarflexion. Forced inversion may increase pain. Sensory deficits are more common at the first web space (DFN) than over the SFN and CFN distribution. Reflexes should be normal. Specific tests:
- CFN: Weakness of ankle and toe dorsiflexors and ankle eversion; hypoesthesia to touch and pain in the lower two thirds of the lateral leg and dorsum of the foot; Tinel's sign may be positive at the CFN (fibular head)
- SFN: Worsening of symptoms with plantar flexion and inversion of the foot
- DFN: Weakness of the extensor digitorum brevis muscle; sensory deficit in the first web space; ankle eversion normal; Tinel's sign may be positive at the DFN (anterior compartment, middistal tibia)
- Consider palpation of the muscular compartments after exercise to evaluate for compartment syndrome, particularly with DFN symptoms
- Inspect the lower back for signs of radiculopathy. Be sure to differentiate back pain secondary to abnormal gait from primary lower back pathology associated with radiculopathy.

**Diagnostics:** Plain radiographs to rule out fracture or compressive exostosis; MRI should rule out mass lesions. Electrodiagnostics are considered the gold standard for fibular nerve injuries. Compartment pressure testing may be warranted if clinically suspected.

**Treatment:** Conservative treatment includes avoidance of continued compression by any object or position (may include use of protective pads), rest, anti-inflammatory medications, and physical therapy. Wearing a looser shoe for DFN compression may help. Corticosteroid injections are also used. Based on the degree of weakness, bracing with an ankle–foot orthosis may be required for foot drop and prevention of contracture. Indications for surgical referral include nerve laceration, compression by a mass lesion, and lack of clinical improvement with conservative measures. Concurrent anterior compartment syndrome with DFN lesions may require decompression.

**Prognosis and RTP:** Resolution should be expected within 2–6 months depending on the etiology. SFN is less likely to respond to conservative treatment and usually requires surgery. If surgical intervention is necessary, typically, a good response is expected, but this may vary with site of compression and degree of palsy.

## COMPLEX REGIONAL PAIN SYNDROME (CRPS)

**Overview:** An array of painful conditions characterized by continued regional pain that is disproportionate in time or degree to usual course of trauma/lesion. Pain is regional (not in a specific nerve territory), usually with distal predominance of abnormal sensory, motor, sudomotor, vasomotor, and/or trophic findings. Synonyms include reflex sympathetic dystrophy (CRPS I), causalgia (CRPS II), posttraumatic pain syndrome, Sudeck's atrophy, and shoulder–hand syndrome. The diagnosis of CRPS is often missed. The average time between symptom onset and diagnosis in children has been reported to be 1 year. Two subtypes are recognized: Type I corresponds with no evidence of peripheral nerve injury (90% of presentations), and Type II corresponds to the presence of peripheral nerve injury.

**Diagnostic criteria:** Multiple diagnostic criteria have been published. The Budapest criteria became the official International Association for Study of Pain (IASP) diagnostic criteria for CRPS in 2012. **Budapest criteria:** For clinical diagnosis of CRPS, the following must be met:
- Continuing pain disproportionate to inciting event
- At least one symptom in three of the four categories must be reported:
  - Sensory: Hyperesthesia and/or allodynia
  - Vasomotor: Temperature asymmetry and/or skin color changes and/or skin asymmetry
  - Sudomotor/Edema: Edema and/or sweating changes and/or sweating asymmetry
  - Motor/Trophic: Decreased ROM and/or motor dysfunction (weakness, tremor, or dystonia) and/or trophic changes (hair, skin, or nails)
- Must display at least one sign at time of evaluation in two or more of the following categories:
  - Sensory: Hyperalgesia (to pinprick) and/or allodynia (to light touch and/or temperature and/or deep somatic pressure and/or joint movement)
  - Vasomotor: Temperature asymmetry (>1°C) and/or skin color changes and/or asymmetry
  - Sudomotor/Edema: Edema and/or sweating changes and/or sweating asymmetry
  - Motor/Trophic: Decreased ROM and/or motor dysfunction (weakness, tremor, or dystonia) and/or trophic changes (hair, skin, or nail)

- No other diagnosis that better explains the signs and symptoms
- Mean age for adults is 36–46 years with 60%–80% of patients being women
- Children aged 9–15 years are most commonly affected, with a girl-to-boy ratio of 3:1.

**Presentation:** Pathophysiology is unclear. Common associated precipitating conditions include immobilization, fracture, strains/sprains, minor trauma, postsurgical, and contusion or crush injury. In certain cases, no initiating event is identified. A deviation from the normal course of recovery may be the first indication of CRPS. Symptoms include neurogenic pain (particularly "burning pain"), allodynia (e.g., minimal stimulus such as bed sheets over affected area cause severe pain), hyperesthesia, asymmetry of color/temperature, increased sweating of the affected limb, edema, sensory deficits, stiffness, weakness, night pain, and skin mottling (particularly after showers). Stressors in the lives of pediatric patients with CRPS may play a greater role than in their adult counterparts. Lower extremity involvement is more common in children at a ratio of 5:1, whereas upper extremity involvement is more common in adults.

**Physical examination:** Pain assessment is critical. General examination should include ROM and a thorough neurologic examination. Specific testing for unilateral weakness, skin temperature changes, allodynia, edema, and sudomotor changes should be included. The key to the examination may be in comparing the affected and unaffected limbs.

**Diagnostics:** The diagnosis of CRPS is clinical, and no objective test is needed. Additional objective tests such as thermography, triple phase bone scan, quantitative sudomotor axon reflex test, and sympathetic ganglion block have been described but are again not necessary. Other objective tests (MRI, electrodiagnostics, duplex ultrasound, etc.) may be needed to rule out conditions that could account for similar signs and symptoms.

**Treatment:** CRPS is a complex biopsychosocial condition that requires a multidisciplinary approach with a goal of improving function. If multidisciplinary resources are not available, sports physicians may design a home program consisting of desensitization exercises (rubbing affected body part with various stimuli such as washcloth, toothbrush, shaving cream, and hot and cold), stretching, and strengthening exercises. Initial exercises of a short duration (few seconds) and progressing through tolerated discomfort for increasing periods; serial follow-up is important, especially initially. Psychological support is recommended if pain lasts for >2 months. Rehabilitation is the core treatment. Additional therapeutics should be aimed at facilitating therapy. Several pharmacologic therapies have been incorporated into the standard treatment regimen of CRPS, including antidepressants, antiepileptic medications, opioids, topical analgesics, NMDA antagonists, bisphosphonates, etc.; rotating schedules should be employed. Interventional pain management (e.g., epidural anesthesia, sympathetic nerve blocks, and neurostimulation, etc.) is common.

**Prognosis and RTP:** Prognosis and RTP decisions are based on individual cases and vary widely; in general, a more prolonged course is associated with poor prognosis. RTP should not be the primary goal of treatment with pediatric athletes; rather, symptom management and activities of daily living should be prioritized. Studies have reported that 50% of pediatric athletes with CRPS will return to competitive sports. Be mindful of the fact that this presentation may offer the pediatric athlete a way out of competitive sports without parental disapproval.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 39 HEADACHE IN THE ATHLETE

Kinshasa C. Morton

## GENERAL PRINCIPLES

- **Headache is one of the most common disorders and symptoms reported to primary care, the emergency department, and team physicians.**
- Complaints of headache account for 1%–4% of primary care office and emergency department visits.
- In the general population, the prevalence of headache in a 1-year period is >90%, and the lifetime prevalence is 93%–99%.
- Headaches are one of the most commonly reported pain complaints among children and adolescents.
  - In a population study of adolescents aged 11–21 years, >90% had experienced headaches, regardless of the type, over 1 year.
  - Among children aged 4–17 years in the United States, 6.7% had frequent headache pain over a 12-month period.
- Various studies on athletes have shown a headache prevalence of 35%–50% related to participation in their sport.

### Causes of Headache

- The exact cause of many headaches is a source of much debate and likely differs for each specific type of headache. Physiologic changes in the head and brain that consist of, but are not limited to, changes in neurotransmitter regulation, vascular dilation and constriction, and cranial nerve irritation that cause activation of pain signals are all contributors to the development of headache. Cyclical hormonal changes and genetic predisposition are also thought to play an important role.
- Tension-type headache (TTH), migraine, and cluster headaches combine to make up >90% of headaches experienced by the general population.
- Adults mostly experience TTHs, whereas children are more likely to have migraine headaches.
  - Among children aged 3–18 years who visit pediatric neurologic clinics, vascular/migrainous headaches account for 52%, chronic headaches/TTHs for 21%, and unclassified headaches for 19%. Remainders are mixed tension-migraine, psychogenic, or posttraumatic.
- Of headache-caused emergency department visits:
  - Migraine and TTHs account for 25%–55% of visits.
  - Headache associated with systemic illness accounts for 33%–39% of visits.
  - Headache caused by a serious neurologic condition (subarachnoid hemorrhage, intracranial mass, meningitis, or hemorrhage) accounts for 1%–16% of visits.

### Classification of Headache Disorders

- **In 1988, the International Headache Society (IHS) created its first classification of headache disorders. This classification was updated and revised in 2003 and then again in 2013.**
- Headache has many causes. The IHS classification gives a physician a logical approach to make a rapid and accurate differential diagnosis.
- Headache disorders are classified as primary, secondary, or those caused by cranial neuralgias.
  - Primary headaches are those that have no underlying cause; there are three main types: migraine headache, TTH, and cluster headache (trigeminal autonomic cephalalgias).
  - Secondary headaches are those that may be attributed to certain underlying pathologic conditions, i.e., infectious, neoplastic, vascular, psychiatric, traumatic, drug-induced, or homeostatic changes.
  - Cranial neuralgia headaches are those that are initiated by compression, distortion, or irritation of specific cranial nerves and subsequently cause pain in the area that those nerves innervate.
- The athletic population suffers from the same headache disorders as the general population but may also have a predisposition to suffer from other headache disorders and subtypes because of the effects of exercise and because of participation in their specific sports.
  - Sports-/exercise-related primary headaches are exercise-/effort-induced migraine and primary exertional headaches.
  - Sports-/exercise-related secondary headaches are acute and chronic posttraumatic headache (PTH), cervicogenic headache, high-altitude headache, and diving headache.
  - Sports-/exercise-related cranial neuralgia headaches are external compression headache and cold-stimulus headache.

## ASSESSING HEADACHE IN THE GENERAL AND ATHLETIC POPULATION

Like all other medical evaluations, the assessment of headache begins with a thorough history and physical examination. Most patients will have a completely normal general physical and neurologic examination, so obtaining a detailed history is critical. After history and physical examination have been completed, a further investigation may be warranted, depending on the history and physical findings. This investigation may include laboratory workup, imaging, and/or diagnostic procedures.

### Headache History

- When was the first onset of headache?
- What is the frequency?
- Where is the location and is there any radiation of pain?
- What is the character of the pain? (dull, throbbing, sharp)
- What is the intensity of the pain?
- Are there any associated symptoms?
  - Nausea/vomiting
  - Photophobia/phonophobia
  - Confusion
  - Blurry vision
  - Gait disturbances
- Were there any medications taken?
- Are there any alleviating or exacerbating factors?
- Is there a prior history of headache?
- Is there a family history of headache?
- Has there been a change in the headache characteristics? (if prior history is positive)
- Is there a history of other medical issues?
- What medications is the athlete taking?

## Physical Examination

- Assess for level of consciousness, orientation, alertness, mini-mental status, and overall affect.
- Check vital signs to rule out hypertension.
- Evaluate head, eyes, ears, nose, and throat to assess for papilledema. Check for central nervous system abnormalities, including cranial nerve testing, ptosis, pupil reactivity, and head trauma.
- Conduct musculoskeletal evaluation to assess for nuchal rigidity, temporomandibular joint problems, cervical spine range of motion, areas of scalp tenderness, and neck tenderness.
- Conduct neurologic examination to assess for motor, sensory, and reflex response; also include evaluation of gait.
- Check for evidence of systemic illness (assess abnormalities in the cardiovascular, respiratory, and gastrointestinal [GI] systems).

## Additional Investigation

**Laboratory evaluation:** To rule out metabolic issues. Laboratory examinations that may aid in diagnosis are erythrocyte sedimentation rate, complete blood count, liver function tests, thyroid function tests, antinuclear antibodies, antiphospholipid antibodies, and drug screening. Appropriate tests must be conducted as indicated by the history and physical examination.

**Imaging tests:** Possibilities include cervical spine films, CT (with or without contrast), MRI, EEG, and MR angiography. Selection of tests should be individualized.

- Cervical spine radiography (flexion/extension views) in patients with trauma/neck pain, possible fracture, and instability.
- CT scan with contrast for patients with new-onset exertional headache.
  - Some experts do not advocate CT unless neurologic examination is abnormal.
  - May not be helpful for suspected posterior fossa lesions
- CT better for acute bleeding and bony fractures; detects surgical lesions as well as MRI scan
- MRI has greater tissue contrast resolution than CT.
  - Better to perform MRI >48 hours after trauma and in patients with arteriovenous malformations (AVMs) or tumor
  - Study of choice when looking for lesions in the posterior fossa
  - Less radiation exposure compared with CT
- According to the Quality Standards Subcommittee of American Academy of Neurology, routine use of brain imaging is not warranted for adults with recurrent headaches that have been defined as migraines with no recent change in pattern, no history of seizure, and no other focal neurological signs or symptoms.

**Diagnostic procedure: Lumbar puncture (LP)** to rule out subarachnoid hemorrhage from aneurysm; detects minor leaks, which occur in 39% of patients who later have ruptures. CT scans are negative in 55% of patients with minor leaks.

## General Considerations

- Consider the same diagnostic evaluation in athletes as in non-athletes, except in cases of trauma with an increased risk of intracranial bleed.
- Workup should be complete when diagnosis is in question. This may include, but is not limited to, evaluation for drug abuse, hypertension, vascular lesions, neoplasms, intracranial bleeding, and psychiatric issues.
- Be cognizant of "red flags" within the history or physical examination that should prompt further neurologic evaluation in the form of either imaging or other diagnostic procedures (Box 39.1).

**BOX 39.1 RED FLAGS IN THE EVALUATION OF HEADACHE**

1. Headache onset after the age of 50 years
2. Sudden onset of severe headache
3. Change in headache pattern
4. Headache associated with systemic illness and fever
5. Headache associated with neck stiffness
6. Headache with focal neurologic deficits
7. Presence of papilledema
8. Headache in the setting of moderate/severe trauma
9. Alteration of consciousness or amnesia
10. Early morning nausea and vomiting without headache
11. Occurrence of seizure(s) after headache

# PRIMARY HEADACHE DISORDERS

See Table 39.1.

# MIGRAINE HEADACHE

- **Definition:** Migraine is a chronic, idiopathic neurologic disorder characterized by episodic headaches of high intensity that may be preceded by, accompanied with, or followed by other associated symptoms (Figs. 39.1 and 39.2).
- Associated symptoms
  - **Premonitory (prodrome) symptoms:** Occur hours up to 1–2 days before development of migraine headache. These symptoms are often thought of as a warning of an impending migraine.
    - Fatigue
    - Difficulty concentrating
    - Yawning
    - Change in appetite
    - Change in sleep patterns
    - Change in mood
  - **Aura:** A set of reversible focal neurologic symptoms that occur at the beginning of a migraine headache or just before its onset. Auras typically last for <60 minutes. Visual symptoms are most common.
    - Visual changes (scintillating scotoma, visual field deficits, and blurry vision)
    - Sensory disturbances (pins and needles sensation and numbness)
    - Speech disturbances (dysphasia)
    - Motor deficits (rare)
  - **Resolution (postdrome) symptoms:** These are symptoms that follow the headache and may include certain premonitory symptoms.
    - Exhaustion
    - Exhilaration
    - Depression
    - Nausea
    - Scalp tenderness
- Migraine headache is divided into two major subtypes by the IHS.
  - Migraine without aura
  - Migraine with aura (10%–15% of migraines)
- Migraine headaches are typically unilateral but can be bilateral. Bilateral migraine is more likely to occur in children/adolescents than adults. They also tend to have a pulsating nature, moderate-to-severe intensity, exacerbation by activity, and an association with nausea, vomiting, photophobia, and phonophobia.

## TABLE 39.1 CHARACTERISTICS OF MIGRAINE, CLUSTER, AND TENSION-TYPE HEADACHES

| | Migraine | Cluster | Tension-Type |
|---|---|---|---|
| **Onset** | Peaks in adolescence | 30′ s or 40′ s | Variable; ≥20′ s |
| **Frequency** | 1–2 attacks/mo, often with menses | ≥1 attacks/day for 6–8 wk | Episodic: <15 days/mo<br>Chronic: >15 days/mo |
| **Location** | Unilateral more common than bilateral; frontotemporal or orbital | 100% unilateral<br>Generally orbitotemporal | Bifrontal, bioccipital, neck |
| **Quality** | Throbbing or intense pressure | Nonthrobbing, penetrating, boring | Squeezing, pressing, aching |
| **Duration** | 4–72 h, usually 12–24 h | 30 min–2 h, usually 45–90 min | Episodic: several hours<br>Chronic: all day |
| **Prodrome** | Changes in mood, energy, appetite | May include brief, mild burning in eye and internal nares | None |
| **Aura** | Up to 60 min, usually 20 min; 'often' visual | None | None |
| **Associated Symptoms** | Nausea, vomiting, photophobia | Ipsilateral ptosis-miosis, conjunctival injection, tearing, stuffed and running nose | Episodic: loss of appetite, light or sound sensitivity<br>Chronic: light or sound sensitivity, nausea |
| **Behavior** | Go to dark, quiet room | Frenetic pacing, rocking | Generally not affected or mild decrease in function |

Modified from Marks DR, Rapoport AM. Practical evaluation and diagnosis of headache. *Semin Neurol*. 1997;17(4):307-312.

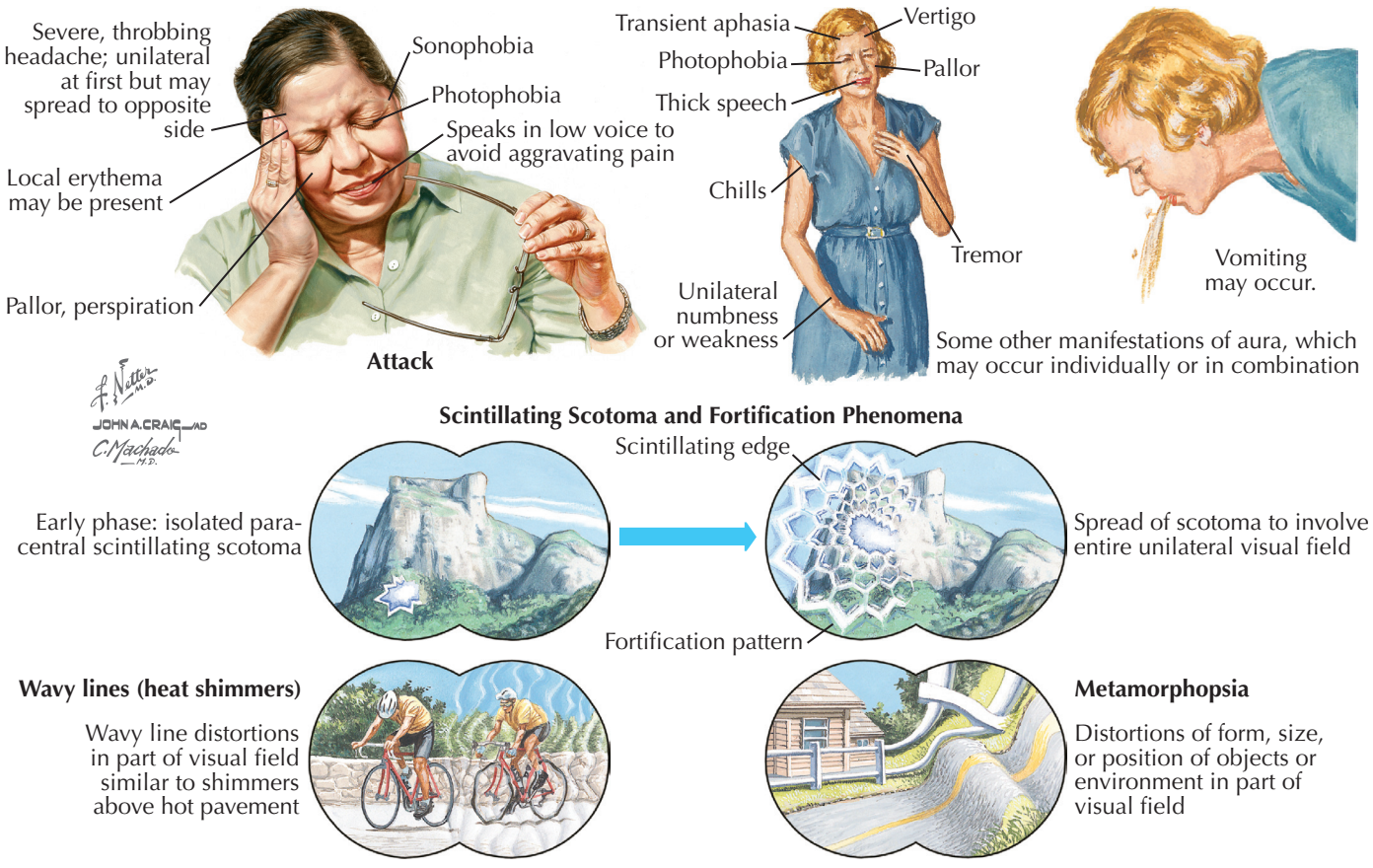
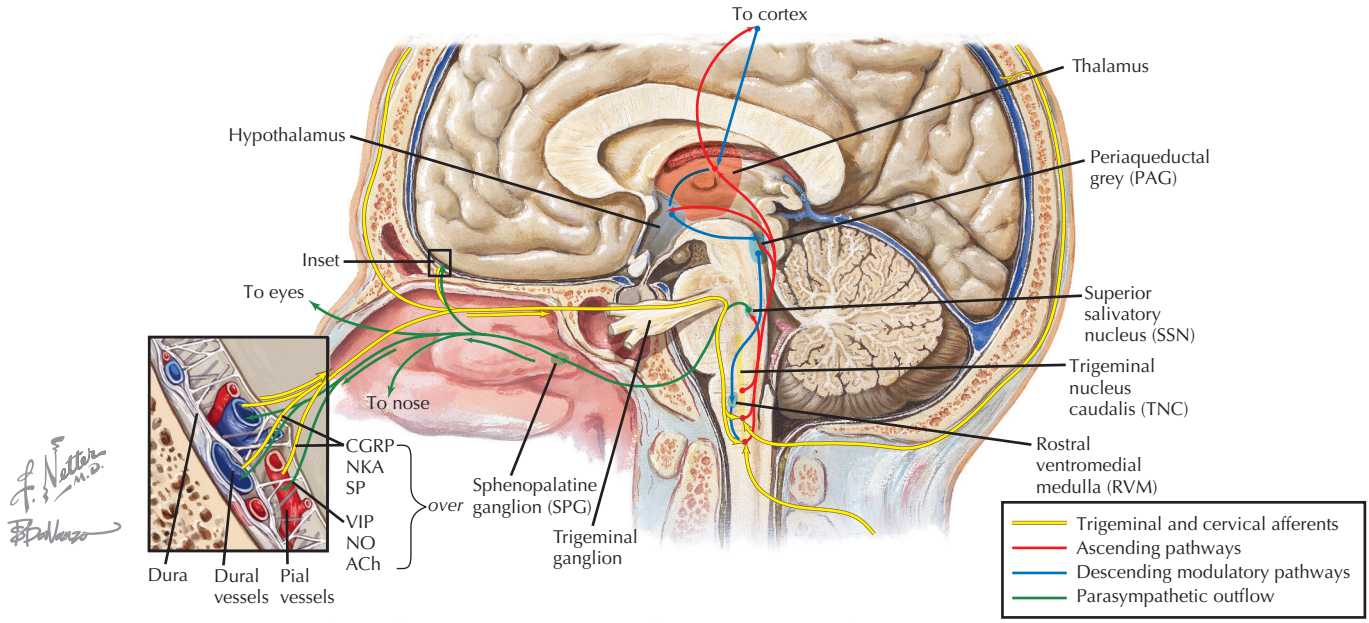
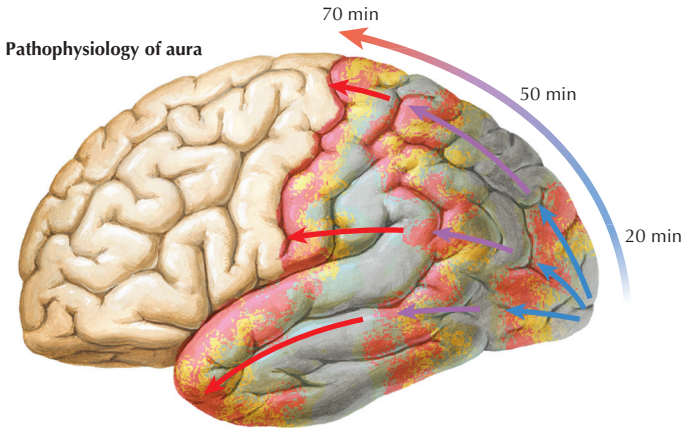



**Figure 39.1.** Mechanisms of migraine.




Pain-producing structures in the head send pain information via primary sensory afferent neurons through the trigeminal nerve and upper cervical roots to synapse on the second-order neurons in the trigeminal nucleus caudalis (TNC) as part of the trigeminocervical complex. Neurons in the TNC send projections to the thalamus (via the trigeminothalamic or quintothalamic tract, which decussates in the brainstem), which then projects to the cortex. The TNC is thought to project to other structures as well, including the periaqueductal gray (PAG), which also send signals to the thalamus and hypothalamus, with projections to the cortex. There are descending projections from the cortex back to the thalamus and hypothalamus. Descending modulation of the TNC takes place via nuclei in the hypothalamus, as well as direct projections from the PAG through the rostral ventromedial medulla (RVM).

Cranial parasympathetic outflow stems from a reflex connection from the TNC to the superior salivatory nucleus (SSN) in the pons. Efferents from the SSN (via the facial nerve) connect with neurons in the sphenopalatine ganglion (SPG; pterygopalatine). The SPG then projects to innervate intracranial vessels (vasodilation), as well as the nasal and lacrimal glands.




A wave of cortical spreading depression (CSD) gradually spreads through the cortex at a rate of 3-5 mm/min. It is thought that the wave of neuronal depolarization associated with transient increased then decreased cortical blood flow is responsible for the patient's symptoms as it moves through the occipital cortex, somatosensory cortex, and language areas.

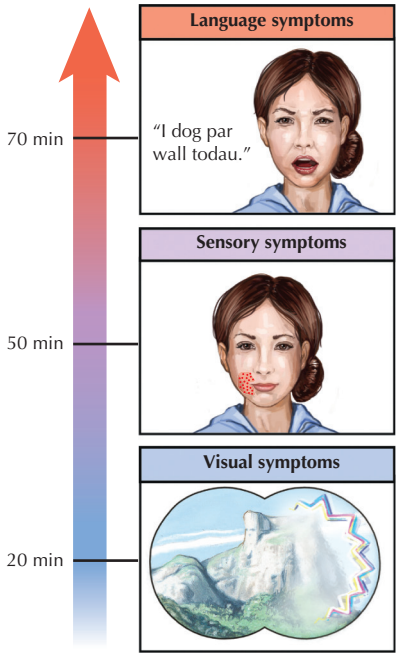



**Figure 39.2.** Migraine pathophysiology.

**BOX 39.2 IHS DIAGNOSTIC CRITERIA FOR MIGRAINE WITHOUT AURA**

**A.** At least five attacks fulfilling criteria B–D
**B.** Headaches lasting for 4–72 hours
**C.** Headache has at least two of the following characteristics:
  **1.** unilateral location
  **2.** pulsating quality
  **3.** moderate or severe pain intensity
  **4.** aggravated by physical activity
**D.** During headache, at least one of the following is present:
  **1.** nausea and/or vomiting
  **2.** photophobia and phonophobia
**E.** Cannot be attributed to another disorder

Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

- IHS diagnostic criteria for migraine are enlisted in Box 39.2.
- Typical age of onset is in adolescence.
- 18% of women and 6% of men report headaches that meet the definition of migraine. The female-to-male ratio is 3:1.
- A survey of 791 male and female NCAA division I basketball players showed a total prevalence rate of 2.9% (0.9% of males, 4.4% of females).
- 70% of females who suffer from migraines note a relationship between their menstrual cycles and migraine attacks.
  - Migraine without aura: highest incidence during first 3 days of menses
- Severe, debilitating forms of migraine include status migrainous, in which migraine lasts for >72 hours, and chronic migraine, in which migraines occur for ≥15 days per month.
- Common migraine triggers include aspartame, caffeine (use or withdrawal), estrogens, monosodium glutamate, nicotine, nitrates, progesterone, alcohol, cheese, chocolate, menstruation, missed meals, perfume, red grapes, sleep (too much or too little), stress, changes in environment/weather, and exercise.

### Treatment Options

**Overview: Abortive treatment** is appropriate if headaches occur once or twice per month, particularly if predicted by aura. It is more effective if used as early as possible, and a large single dose is more effective than multiple smaller doses.

**Nonsteroidal anti-inflammatory drugs (NSAIDs):** Chronic use should be avoided, particularly in those with a history of peptic ulcer disease, gastritis, and renal insufficiency. Specific medication and initial dosages:
- Ibuprofen: 1200 mg PO (peak effect at 1 hour)
- Naproxen: 500–825 mg PO (peak effect at 2 hours)
- Indomethacin: 50 mg PO
- Ketorolac: 30–60 mg IM, with additional 30 mg in 8 hours if necessary

**Dihydroergotamine (DHE-45):** Is available in parenteral form and is typically given with an antiemetic
- **DHE-45:** 1.0 mg IV/IM/SQ, given 30 minutes after metoclopramide 5 mg or prochlorperazine 5 mg; may be repeated in 1 hour (90% effective)
- **DHE-45 (nasal):** 1.0 mg of intranasal puffs, repeating in 15 minutes
- Favorable side-effect profile when compared to ergotamine (minimal cardiovascular effects and nausea; no rebound headache; nausea/vomiting, GI upset, and muscle cramping have been reported)
- DHE-45 is a vasoconstrictor and is therefore contraindicated in patients with cerebrovascular, cardiovascular, or peripheral vascular disease. It is also contraindicated in patients with severe hypertension, ischemic heart disease, renal or hepatic disease, sepsis, recent infection, or those who may be pregnant.

**Triptans:** This class of medication causes vasoconstriction and blocks pain pathways within the brainstem. They are selective 5-hydroxytryptamine (5-HT) receptor agonists and are specifically used to treat migraines. All medications belonging to this class have similar side-effect profiles (nausea, dizziness, chest pressure, asthenia, and dry mouth). They also carry the same precautions in that they should be avoided if possible in those with cardiovascular disease, peripheral vascular disease, hypertension, and hepatic dysfunction.
- **Sumatriptan (Imitrex):** 5-HT1D receptor agonist; the first triptan developed, it is available in injectable, oral, and intranasal spray forms
  - Injectable dosage: 4–6 mg SC initially; may repeat in 1 hour (maximum of two 6-mg injections in 24 hours); recommended initial dose is 6 mg
  - Oral dosage: 25–100 mg initially; may repeat dosage in 2 hours (maximum of 200 mg in 24 hours). The recommended initial dose is 50 mg because it is more efficacious than 25 mg and has fewer side effects than 100 mg without loss in efficacy.
  - Nasal spray dosage: 5–20 mg initially; may repeat in 2 hours (maximum of 40 mg in 24 hours); recommended initial dose is 20 mg
  - Tfelt-Hanson showed that 6 mg of the injectable form was more efficacious than 100 mg of the oral form and also had the fastest onset of action of all forms. The intranasal form has the same efficacy and a faster onset of action than the oral form, but limited therapeutic effects are seen for the first 30 minutes.
  - DHE versus SC sumatriptan: DHE less effective at 2 hours (73% vs. 85%), but no difference after 2 hours; headache recurred in 45% of sumatriptan-treated patients and 17.7% of DHE-treated patients.
  - Other efficacious triptans include: **Naratriptan (Amerge), Frovatriptan (Frova), Rizatriptan (Maxalt), Eletriptan (Relpax), Zolmitriptan (Zomig), and Almotriptan (Axert)**

**Others:**
- **Midrin** contains isometheptene (sympathomimetic amine vasoconstrictor), dichloralphenazone (sedative), and acetaminophen. Consider it in patients unable to use ergots. Dosage: 2 tablets PO; may take 1 tablet every 1 hour after initial dose until improvement (maximum of 5 tablets per attack)
- **Butalbital combinations** (Fiorinal or Fioricet) may also be used. Special concerns may arise regarding abuse potential and sedation. Dosage: 2 tablets PO; may repeat (in 4 hours maximum of 6 tablets in 24 hours)
- **Intranasal butorphanol:** Mixed agonist-antagonist opioid analgesic; used for severe attacks; one puff equals 5 mg morphine
  Dosage: 1 spray; may repeat in 1 hour
- **Intranasal lidocaine 4% solution** provided rapid relief (within 5 minutes) of headache, nausea, and photophobia in a prospective, double-blind, placebo-controlled trial. Relapse was common.
- **Intravenous antiemetic medications** (chlorpromazine, prochlorperazine, and metoclopramide) have shown some efficacy as monotherapy in the treatment of migraine. Practical use of such medications outside of the emergency department setting is low.

### First-Line Choice in Athletes

**Overview:** Because of the efficacy, ease of administration, and relatively quick onset of action, athletes should use oral or

injectable forms of triptans for first-line therapy of moderate migraines. Nasal DHE may be another good option secondary to ease of administration. For mild migraines, NSAIDs in any form should be tried initially.

**Prophylactic treatment:** Indicated in patients with ≥4 attacks per month or attacks lasting several days that cause severe disability

- **Beta-blockers** are agents of choice.
  - Only two are FDA approved: propranolol and timolol, which lack intrinsic sympathomimetic activity (ISA)
  - Long-acting form of propranolol increases compliance and is useful in patients with coexisting hypertension, angina pectoris, or thyrotoxicosis.
  - Contraindicated in patients with asthma, chronic obstructive pulmonary disease, congestive heart failure, atrioventricular conduction problem, or prescription for monoamine oxidase inhibitors
  - Other beta-blockers used despite lack of FDA approval: nadolol, metoprolol, and atenolol

  Metoprolol is a selective beta-blocker that can be used in patients with concomitant pulmonary disease.
  - **Beta-blockers and athletes:** Negative effects on aerobic performance cause some concern for use in athletes. Moreover, beta-blockers are banned in certain sports and by certain athletic rules committees because they may provide an unfair advantage.
- **Calcium channel blockers:** Nimodipine, a selective cerebrovascular vasoconstrictor, and verapamil have been shown to be useful in migraine prophylaxis.
- **NSAIDs:** Various NSAIDs may be used with varying degrees of efficacy. They are more efficacious for **menstrual migraines** and should be started 2–3 days before menses (see later discussion).
- **Antidepressants (tricyclic):** Amitriptyline and clomipramine are used most often. Efficacy is attributed to antidepressant and analgesic actions. They are particularly effective in patients with coexisting migraine and TTH. They are sedating and hence should be used at bedtime.
- **Antiepileptics:** Valproate, topiramate, and gabapentin are used most often. Only valproate and topiramate are FDA approved for this indication. They are category C and D drugs, respectively, so there should be a concern for use in premenopausal female athletes; ensure birth control. There are no known adverse effects on exercise, although certain antiepileptics are sedating.

**Menstrual migraine treatment: "Miniprophylaxis"** may be achieved with the use of NSAIDs or long-acting triptans (frovatriptan and naratriptan) when initiated 2–3 days before menstruation and continued until 3 days after. **Hormonal treatment** can be used to prevent estrogen withdrawal, which contributes to the development of menstrual migraine. Treatment strategies include the use of extended- and regular-cycle oral contraceptive pills with supplemental estrogen during the inactive pill week, and the use of supplemental estrogen alone during menstruation.

## Tension-Type Headache

- Definition: TTHs are headaches of mild to moderate intensity that can be either episodic with varying frequency or chronic with episodic headaches being much more common. Pain is usually bilateral, and patients often report it being "vice-like." The most significant examination finding is pericranial tenderness to palpation (Fig. 39.3).
- IHS diagnostic criteria for episodic TTH are listed in Table 39.2.
- TTH is the most common type of primary headache, with prevalence reaching 80% in the general population.
- Both episodic and chronic TTHs may exhibit photophobia and phonophobia, but nausea is usually isolated to chronic TTH.
- Chronic TTH (CTTH): defined by IHS as headache present for >15 days per month for >3 months.
- CTTH prevalence in general population is 2% in men and 5% in women.

## Treatment Options

**Overview:** TTHs are often responsive to nonpharmacologic therapy or over-the-counter headache medications. Those who suffer from frequent TTHs may benefit from stress reduction and relaxation techniques. For athletes, alteration of training regimens may be helpful. Counseling for patients with psychosocial issues including, but not limited to, anxiety and depression is also beneficial.

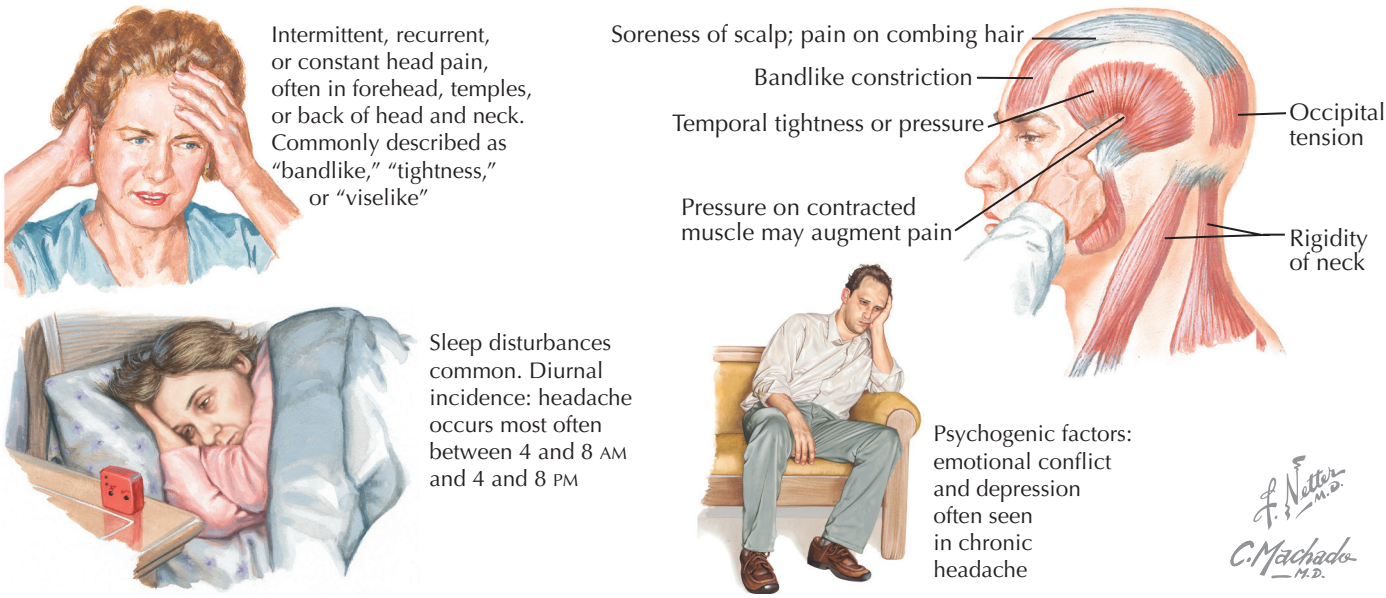



**Figure 39.3.** Tension-type headache.

**TABLE 39.2 IHS CRITERIA FOR EPISODIC TTH**

| Infrequent Episodic TTH | Frequent Episodic TTH |
|---|---|
| A. At least 10 episodes occurring on <12 days per year and fulfilling criteria B–D | A. At least 10 episodes occurring on >1, but <15 days of a month for 3 months and fulfilling criteria B–D |
| B. Headache lasts from 30 min to 7 days | |
| C. Headache has at least two of the following:<br>1. bilateral location<br>2. pressing/tightening (nonpulsating) nature<br>3. mild or moderate intensity<br>4. not worsened by physical activity | |
| D. Headache has both of the following:<br>1. no nausea or vomiting<br>2. only one of photophobia or phonophobia | |
| E. Cannot be attributed to another disorder | |

Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

**Abortive treatment:**

- **NSAIDs:** Ibuprofen, naproxen, indomethacin, ketorolac, and others are used. Smaller dosages than used in migraine are typically effective for TTHs.
- **Butalbital combinations** (Fiorinal, Fioricet) may be used as well; less frequent dosing intervals than those used for migraine are usual. Special concerns may arise regarding abuse potential and sedation

  Dosage: 2 tablets PO; may repeat (in 4 hours maximum of 6 tablets in 24 hours)
- **Tramadol (Ultram):** An opioid analog; it may have some abuse potential

  Dosage: 100 mg PO four times daily (maximum of 400 mg in 24 hours)

**Prophylactic treatment:**

- **Tricyclic antidepressants: Amytriptyline is most commonly used.**
- **Beta-blockers:** Propranolol and timolol are used; not a great choice for athletes because they impair aerobic exercise. They act by reducing $VO_2$ max and maximum heart rate and increasing fatigue. They are banned by the International Olympic Committee in diving and shooting.

**General TTH treatment considerations:** Watch for headache secondary to analgesic use and "rebound pain." Concerns and side effects in athletes differ from those in the general population; individualize treatment if possible.

## Cluster Headaches

- **Definition:** Cluster headaches present as a series of reoccurring headaches that persist from weeks to months at a time, followed by remission periods. Pain is always unilateral and is of a severe, penetrating, and stabbing nature. The location of these headaches is orbital, periorbital, or temporal (Fig. 39.4).

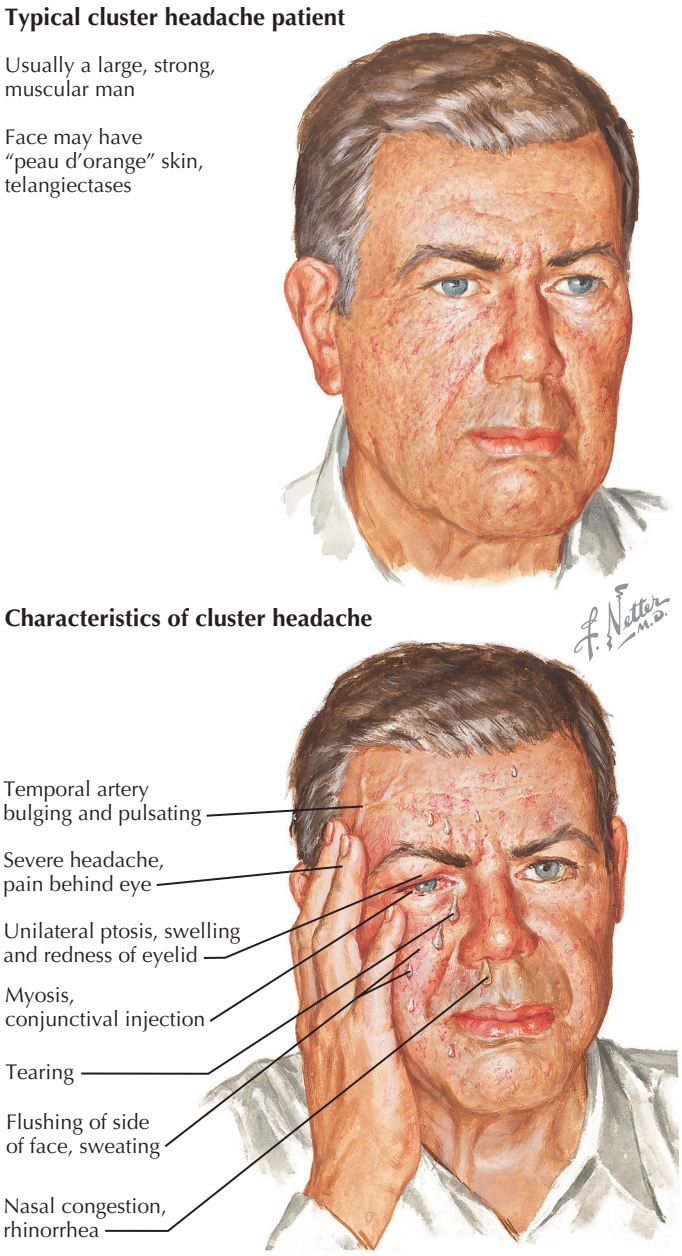



**Figure 39.4.** Cluster headache.

- Most important differentiating feature of cluster headaches is the presence of transient autonomic symptoms.
- Recur throughout the day and wake approximately 50% of patients from sleep
- IHS diagnostic criteria for cluster headaches are listed in Box 39.3.
- Occur more frequently in men than in women, with research showing male-to-female ratios that range from 2:1 to 6.7:1
- Typical age of onset is between the 3rd and 5th decades.
- Alcohol, nitroglycerine, and histamine may trigger headaches.
- Patients pace, rock, or bang head against the wall (vs. patients with migraine, who sleep).
- Chronic cluster headache is a more severe form wherein headache attacks occur for >1 year either without remission or remission lasting for <1 month.

**BOX 39.3 IHS CRITERIA FOR CLUSTER HEADACHES**

**A.** At least five attacks fulfilling criteria B–D
**B.** Severe unilateral orbital, supraorbital, and/or temporal pain lasting for 15–180 minutes
**C.** Headache is accompanied by at least one of the following transient autonomic symptoms:
  **1.** ipsilateral conjunctival injection and/or lacrimation
  **2.** ipsilateral nasal congestion and/or rhinorrhea
  **3.** ipsilateral eyelid edema
  **4.** ipsilateral forehead and facial sweating
  **5.** ipsilateral miosis and/or ptosis
  **6.** sensation of fullness in the ear
  **7.** a sense of restlessness or agitation
**D.** Attacks have a frequency of one every other day to up to eight per day
**E.** Cannot be attributed to another disorder

Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

### Treatment Options

ABORTIVE TREATMENT

- Limited duration of headache makes most abortive treatment ineffective.
- **DHE-45** as used with migraine; DHE (0.5–1.0 mg) IV, IM, or intranasal
- **Oxygen:** Inhalation with a nonrebreather mask; 8 L per minute over 10–15 minutes; 70% response
- **Sumatriptan** as used with migraine; because of its rapid onset, it is considered to be the most effective agent
- **Intranasal lidocaine 4%:** Injected into sphenopalatine fossa, effective in certain cases
- Oxygen and sumatriptan are abortive treatments of choice in athletes.

PROPHYLACTIC TREATMENT

- **Corticosteroids:** Used as a bridge between abortive and prophylactic therapy, they are effective in halting the cluster cycle
  **Prednisone:** 40–60 mg PO daily for 5 days and tapered over 2–4 weeks
- **Lithium carbonate:** One of the first drugs used for prophylactic treatment of chronic cluster headache. Blood levels must be monitored and thyroid function tests must be followed; concern that in athletes, dehydration may cause increase in lithium levels; generally only used when other drugs are ineffective or contraindicated
- **Calcium channel blockers:** They inhibit the initial vasoconstrictive phase of cluster headaches. Verapamil is most often used and is the drug of choice for prophylaxis against cluster headaches in athletes. Verapamil 120–360 mg daily divided into two, three, or four doses; one-time daily dosing may be achieved with use of extended-release tablets.
- **Anticonvulsants:** Valproate and topiramate may play a role in nonpharmacologic treatment of cluster headaches.

## SPORTS-/EXERCISE-RELATED PRIMARY HEADACHES

Athletes make up a special subset of the headache-suffering population. This is because they may suffer from specific types of headaches that are the direct result of their participation in their sport.

**BOX 39.4 IHS CRITERIA FOR PRIMARY EXERCISE HEADACHES**

**A.** At least two headache episodes fulfilling criteria B and C
**B.** Lasting for <48 hours
**C.** Brought on by and occurring only during or after physical exertion
**D.** Cannot be attributed to another disorder

Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

### Exercise-/Effort-Induced Migraine

- **Definition:** Exercise-/effort-induced migraines are headaches that fit the criteria for migraine headache (primarily in that they are severe, unilateral, throbbing, and last for 4–72 hours) and are triggered by exercise. Those who experience exercise-/effort-induced migraines usually have a history of nonexercise-related migraines.
  - In a previous study, high-intensity bicycling for a duration of 30 seconds was shown to precipitate a typical migraine 4.5–5.5 hours after the cessation of the exercise in women with a previous history of migraine.
- Headache occurs after activity and may be with or without aura.
- Activities that usually trigger the migraines are aerobic, such as running and swimming.
- Dehydration, excessive heat, hypoglycemia, and hypomagnesemia are predisposing factors.
- Elevation of nitric oxide, which has been shown to increase during exercise and is responsible for causing central vasodilation and subsequent increased central nervous system sensitivity, has been proposed as a potential cause of these headaches.
- **Treatment:** Responds to the same prophylactic and abortive treatments as ordinary migraine. A slow warm-up regimen has been recommended as an adjunct in the prevention of exercise-/effort-induced migraines.

### Primary Exercise Headache

- **Definition:** Primary exercise headaches (PEHs) are headaches that are precipitated by any form of exercise in the absence of any intracranial disorder. This IHS classification combines two headache entities that were previously well known to the sports medicine community as "benign exertional headache (BEH)" and "effort headache."
  - BEHs are thought to arise from brief, strenuous physical exercise that involved increasing intrathoracic pressure and performing Valsalva maneuvers (e.g., weightlifting, sprinting, and specific swimming events). These headaches are brief in duration.
  - Effort headaches are thought to arise from longer aerobic exercise activity common to a multitude of sports. These headaches typically have more migrainous features.
- This type of headaches was first described by Tinel in 1932 and termed "la cephalee a l'effort."
- PEH is the most commonly diagnosed headache type in athletes.
- PEH occurs in trained athletes and in those who exercise infrequently.
- IHS diagnostic criteria for primary exertional headache are listed in Box 39.4.
- This type of headache often occurs with exercise in hot weather or higher altitude.

- Thorough evaluation should be conducted before diagnosing exertional headache because it may not always arise from primary causes.
  - An early study in 1968 followed 103 patients diagnosed with BEH; 10 eventually had underlying organic disease.
  - In another series of 221 patients with brain tumor, 60% had headaches, and 22% of these had headaches that worsened with cough and exertion; in 18%, pain increased with stooping or bending over.
  - There have also been case reports of spontaneous cerebrospinal fluid leaks causing headache with Valsalva being misdiagnosed as BEH.
- **Treatment:** May be prevented by limiting the amount of exercise and controlling the exercise environment; most primary exertional headaches respond to NSAID therapy. Indomethacin can be used prophylactically, taken in 25- to 50-mg doses 1–2 hours before exercise. Moreover, twice or thrice daily dosing of 50–150 mg of indomethacin may be used to abort such headaches in those who have begun to have frequent episodes.

## SPORTS-/EXERCISE-RELATED PRIMARY HEADACHE PRETENDERS

- There are several conditions that may present as primary exercise-related headaches. An intuitive physician will at least be cognizant of this fact and will take into account all the differentials before making the final diagnosis.
- **Cardiac cephalalgia:** Case reports describe these as headaches that begin with vigorous exercise and are relieved by rest. The severity of the headache may increase as the intensity and duration of the exercise increases.
  - Headache is caused by cardiac ischemia.
  - Neurologic evaluation is normal.
  - Diagnosis is made using exercise stress testing. The onset of the patient's headache may correlate with ECG changes indicative of myocardial ischemia.
  - Headache may improve with nitroglycerin treatment (vasodilator) and worsen with triptan treatment (vasoconstrictor), which is in marked contrast to primary exercise-related headaches such as exercise-induced migraine that will likely worsen with nitroglycerin and improve with triptan treatment.
  - **Treatment:** Myocardial revascularization has been shown to lead to complete resolution of such headaches.
- **Other differential diagnoses of exercise-related primary headaches**
  - Intracranial: brain tumors, subacute and chronic subdural hematomas, and vascular malformations
  - Craniospinal abnormalities: platybasia, Arnold–Chiari malformation, and basilar impression
  - Metabolic disorders: pheochromocytoma, Cushing's disease, myxedema, thyrotoxicosis, and hypoglycemia of any etiology
  - Neurologic diseases: hydrocephalus, chronic central nervous system infections, and multiple sclerosis

## SPORTS-/EXERCISE-RELATED SECONDARY HEADACHES

### Acute and Persistent Posttraumatic Headache

- **Definition:** PTH is any form of headache (migraine, TTH, or cluster) that occurs after a known head trauma. They are categorized by the IHS as either acute or persistent and also as either occurring from mild or moderate/severe head trauma.
  - Acute PTH occurs within 7 days after the head trauma and resolves within 3 months.
  - Persistent PTH also occurs within 7 days after trauma to the head but persists for >3 months.

**TABLE 39.3 IHS DEFINITION OF MODERATE/SEVERE VS. MILD HEAD TRAUMA**

| Moderate/Severe Head Trauma | Mild Head Trauma |
|---|---|
| At least 1 of the following:<br>1. LOC >30 min<br>2. Glasgow Coma Scale <13<br>3. Posttraumatic amnesia for >24 h<br>4. Alteration in level of awareness for >24 h<br>5. Imaging shows traumatic head/brain injury | Having **NONE** of the characteristics of moderate/severe head trauma (1–5) and associated with one or more of the following symptoms occurring immediately following the head injury:<br>1. Transient confusion, impaired consciousness, or disorientation<br>2. Memory loss for events occurring immediately before or after the trauma<br>3. ≥2 symptoms consistent with MTBI (nausea, vomiting, visual disturbances, dizziness, and memory/concentration impairment) |

*LOC,* Loss of consciousness; *MTBI,* mild traumatic brain injury.
Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

- When PTH does not occur in isolation, but instead is accompanied by additional symptoms including difficulty balancing, inability to concentrate, memory problems, fatigue, dizziness, emotional lability, and insomnia, the athlete may be suffering from postconcussion syndrome.
- IHS definitions of mild and moderate/severe head trauma are enlisted in Table 39.3.
- Headache is the most common neurologic symptom after head trauma; it persists for >2 months in 60% of patients.
- Location, severity, and pain characteristics vary considerably. Most studies have shown that PTH is *less* common when the head trauma is *more* severe.
- TTH is the most common PTH.
- Women are at a higher risk of developing PTH.
- In a study of 443 high school and collegiate football players, 85% reported developing headache as the direct result of hitting.
- **Etiology** is controversial: both organic and psychogenic theories exist.
  - May be related to axonal injury or excitotoxic amino acid release, which results in neural injury
  - May be related to altered cerebral hemodynamics and/or slowed cerebral circulation
- **Posttraumatic migraine (footballer's migraine)** has been noted in sports such as boxing and soccer, where taking blows to the head or repetitively heading the ball causes migraine.
  - Does not require significant trauma; not associated with significant amnesia
  - After symptom-free interval of several minutes, visual, motor, sensory, or brainstem signs and symptoms begin (scintillating scotoma, flashing lights, blurred vision, or other sensory symptoms); last for 15–30 minutes
  - Subsequently, severe throbbing headache develops, associated with nausea and vomiting
  - Predisposing factors include history of nonsports-related migraine and family history of migraine (60% have a parent with migraine, 70% have a parent or sibling with migraine, 77% have a parent, sibling, or grandparent with migraine).

- **Important to rule out an organic basis for symptoms**
  - Do not return athlete to play if he or she remains symptomatic.
  - Immediate concerns include second-impact syndrome, vascular abnormalities, and other neurologic disorders.
  - **Intracranial bleeds that cause PTH** include epidural hematoma, subdural hematoma, and subarachnoid hemorrhage.
- **Treatment:** Once an organic cause is ruled out, treatment depends on the type of headache pattern (migraine, TTH, or cluster). Acute PTH that is nonmigrainous in nature and occurs within hours of a trauma should be treated in the short term with non-NSAID analgesics. This is done to prevent worsening of an intercranial bleed by the antiplatelet effect of the NSAID in the event an intercranial bleed has occurred.

### Cervicogenic Headache

- **Definition:** A cervicogenic headache is a headache that is caused by abnormalities within structures of the cervical spine, including the muscular, ligamentous, nervous, vascular, articular, and discogenic elements. It is usually, but not always, accompanied by neck pain.
- Pain typically starts in the neck and spreads to the oculofrontotemporal area. Pain is "dull" or "boring" in nature and fluctuates in intensity.
- Headache is constant and may last from hours to weeks.
- Often accompanied by restricted range of motion of the neck, and by ipsilateral, nonradicular, neck, shoulder, or arm pain
- Often seen in response to moderate/severe head trauma
- Affects women more than men with a female-to-male ratio of 4:1
- IHS diagnostic criteria for cervicogenic headache are listed in Box 39.5.
- **Treatment:** Physical therapy has been shown to provide the most long-term relief of cervicogenic headache. This may include cervical traction, massage, and strengthening. Anesthetic blockade may provide temporary relief but is not a long-term solution. Those who are provided relief with blockade but are not helped by physical therapy may benefit from radiofrequency neurotomy.

### High-Altitude Headache

- **Definition:** Headache that occurs with ascent to altitudes above 2400–3000 meters and is caused by hypoxia. They are dull and are of mild to moderate intensity. They typically develop within 6–24 hours of ascent and resolve within 8 hours after descent.
- Headache is the primary symptom of acute mountain sickness (AMS).
- 20%–50% of skiers and mountaineers experience headache at altitudes of 3000–5000 meters.
- **Treatment:** Primary treatment is descent to a lower altitude. High-altitude headaches may be prevented by allowing appropriate acclimation to one altitude before climbing to a higher altitude. Pharmacologic treatments include:
  - **Pretreatment with aspirin:** Raises headache threshold; associated with less pronounced cardiorespiratory response to short-term exercise at altitude; can prevent headache
  - **Ibuprofen:** More effective than placebo in altitude-related headache. In randomized, double-blind crossover study, ibuprofen 600 mg was effective in treating headache, whereas sumatriptan 100 mg was ineffective.
  - **Prophylaxis:** Acetazolamide 250 mg PO twice daily or 500 mg PO once daily with use of extended-release tablets; prophylaxis should be initiated on the day before the climb

### Diving Headache

- **Definition:** Headache that is caused secondary to hypercapnia and most readily associated with deep-water diving; carbon dioxide builds up in divers who practice "skip breathing," which is the act of intentionally holding respiration to conserve air or affect buoyancy in the water.
  - Hypercapnia causes cerebrovascular vasodilation and elevated intracranial pressure, which leads to headache.
  - Diving headache is nonspecific in character, but migrainous-type headaches may occur.
  - Occurs at depths below 10 meters
  - Worsens during resurfacing or decompression phase of the dive
- **Treatment:** Taking deep, slow breaths may help prevent these headaches from occurring. Treatment with 100% oxygen will usually resolve these headaches within 1 hour.

**BOX 39.5 IHS CRITERIA FOR CERVICOGENIC HEADACHE**

**A.** Any headache fulfilling criteria C
**B.** Clinical, laboratory, or imaging evidence of a disorder within the cervical spine or soft tissues of the neck, which is known to cause headache
**C.** Evidence that the pain caused by the neck disorder is based on at least two of the following:
  **1.** Headache has developed in temporal relation to the onset of the cervical disorder
  **2.** Headache improves or resolves with the improvement or resolution of the cervical disorder
  **3.** Cervical range of motion is reduced and headache worsens with provocative maneuvers
  **4.** Headache abolished with diagnostic blockage of the cervical structure or its nerve supply
**D.** Cannot be attributed to another disorder

Modified from Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. 3rd edition (beta version). *Cephalalgia.* 2013;33(9):629-808.

## SPORTS-/EXERCISE-RELATED CRANIAL NEURALGIA HEADACHES

### External Compression Headache

- **Definition:** Headache that results from continuous stimulation of the cutaneous nerves of the head. The continuous nerve stimulation is caused by externally applied pressure. This pressure usually results from wearing tight headbands, swim goggles, or diving masks.
- Headache is dull and increases in intensity as duration of compression increases.
- May contribute to the development of diving headache
- **Treatment:** Removal of the compressing device (headband, mask, or swim goggles) results in complete resolution of the headache.

### Cold-Stimulus Headache

- **Definition:** Headache that occurs after exposing unprotected head to cold temperatures (e.g., diving in cold water)
- Headache is dull and diffuse.
- May contribute to the development of diving headache
- **Treatment:** Headache resolves with removal of the cold stimulus.

**TABLE 39.4 DRUGS OF CHOICE FOR HEADACHES IN ATHLETES**

| | Tension Headache | Migraine Headache | Cluster Headache |
|---|---|---|---|
| **Abortive Treatment** | Acetaminophen<br>NSAIDs | Sumatriptan<br>Other 5-HT agonists<br>DHE-45 (nasal)<br>Intranasal lidocaine | Oxygen<br>Sumatriptan<br>DHE-45 |
| **Prophylactic Treatment** | NSAIDs<br>TCAs at bedtime | Verapamil<br>Valproate, topiramate<br>TCAs at bedtime | Verapamil |

*DHE,* Dihydroergotamine; *NSAIDs,* nonsteroidal anti-inflammatory drugs; *TCAs,* tricyclic antidepressants.

## CHARACTERISTICS OF PRIMARY HEADACHE DISORDERS

See Table 39.1.

## DRUG CHOICES FOR HEADACHES IN ATHLETES

See Table 39.4.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


1. Headache Classification Subcommittee of the International Headache Society (IHS). The International Classification of Headache Disorders. *Cephalalgia*. 3rd edition (beta version). 2013;33(9):629-808.
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12. Williams SJ, Nukada H. Sport and exercise headache: Part 2. Diagnosis and classification. *Br J Sports Med*. 1994;28(2):96-100.

# SKIN PROBLEMS IN THE ATHLETE

# 40

R. Robert Franks Jr.

## GENERAL PRINCIPLES

- Skin infections account for 21% of illnesses and injuries reported in collegiate and 8.5% in high school sports.
- In collegiate sports, skin infections account for 1%–2% of all time-loss injuries.
- Common dermatologic lesions and examples are listed in Table 40.1.

### Pathophysiology

- Increase in methicillin-resistant *Staphylococcus aureus* (MRSA) transmission is of concern in the contact sports of wrestling, fencing, and football.
- Other etiologies for skin infections include other bacterial, viral, and fungal infections. Other skin issues apart from infections can occur in sports.
- While the overall incidence of skin infections is low, prevention is possible.
- Skin lesions are most often transmitted from person to person but can also be from fomites.
- Risk factors for skin infections include:
  - Previous history of infection
  - Compromised host immunity
  - Close contact sports
  - Poor personal hygiene
  - Body shaving
  - Antibiotic overuse
  - Sharing of towels, razors, uniforms, and equipment
  - Facility and equipment cleanliness

### Methods to Reduce Infection Transmission

- History of past infections and treatment should be noted.
- Routine skin checks are recommended in all sports, particularly in high-risk sports (e.g., wrestling, football, and rugby).
- Infection spread can be decreased by withholding infected athletes from practice and/or competition until completely treated.
- Good hygiene should be promoted with immediate bathing after practices/games and trimming of nails.
  - Wash uniforms, towels, and equipment immediately after their use.
  - Instruct athletes to dry the area of any skin lesion last and discard the towel.
  - Regularly wash hands with soap and water or an alcohol-based hand gel.
  - Avoid sharing of equipment, uniforms, towels, clothing, bedding, bar soap, razors, and toothbrushes.
- Wound coverage as the primary treatment or prevention of spread of a skin lesion is never appropriate.
- Skin lesions should be covered only after they are noninfectious to prevent secondary infection. Consider prophylactic medication for those with frequent outbreaks.
- Equipment and other surfaces that multiple athletes have been having contact with should be frequently cleaned.
- Student-athletes should be encouraged to immediately report skin wounds and lesions.
- Education of coaches, officials, and healthcare practitioners regarding common skin lesions should be conducted at least annually.
- The efficacy of treating potential MRSA carriers in order to prevent spread of infection remains inconclusive.

## VIRAL INFECTIONS

### Herpes Gladiatorum (Traumatic Herpes)

**Overview**: Caused by the herpes simplex virus (HSV); HSV is common in the general population as well as among athletes, particularly wrestlers. Herpes consists of 2 types, HSV-1 or HSV-2, but can be mixed and found anywhere on the body. HSV-I is associated with cold sores and fever blisters. HSV-2 is associated with genital herpes. Herpes gladiatorum is caused by HSV-1. In a typical season, 2.6% of high school wrestlers and 7.6% of college wrestlers will suffer from herpes gladiatorum; it spreads skin to skin via an active rash or rash fluid, from fomites to skin, or by respiratory droplets or saliva. Breaks in the skin barrier can increase the risk of infection. Stress, either physical or emotional, and sun exposure can increase the likelihood of infection. The virus incubation period is typically 2–14 days.

**Presentation:** One in four infected will begin with flu-like symptoms approximately 2–24 hours before appearance of rash. Rashes may have a prodrome of itching or burning. In wrestling, common appearance of the rash is usually on the right side of the wrestler's face, neck, or arm, which are points of most contact with the mat. The rash disappears in 2 weeks without scarring.

**Physical examination:** The rash begins as a burning or tingling sensation on the skin, which is already in its contagious phase. This area then becomes the site of red bumps that form clusters of tiny blisters filled with cloudy fluid on an erythematous base. The blisters collapse to form yellowish-brown scabs, which form in 2–4 days.

**Diagnosis:** The rash is usually characteristic, but a Tzanck smear can be performed in questionable cases. Viral cultures can also be performed to confirm diagnosis and type (HSV-1 vs. HSV-2).

**Treatment:** Treatment is with oral antivirals for 7–10 days for initial infections. Consider valacyclovir (Valtrex) 1 gm BID for 7–10 days. Treatment for recurrent infections should be valacyclovir 1 gm QD for 5 days. Consider suppression dosing for those with chronic recurrent infections approximately six times per year. The drug of choice would be valacyclovir 1 gm QD. Acyclovir can be used in place of the above but resistance does exist. The initial treatment dose is 400 mg TID for 7–10 days. The dose for recurrence is 400 mg TID for 5 days, and the suppression dose is 400 mg BID. Famciclovir can also be considered if there is resistance to both acyclovir and valacyclovir. Topical antivirals are often not effective if used alone.

**Return to play:** Varies by sport but most often is after a course of 5 days of oral antiviral treatment with no new systemic symptoms or lesions for 72 hours; all lesions must be dry and crusted

### Herpes Labialis (Fever Blister, Oral Herpes, and Cold Sores)

**Overview:** Caused by HSV-I and is spread skin to skin or by fomites (Fig. 40.1); the infection is seen around the lips,

## TABLE 40.1 DESCRIPTION OF COMMON DERMATOLOGIC TERMS

| Terminology | Definition | Example |
|---|---|---|
| Macule | Nonraised change of skin color <1 cm | Vitiligo<br>Petechiae |
| Patch | Nonraised change of skin color >1 cm | Vitiligo<br>Pityriasis |
| Papule | Raised skin change with defined borders <1 cm in diameter that comes in a variety of shapes, i.e., domed, umbilicated | Measles<br>Acne vulgaris |
| Nodule | Raised skin change >1 cm in diameter appearing in epidermis, dermis, or subcutaneous tissue | Warts<br>Squamous or basal cell carcinoma |
| Tumor | Solid mass within skin or subcutaneous tissue greater than a nodule | Lipoma<br>Seborrheic keratosis |
| Plaque | Raised, solid lesion >1 cm in diameter on skin surface | Psoriasis<br>Mycosis fungoides |
| Vesicle | Raised fluid-filled lesions <1 cm in diameter appearing on skin surface | Herpes simplex<br>Herpes zoster |
| Bulla | Larger fluid-filled lesion >1 cm on skin surface with true circumscribed border | Blisters<br>Pemphigus |
| Pustule | Elevated lesion on skin surface containing pus with circumscribed border<br>Vesicles can become pustules<br>Fluid may be infectious or noninfectious | Acne vulgaris<br>Impetigo |
| Wheal | Area of edema found in epidermis | Urticaria<br>Bee or wasp sting |
| Burrow | Linear change in skin due to tunnel formation by skin infestation | Scabies |
| Telangiectasia | Prominent, permanent dilation of superficial blood vessels in skin | Osler–Weber–Rendu disease<br>Ataxia-telangiectasia |
| Ulcer | Open, crater-like lesion of epidermis or mucus membranes | Decubitus ulcer<br>Aphthous ulcer |
| Lichenification | Elevated lesion containing proliferation of keratinocytes and stratum corneum due to continued skin irritation | Eczematous dermatitis |
| Scales | Peeling or flaking skin areas due to abnormal formation of stratum corneum due to increased production of epidermal cells | Psoriasis<br>Eczema |
| Crusts | Skin change due to dried serum, blood, or purulence | Impetigo<br>Herpes simplex |
| Atrophy | Thinning or absence of epidermis or subcutaneous fat | Steroid-induced atrophy<br>Scleroderma |
| Erosion | Depressed skin area where epidermis is partially or completely removed | Infection<br>Trauma |
| Excoriation | Loss of skin from scratching, friction, or rubbing | Chronic hepatitis C<br>Skin picking disorder |
| Fissure | Linear skin break that leads into the dermis | Dry skin<br>Trauma (scratches) |
| Scar | Fibrotic changes to skin due to dermal damage<br>Change of pigment is often associated with scars | Surgery<br>Trauma |
| Eschar | Hard, darkened plaque covering ulcer with significant tissue necrosis | Burns<br>Pressure wounds |
| Keloids | Extensive connective tissue response to skin injury that is larger than original wound | Surgery<br>Trauma |
| Petechiae | Smaller bleeding skin lesion. Does not blanch when pressed | Strep throat<br>Vasculitis |
| Purpura, ecchymosis | Larger bleeding skin lesion<br>Does not blanch when pressed<br>Purpura may be palpable | Hemangiomas<br>Senile purpura |

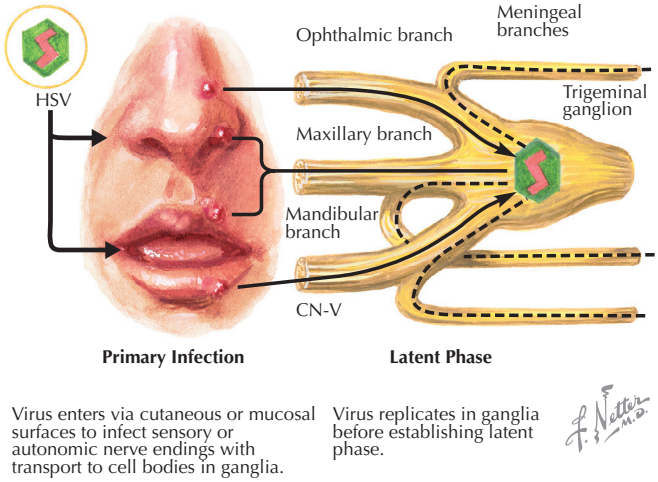



**Figure 40.1.** Herpes simplex.

beginning as vesicles on an erythematous base that break and then crust over. The virus can express itself during times of physical or emotional stress or from environmental factors such as direct sunlight.

**Presentation:** Begins with prodrome of pain or pruritus at the site of lesion, followed by clear vesicles on an erythematous base that eventually break and crust over. The athlete becomes infectious during the prodrome and is infectious until the lesions crust. Infection course is generally 7–14 days.

**Physical examination:** Clear vesicles on an erythematous base around the lip and possibly between the nose and lip that evolve into areas of ruptured vesicles on an ulcerated base.

**Diagnosis:** Characteristic rash but viral culture is definitive

**Treatment:** Treatment includes oral antivirals: consider valacyclovir 2 gm BID for 1 day, which should be started as soon as the athlete experiences the viral prodrome.

**Return to sport:** Varies by sport

Available literature is more consistent for return to sport with herpes gladiatorum, but in most recommendations for return with herpes labialis, the lesions should be crusted over and dry before return.

### Herpes Zoster (Shingles)

**Overview:** Reactivation of the herpes virus expressed in the dermatome of the infected nerve root. Reactivation is usually preceded by a pain prodrome along the same root usually initiated by either physical or emotional stress. Vaccination may decrease the risk of developing postherpetic neuralgia.

**Presentation:** Usually begins with a painful prodrome, followed by infection along the affected nerve root

**Physical examination:** Initial infection is papules changing to vesicles, then pustules in a dermatomal pattern before crusting over during the course of a week. Regional lymph glands are often swollen and tender.

**Diagnosis:** Characteristic dermatomal rash but viral culture is diagnostic

**Treatment:** Via oral antivirals: consider valacyclovir 1 gm every 8 hours for 7 days and pain medications as soon as prodromal symptoms occur; physicians must be cautious not to prescribe banned substances for pain

**Return to sport:** Varies by sport, but most often is after a course of oral antiviral treatment with no new systemic symptoms or lesions for 72 hours; all lesions must be dry and crusted

### Molluscum Contagiosum (Water Warts)

**Overview:** Viral lesion that spreads skin to skin or via fomites; characterized by single or multiple discreet, flesh-colored, dome-shaped papules with a central dimple. The rash most often occurs in contact athletes, on the face, trunk, or hands.

**Presentation:** Athletes have these lesions, painless and nonpruritic, in clusters for several weeks to months and often only present when their spread is noticed.

**Physical examination:** Clustered, dimpled papules found on face, trunk, or hands

**Diagnosis:** Characteristic rash but also identified by observation under microscope or biopsy

**Treatment:** Mechanical via curettage, electrotherapy, or cryotherapy; chemical and immunologic methods can also be used

**Return to sport:** Data inconclusive; often lesion removal and 48 hours of recovery to prevent secondary infection

### Verruca Vulgaris/Plantaris (Common/Plantar Warts)

**Overview:** Verrucous plaques and papules found most often on the epidermis of the hands due to the human papilloma virus. They can, however, be seen anywhere on the body. Warts on the plantar surface of the athlete's foot, most often the heel or ball, can cause a change in athlete's ambulation.

**Presentation:** Painless papules or plaques most often on hands, present for weeks to months; athletes usually try self-removal before presentation via over-the-counter preparations, cutting, scraping, or picking

**Physical examination:** Seen anywhere but usually on hands; papules with a typical "cauliflower" appearance

**Diagnosis:** Distinguished from callouses by the presence of hemorrhages seen when the wart is shaved; no normal skin lines in the wart

**Treatment:** Removed by excision, cryotherapy, or use of salicylic acid; immunotherapy is also successful in wart resolution. Treatments may have to be repeated. Care must be taken not to permanently damage or destroy skin with continued attempts at removal. If a wart is present in a sensitive area, paring and covering may be used in-season with definitive treatment after the season. Warts should be covered; low risk of transmission. The wart can be protected by use of doughnut pads or moleskin until definitive treatment can be administered. Use of footwear in locker rooms and shower areas can prevent transmission.

**Return to sport:** No restrictions

## BACTERIAL INFECTIONS (Fig. 40.2)

### MRSA

**Overview:** Rapidly spreading bacterial infection characterized by *Staphylococcus aureus* that is resistant to several antibiotic classes; the most common strain seen in athletes is community-acquired (CA) MRSA species. Spread is via skin to skin contact, fomites, crowding, poor hygiene, scratching, and compromised skin integrity.

**Presentation:** Most often confused with spider bites or simple pustules

**Physical examination:** Infection begins with a small pimple-like lesion that quickly progresses to a hot, erythematous, inflamed, painful indurated area much larger than the original lesion; can occur on any part of the body

**Diagnosis:** Challenging as the infection looks like other common infections; bacterial culture is diagnostic

**Treatment:** Treatment should begin, if suspected, before the culture results are identified. Treatment is via incision and drainage and IV antibiotics such as vancomycin for serious lesions. Minor or

**Figure 40.2.** Common bacterial infections.

less serious lesions are treated with oral antibiotics for 14 days. Consider trimethoprim with sulfamethoxazole (Bactrim) 2 DS BID for 14 days or doxycycline 100 mg BID for one day and then 100 mg QD for 13 days, unless an athlete will be spending time in direct sunlight; prevented by not sharing uniforms, towels, or equipment. Area of infection should be dried last after showering, and the towel must be immediately laundered. Treatment of chronic carriers is controversial.

**Return to sport:** After complete antibiotic course with wounds dried and crusted and formation of no new lesions

## Impetigo

**Overview:** Caused by beta-hemolytic *Streptococcus* or *Staphylococcus aureus*

Infection usually begins as small red blisters on erythematous skin. Blisters then break, forming characteristic honey-colored scabs that may be pruritic. If left untreated, impetigo can cause renal damage. Infection usually resolves within 2 weeks without scarring. Spread is either skin to skin or via fomites; can spread by scratching or drying after shower

**Presentation:** Often begins as a small lesion; often, if initial rash begins around the mouth, it can be confused with herpes labialis

**Physical examination:** Most often seen on face or forehead but can occur anywhere

**Diagnosis:** Characteristic rash but bacterial culture is definitive

**Treatment:** Small areas may be treated with antibiotic creams or ointments

Consider mupirocin (Bactroban) TID for 7–14 days. Oral antibiotics should be used for larger areas. Consider a first-generation cephalosporin, erythromycin, or clindamycin. Treatment is usually for 7–14 days. Consideration should be given to immediate treatment for MRSA, if appropriate. Infection often prevented by not sharing uniforms, towels, or equipment. Area of infection should be dried last after showering and the towel immediately laundered.

**Return to sport:** Return is not advisable until all blisters have crusted and 3 days of antibiotic treatment has been completed without new lesions for 48 hours before competition

## Cellulitis

**Overview:** Infection of the dermis and subcutaneous tissues usually by group A *Streptococcus* and/or *Staphylococcus aureus*

Cellulitis most often occurs after a break in the skin such as a laceration, abrasion, or puncture wound (Fig. 40.2); can spread skin to skin or by fomites

**Presentation:** Often begins with a gradual onset of pain and swelling that may or may not be associated with a recent break in the skin

**Physical examination:** Presents initially as a painful erythematous swollen patch that spreads as the infection spreads; the infection can be associated with fever and chills

**Diagnosis:** Characteristic rash of an erythematous, edematous patch; wound and/or blood cultures are diagnostic

**Treatment:** Oral antibiotics, and if severe, IV antibiotics

Oral antibiotics can include dicloxacillin, cephalexin, clindamycin, or a macrolide. Warm compresses, pain medications, and incision and drainage can be helpful. Marking location and looking for spread beyond the line of demarcation can be suggestive of antibiotic resistance. Consideration should be given to immediate treatment for MRSA, if appropriate.

**Return to sport:** Antibiotic treatment, afebrile, cessation of pain, and healing of wound

## Folliculitis (Occlusive Folliculitis, Steroid Folliculitis, Bikini Bottom)

**Overview:** Characterized by red-ringed papules, pustules, or crusts at hair follicles most commonly caused by *Staphylococcus aureus*, *Candida*, or *Pseudomonas aeruginosa*, demodex (Fig. 40.2). Steroids may also cause folliculitis. Infection may evolve to boils or carbuncles.

**Presentation:** Seen in areas of body that have been shaved or areas with friction from equipment or uniforms; spread is via skin to skin or fomites

**Physical examination:** Seen in areas of friction such as groin, thighs, buttocks, or gluteal folds

**Diagnosis:** History and characteristic rash but bacterial culture is diagnostic

**Treatment:** Removal of irritant, topical antiseptics, antibiotic creams or ointment, and good hygiene; significant cases require oral antibiotics
**Return to sport:** No restriction with lesion coverage

### Pseudomonas Folliculitis (Hot Tub Folliculitis)

**Overview:** Folliculitis seen in athletes who use hot tubs or whirlpools caused by *Pseudomonas aeruginosa* type 0:11
**Presentation:** Appears on areas of skin submerged in the water, particularly the axilla and the groin; systemic symptoms, including fever, chills, lymphadenopathy, and fatigue, may occur. Rash duration is usually 7–10 days.
**Physical examination:** Small erythematous papules that may convert to green pustules seen in the axilla or the groin
**Diagnosis:** Characteristic rash but definitive diagnosis via bacterial culture
**Treatment:** Often self-limiting
Oral antibiotics should be considered for patients who have systemic symptoms. The affected hot tubs and whirlpools should be drained and cleaned with discovery of infection.
**Return to sport:** No restriction; cover with bio-occlusive dressing

### Furunculosis (Boil, Furuncle, Carbuncle)

**Overview:** Hair follicle infection by *Staphylococcus aureus* or *Streptococcus* from trauma or direct contact with a furuncle on another athlete (Fig. 40.2). Fomites can also spread infection.
**Presentation:** Begins as infection of hair follicle that often evolves to a loculated pustule
**Physical examination:** Usually on the upper extremities or trunk and present as erythematous nodules with or without purulence; often painful
**Diagnosis:** Lesion appearance; bacterial culture is diagnostic
**Treatment:** Purulent lesions should be incised and drained. Treatment is via topical or oral antibiotics; if suspicious, presumptive treatment for MRSA should be considered before culture results are obtained. Infection can often be prevented by not sharing uniforms, towels, or equipment. Area of infection should be dried last and the towel immediately laundered.
**Return to sport:** Absence of systemic symptoms and lesion coverage with a bio-occlusive dressing

### Acne Mechanica (Sports Acne, Football Acne)

**Overview:** Acne breakout including papules, pustules, comedones, nodules, and cysts that is caused by bacterial infection in an area of tight-fitting clothing or ill-fitted equipment such as helmets or shoulder pads (Fig. 40.2).
**Presentation:** The equipment causes erythema and friction at the site of contact with the skin. The site then often becomes infected.
**Physical examination:** Rash appears as erythematous papules and pustules on the back, shoulders, chin, or forehead.
**Diagnosis:** Characteristic rash in area of equipment/clothing; bacterial culture is definitive
**Treatment:** Topical antibiotics and astringents
More severe outbreaks can be treated with oral antibiotics. Good hygiene, wicking undergarments, and refitting of equipment can help prevent further breakout.
**Return to sport:** No restrictions

### Pitted Keratolysis (Tennis Shoe Foot)

**Overview:** Bacterial infection caused by *Kytococcus sedentarius*, *Dermatophilus congolensis*, *Corynebacterium*, *Actinomyces*, or *Streptomyces*. These bacteria secrete proteinases that destroy the keratin of the stratum corneum of the skin, producing depressed lesions and pits on the sole of the foot but can be on hands as well (Fig. 40.2).
**Presentation:** Infection in areas of macerated tissue from extended exposure to moist, warm conditions in nonbreathable shoes seen with cross-country runners or marathoners. Most cases exhibit no discomfort, but painful plaque-like lesions can cause pain. Patients may have foul-smelling infectious lesions for months before presentation.
**Physical examination:** The rash appears as scaly pits with associated foot odor.
**Diagnosis:** Characteristic rash or bacterial culture is definitive.
**Treatment:** Infection is treated with topical clindamycin or erythromycin. Benzoyl peroxide may be helpful. The infection can be prevented with good hygiene, synthetic socks, and allowing ventilation of the feet.
**Return to sport:** No restrictions

### Onychocryptosis (Ingrown Toenail)

**Overview:** Occurs when inappropriately cut nails or nails damaged from sport grow into the subcutaneous tissue causing infection by *Streptococcus* and *Staphylococcus aureus* (Fig. 40.2).
**Presentation:** Pain, inflammation, and erythema, followed by purulent drainage at the advancing nail edge
**Physical examination:** Seen on any toe
**Diagnosis:** Lesion appearance; bacterial culture is diagnostic
**Treatment:** Incision and drainage of the purulent pocket and trimming or cutting back of the nail; oral antibiotics, pain medications, and warm compresses also used; prevention by appropriate nail trimming and wearing of appropriately fitting footwear
**Return to sport:** Passing of functional testing

## FUNGAL INFECTIONS (Fig. 40.3)

### Tinea Pedis/Cruris (Athlete's Foot/Jock Itch)

**Overview:** Caused by the fungus *Trichophyton rubrum*, *Trichophyton mentagrophytes*, *Epidermophyton floccusum*, or *Candida*. Scaly erythematous rash on plantar surface of the foot, between the toes, or in the groin, accompanied by a burning or itching sensation (Fig. 40.3). In general, cases improve in 10–14 days and resolve in 4–6 weeks. Spread is by contact of skin with damp, warm conditions.
**Presentation:** Often seen in those with exposure to warm, damp conditions, in diabetics, and in immunocompromised individuals; athletes often present after trying over-the-counter medications; may spread from sharing of uniforms or towels
**Physical examination:** Infection is characterized by inflamed, flaky, red and white skin with tiny blisters and/or pimples. Skin may peel or crack, particularly between the toes in Pedis. In Cruris, it starts at crural folds and expands to thighs. Satellite lesions, i.e., erythematous bumps or blisters, may be seen at the leading edge of the rash or spreading out from the central rash.
**Diagnosis:** Characteristic rash but examination of KOH skin scraping under microscope or fungal culture is definite
**Treatment:** Mild cases are treated with over-the-counter antifungals. Consider Tinactin or Desenex powders. Moderate cases are treated with prescription topical antifungals. Consider Lotrimin AF or Nizoral. Severe cases are treated with oral antifungals. Consider itraconazole (Sporanox) or terbinafine (Lamisil). Monitoring of liver functions is necessary with extended treatment. Antihistamine or low-potency steroid creams may be used for 1–2 days to decrease inflammation and itching. Keeping toes dry and separated promotes healing. Prevented by wearing footwear in the locker room and shower areas, changing socks often, and allowing breathable sneakers to dry. Socks in Pedis should be put on before underwear.
**Return to sport:** No restrictions as feet and groin are covered

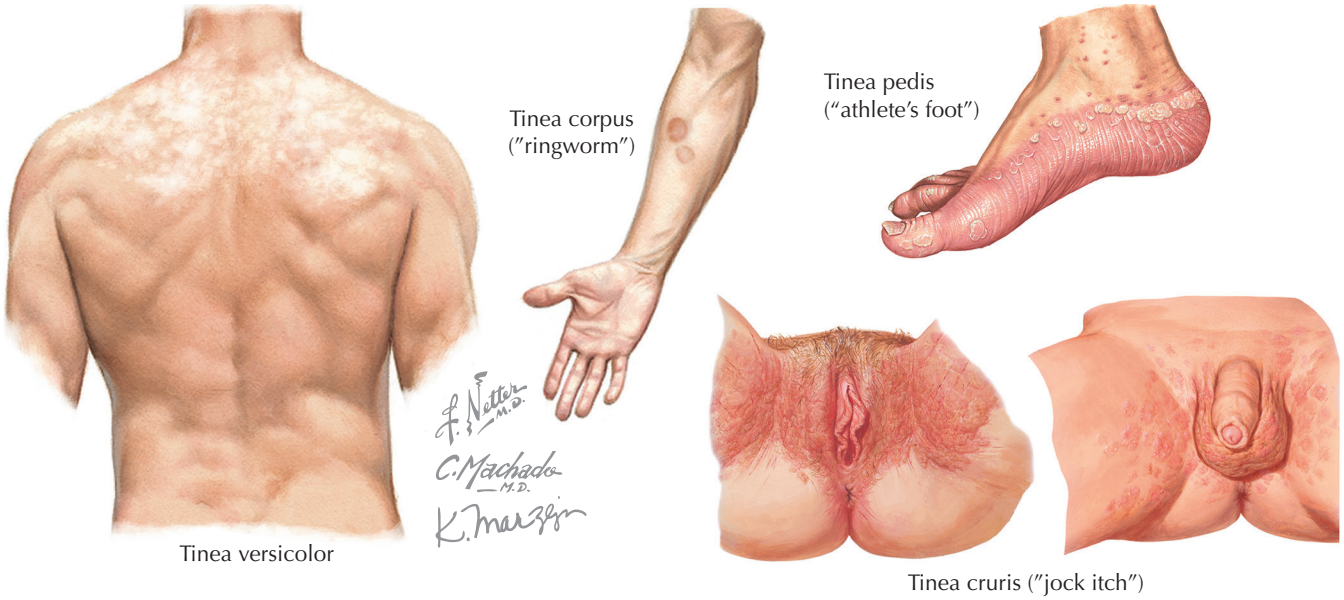



**Figure 40.3.** Common fungal infections.

## Tinea Corporis (Ringworm)

**Overview:** Caused by *Microsporum*, *Trichophyton*, or *Epidermophyton*; called *ringworm* due to its characteristic circular appearance (Fig. 40.3); spread is via fomites, skin to skin, or animals to humans

**Presentation:** The degree of rash is dependent on the host cellular-based immune response. Several athletes carry the spores but do not demonstrate the rash.

**Physical examination:** Most often seen on the torso, upper extremities, and head and neck. Rash begins as flat scaly spot in the shape of a small red or brown circle. It spreads as enlarging circle with a scaly border. There may be small papules, blisters, or scales on the leading edge. As it grows, the center fades to a lighter brown or red color. The athlete may have several areas of infection in differing degrees of development, with leading edges being the most reactive. The rash may be pruritic.

**Diagnosis:** Characteristic rash but examination of KOH skin scraping under microscope or fungal culture is definite

**Treatment:** Mild cases are treated with antifungal creams. Consider Lotrimin or Nizoral cream BID to TID for 2–4 weeks. More severe cases usually require oral antifungal agents. Consider itraconazole (Sporanox) 200 mg QD for 1 week or terbinafine (Lamisil) 250 mg QD for 1 week. Monitoring of liver functions is necessary with extended treatment. It can take up to 3 months for this rash to completely resolve. Consider prophylaxis for those with recurrent infection. Coverage of the rash alone is not sufficient to prevent its spread. Cleaning of mats, equipment, and clothing can decrease transmission.

**Return to sport:** Conflicting literature, but most recommend 72 hours of drug treatment with coverage with gas-permeable dressing until flaking stops.

## Tinea Capitis

**Overview:** Caused by the fungus *Trichophyton tonosurans*; spread is skin to skin or fomite and by contact with the fungus

**Presentation:** Lesions appear on the head and hair shafts.

**Physical examination:** Rash is characterized as a scaly, erythematous, pruritic plaques with raised ring-shaped borders. Hair loss is often present.

**Diagnosis:** Characteristic rash but examination of KOH skin scraping under microscope or fungal culture is definite

**Treatment:** Griseofulvin (Grifulvin) is standard treatment for Tinea Capitis. Treatment duration should be 6 to 8 weeks. Terbinafine (Lamisil) can used for 6 weeks as an alternative.

**Return to sport:** Extensive and active lesions require disqualification of the athlete.

## Tinea Versicolor

**Overview:** Fungal infection caused by *Pityrosporum orbiculare* or *P. ovale*; identified by hypopigmented or hyperpigmented areas on the skin (Fig. 40.3). Spread is skin to skin or fomite and by contact with the fungus.

**Presentation:** Rash usually begins at nape of neck and extends to arms or trunk.

**Physical examination:** Characterized by hypopigmentation at the site of infection, which can last even after the infection has been treated. Rash is neither painful or pruritic and often is found incidentally on physical examination.

**Diagnosis:** Observation of macular hypopigmented or hyperpigmented rash, but examination of KOH skin scraping under microscope or fungal culture is definite

**Treatment:** Topical or oral antifungals and selenium sulfide; good hygiene practices can often reduce the chances of infection

**Return to sport:** No restrictions

## Tinea Unguium (Onochomycosis)

**Overview:** Fungal infection of nails caused most often by *Trichophyton rubrum*. Can involve matrix, bed, or plate. Identified by scaling, thickened, opaque, yellow-brown nails. Spread is skin to skin or fomite and by contact with fungus. Can be on fingers but mostly seen on toes.

**Presentation:** Initial complaints concern appearance of nail. Evolves to difficulty with ambulation. Can be associated with pain, paresthesia, and decreased motion in affected toe. Appearance can lead to social withdraw and low self-esteem.

**Physical examination:** Exact examined appearance of the nail is determined by clinical subtype with distal lateral subungual onochomycosis most common.

**Diagnosis:** Characteristic appearing nail is diagnostic but examination of KOH scraping under microscope or fungal culture is definitive.

**Treatment:** Treatment options include topical Ciclopirox solution (Penlac) for mild to moderate cases. For more severe cases, oral anti-fungals including fluconazole, itraconazole, or terbinafine can be considered. Use of less occlusive footwear in sport and use of footwear in locker room is preventative.
**Return to sport:** No restrictions.

## INFESTATIONS

### Scabies

**Overview:** Infestation caused by *Sarcoptes scabiei* var. *bominis* mite; appear on the body as scattered groups of pruritic vesicles and pustules in runs or burrows (Fig. 40.4)
Transmission from skin to skin or fomite to skin.
**Presentation:** Intense pruritus at the affected area; the pruritus is often worse at night
**Physical examination:** Burrows are seen most often on the sides of the fingers, palms, wrists, elbows, knees, axilla, waist, groin, and ankles. Breasts, penis, and scrotum may also be involved.
**Diagnosis:** Identification of rashes/burrows is usually diagnostic. Microscopic examination of skin scraping yields mites, ova, and feces.
**Treatment:** Permethrin cream 5% (Elimite) and crotamiton cream 10% (Eurax, Crotan)
It is absolutely necessary to wash all uniforms, clothing, bathing items, and bedding. Consideration of treatment of teammates with close contact with athlete and athlete's household members should be considered. Treatment may have to be repeated.
**Return to sport:** After all living mites have been eliminated by treatment. This is usually achieved in 24 hours, although itching may last for up to 1 week. Re-treatment is often necessary. A negative scraping should be obtained before return to play.

### Pediculosis (Lice)

**Overview:** Parasitic infestation of the skin of the scalp, trunk, or pubic area by *Pediculosis humanus* (Fig. 40.4). Can be spread skin to skin or skin to fomites
**Presentation:** Intense pruritus may lead to deep excoriations over the affected area.
**Physical examination:** Presence of nits or egg sacs usually on the hair of scalp, trunk, or pubic area.
**Diagnosis:** The presence of nits or egg sacs attached to the hair close to the skin is diagnostic. Visible nits are easiest to see on head or at the nape of the neck.
**Treatment:** Head lice shampoo such as Nix or Rid. Treatment may have to be repeated. It is absolutely necessary wash all uniforms, clothing, bathing items, and bedding.
**Return to sport:** After removal and elimination of all nits

## MECHANICAL FRICTION INJURIES (Fig. 40.5)

### Blisters

**Overview:** Bullae that occur from repetitive friction in sports
**Presentation:** Seen commonly on feet and posterior calcaneus; formed from loose ill-fitting shoes or change in training pattern; most often seen early during a workout or early in a season
**Physical examination:** Bullae commonly found on posterior or plantar surfaces of foot but can occur anywhere friction occurs
**Diagnosis:** Characteristic bullae

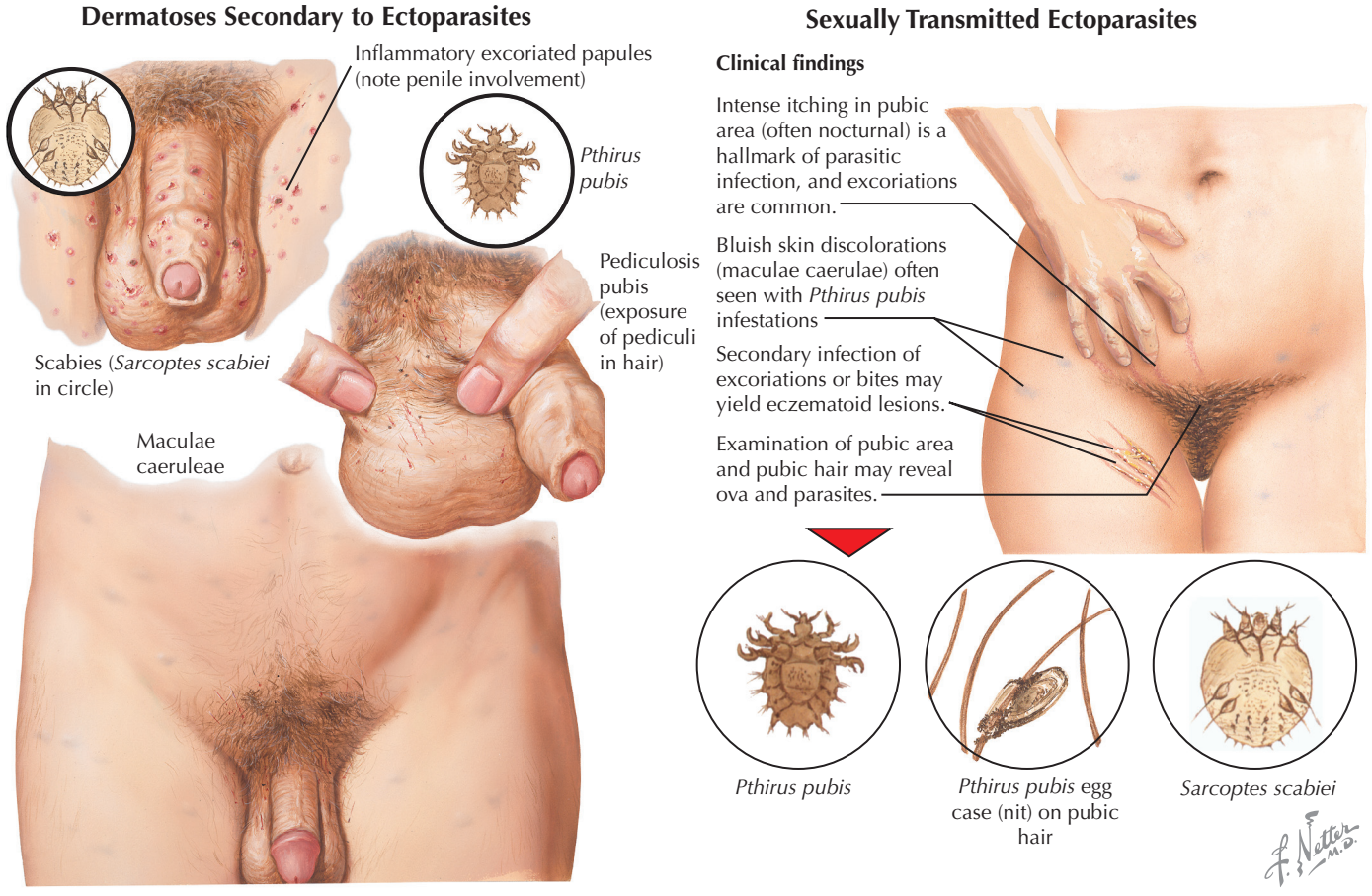



**Figure 40.4.** Scabies and pediculosis.

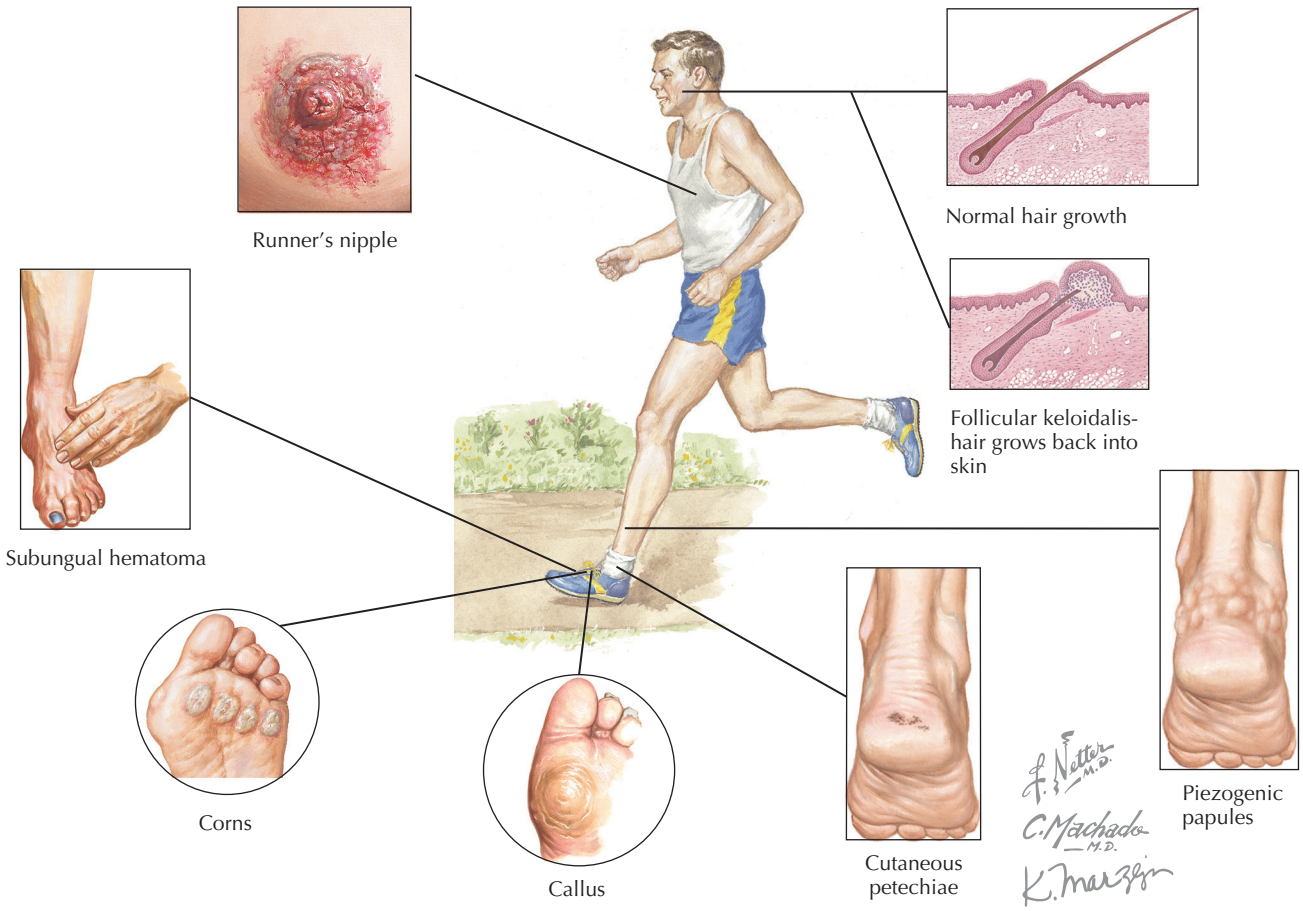



**Figure 40.5.** Common problems caused by friction and pressure.

**Treatment:** Blisters are best managed without breaking. They can be opened and drained if large or particularly painful. The goal of drainage is to allow adherence of separated tissue layers. The overlying tissue should be left intact. Use antibiotic creams if blister is open. Use of moleskin around the blister is protective. Use of two sets of socks, synthetic socks, and hydrocolloidal antifriction products can prevent blisters.
**Return to sport:** No restrictions

## Calluses (Tylomata)

**Overview:** Occur from repetitive trauma, which results in hyperkeratosis of the epidermis (Fig. 40.5); can occur from change in equipment or surface
**Presentation:** They are the most common keratosis in sports and protect the skin; most often not painful
**Physical examination:** Occur most often on the palms and feet
**Diagnosis:** Characteristic lesion most often over pressure points
**Treatment:** If painful, calluses can be debrided by a pumice stone, topical salicylic acid, or keratolytic emollients. Moleskin placed over a callous can decrease pressure and spread forces to other areas of the foot. Use of orthotics and change in footwear is often helpful.
**Return to sport:** No restriction

## Corns

**Overview:** Accumulations of keratin that develop over bony prominences or pressure points (Fig. 40.5)
**Presentation:** Most commonly seen on the toes
**Physical examination:** Keratin accumulation on bony areas of feet or toes
**Diagnosis:** Characteristic lesion
**Treatment:** Appropriately fitting footwear and padding may prevent the development. Symptomatic corns can be treated with debridement as described earlier.
**Return to sport:** No restrictions

## Talon Noir and Tache Noir (Plantar Petechiae, Black Heel, Black Dot Syndrome)

**Overview:** Horizontal petechiae seen on the posterior or lateral heel or palms
**Presentation:** Occur in stopping and starting sports on the feet and on the hands of gymnasts, golfers, and weightlifters; they are painless and resolve spontaneously
**Physical examination:** Horizontal petechiae on feet or hands; no additional bleeding with paring and skin lines are maintained
**Diagnosis:** Lesion itself is diagnostic; however, a skin scraping looking for occult blood is diagnostic.
**Treatment:** Use of appropriately fitting shoes and form fitting socks
**Return to sport:** No restrictions

## Piezogenic Papules

**Overview:** Small, often painful, fat papules on the outside aspect of the heels (Fig. 40.5)
**Presentation:** Flesh-colored and found in long-distance runners due to fat herniation through the dermis; often painful

**Physical examination:** Fat collection seen on the outside of the heels when pressure is applied; often not painful when foot is not weight bearing
**Diagnosis:** Lesion is diagnostic
**Treatment:** Silicone gel heel cups or orthotics may reduce the symptoms.
**Return to sport:** No restrictions

### Hematoma

**Overview:** Caused by separation of skin from underlying tissues by shear forces (Fig. 40.5)
**Presentation:** Most common is auricular hematoma seen from contact of the ear with a wrestling mat
**Physical examination:** Blood-filled bullae seen most commonly on a wrestler's ear
**Diagnosis:** Lesion is diagnostic
**Treatment:** When recognized, hematoma should be drained and splinted with a compression dressing to prevent development of a cauliflower ear.
**Return to sport:** After incision and drainage, allow with adhered compression dressing

### Abrasions (Road Rash, Turf Burn, Raspberry)

**Overview:** Occur by contact of skin with artificial turf, mats, or ill-fitting equipment
**Presentation:** Loss of epidermis and/or dermis with bleeding and weeping secondary to skin contact with a surface; chronic abrasions can lead to skin changes, hair loss, and callous formation
**Physical examination:** Skin of an abrasion is erythematous and may ooze serous fluid. Debris may be present in the wound.
**Diagnosis:** Lesion is diagnostic
**Treatment:** Area should be cleaned with soap and water and protected with antifriction lubricants. Nonstick padding with petroleum jelly or antibiotic ointment is therapeutic. Monitor for missed debris or infection.
**Return to sport:** No restrictions upon covering of abrasion

### Subungual Hematoma (Black, Runner's, Tennis, Skier's, Hiker's Climber's Toe)

**Overview:** Splinter hemorrhages often on the toes secondary to repetitive trauma (Fig. 40.5)
**Presentation:** Can also be caused acutely by being stepped on or cleated or by pressure from tight shoes or rapid deceleration
**Physical examination:** Presence of blood underneath nails of fingers or toes
**Diagnosis:** Presence of blood under nails of hands or feet is diagnostic
**Treatment:** Drainage using a needle through the nail is therapeutic. Padding can then be applied for protection. Appropriately fitting shoes and change of running technique can prevent subungual hematomas.
**Return to sport:** No restrictions

### Chafing

**Overview:** Denuding of epidermis due to mechanical contact between two opposing areas of the body
**Presentation:** Seen in areas of contact where skin meets skin or fabric meets skin; worsened by moisture and muscle bulk
**Physical examination:** Erythematous patch often at thighs, axilla, or neck
**Diagnosis:** Characteristic rash and location
**Treatment:** Removal of irritant, if possible; cold compresses, evaporation of moisture/sweat, lubricants, nonirritating clothing/equipment, and weight loss; antibiotic cream if infected
**Return to play:** No restrictions

### Runner's Nipples (Jogger's, Weightlifter's Nipples)

**Overview:** Caused from friction between the nipple/areola complex and shirts during a run or lift (Fig. 40.5)
**Presentation:** Nipples often present as raw and painful; bleeding and dryness may be present; most commonly seen in men as women are often protected by sports bra
**Physical examination:** Erythematous, swollen, scaling, fissuring, or raw nipple/areola complex; bleeding may be present
**Diagnosis:** Rash is diagnostic
**Treatment:** Good hygiene followed by triple antibiotic ointment, if infected; in men, these can be prevented by using Band-Aids or petroleum jelly before running or running shirtless, while prevention in women is the sports bra
**Return to sport:** As tolerated

## COLD INJURIES (Fig. 40.6)

### Frostnip

**Overview:** Superficial injury from the cold (Fig. 40.6)
**Presentation:** Most common cold injury; it revolves skin and subcutaneous tissues
**Physical examination:** Seen as flaking epidermis with possible vesicles associated with pain; most common area affected is the tip of the nose and ears; original presentation is white areas of skin with possible vessel formation
**Diagnosis:** Lesion is characteristic
**Treatment:** Gentle rewarming in 20-minute intervals preventing the freeze law cycle; covering of exposed skin is preventative
**Return to sport:** No restrictions

### Frostbite

**Overview:** Deep tissue injury due to exposure to long periods of cold, particularly at subzero temperatures (Fig. 40.6)
**Presentation:** Begins with numbness of the skin that progresses to blisters and then to tissue necrosis; exposed skin may harden as well
**Physical examination:** Seen most often on hands, feet, ears, and penis; athletes usually present with first- or second-degree frostbite
**Diagnosis:** Characteristic lesion
**Treatment:** Removal from cold environment; athletes should be placed in an area where there will not be a continuous freeze–thaw cycle; rewarming in water 39°C–44°C for 20 minutes; affected area may become cold sensitive
**Return to sport:** As tolerated depending on the extent of injury

### Pernio (Chilblains)

**Overview:** Development of painful nodules secondary to an area of vasoconstriction from nonfreezing temperatures
**Presentation:** Often associated with pruritis and/or paresthesias secondary to dermal damage
**Physical examination:** Painful nodules most often on exposed skin, particularly the extremities, are most often affected.
**Diagnosis:** Characteristic lesion
**Treatment:** Topical steroid ointment and/or vasodilators; coverage of exposed areas can prevent development; avoidance of cold is preventative; affected area may become cold sensitive
**Return to sport:** As tolerated depending on the extent of injury

## SUN AND HEAT INJURIES (Fig. 40.7)

### Sunburn

**Overview:** Caused by skin exposure to UV rays; it is most common skin disorder (Fig. 40.7)
**Presentation:** Usually occurs between 10 AM and 3 PM on exposed skin; water sports and ingestion of photosensitizing

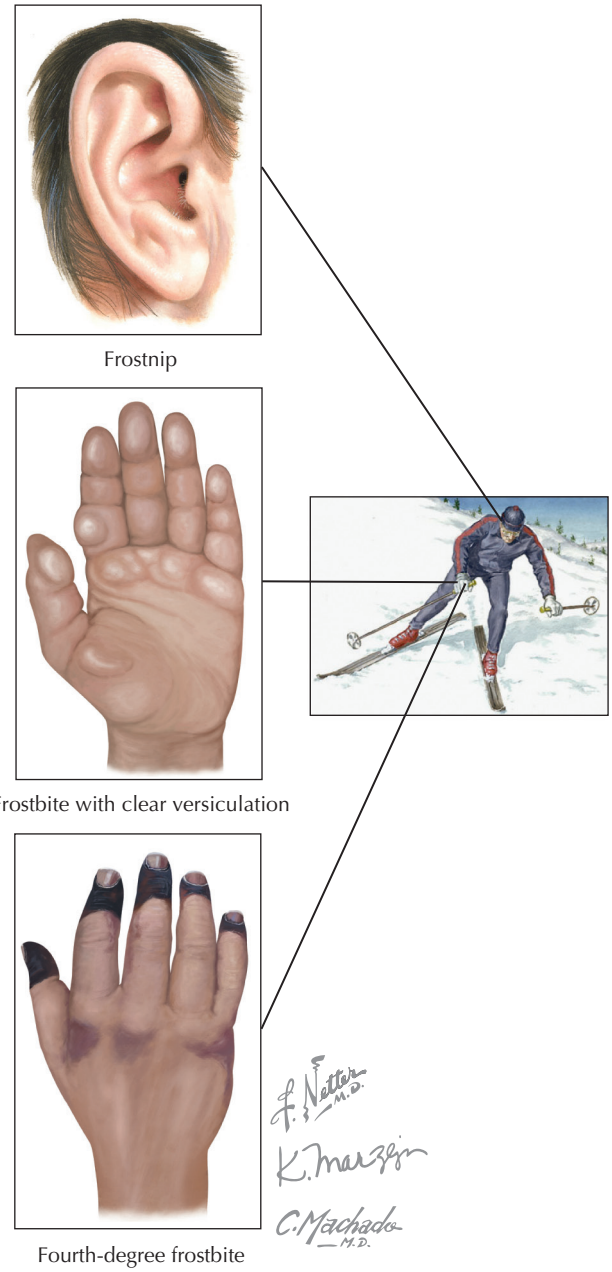



**Figure 40.6.** Common cold-related injuries.

drugs put athletes at greater risk; athletes with fair skin are also at a higher risk

**Physical examination:** Erythema and pain to the most superficial layers of the skin; deeper burns can cause damage to the deeper skin layers including nerves causing blisters and bullae resulting in significant discomfort. Repeated exposure can cause skin cancer including melanoma.

**Diagnosis:** Characteristic rash

**Treatment:** Minor burns are treated with symptomatic treatment including cold compresses, and possibly, low-dose topical steroids, antihistamines, or anesthetics. Second-degree burns are treated with systemic corticosteroids and topical anesthetics. Avoid skin breakdown. Oral fluids may be necessary with severe burns. Prevention: exercising before 10 AM and after 3 PM. Sunscreens protecting against both UVA and UVB should be applied 30 minutes before exercise and repeated after excessive sweating and/or water exposure. Waterproof formulas allow for increased protection, covering exposed skin, protective clothing, and headgear.

**Return to sport:** No restrictions

## Photodermatitis

**Overview:** Immune reaction against the skin due to exposure to the sun's UV rays (Fig. 40.7); can be caused secondary to medications, soaps, deodorants, or perfumes or by autoimmune conditions such as lupus or a vitamin deficiency

**Presentation:** May present similar to sunburn with erythema, pain, blisters, and some swelling

If athlete exhibits reactivity to a product, the athlete should be counseled to avoid products.

**Physical examination:** Can be seen on any part of body

**Diagnosis:** Characteristic rash and patient history

**Treatment:** Best achieved by avoidance of sun exposure; severe cases may be treated with corticosteroids or immunosuppressant therapy; alternative medication should be considered with those photosensitive to current prescription drugs

**Return to sport:** No restrictions

## Atopic Dermatitis

**Overview:** Eczematous outbreaks that occur in response to heat, sweat, temperature changes, or exertion (Fig. 40.7); mechanism unknown but has polygenic inheritance pattern

**Presentation:** Often occur for the first time early in life and present as recurrent pruritic, dry, scaly patches; outbreaks wax and wane often appearing early in life, resolving by the age of 30 years

**Physical examination:** Appear anywhere on body as pruritic, symmetric, and flexural eczematous lesions

**Diagnosis:** Diagnostic lesion and history of outbreaks

**Treatment:** Avoiding temperature changes and excessive sweating; sun exposure, emollients, and topical steroids are effective

**Return to sport:** No restrictions

## Hyperhidrosis

**Overview:** Excessive sweating in the setting of sports due usually to stress but can be congenital (Fig. 40.7)

**Presentation:** Benign but can interfere with vision or grip in sport

**Physical examination:** Findings of excessive sweat in the groin, axilla, palms, feet, forehead, and scalp

**Diagnosis:** History and presence of excessive sweating

**Treatment:** Aluminum chloride over time may decrease the excessive sweating

**Return to sport:** No restrictions

## Miliaria Crystalline (Sudamina)

**Overview:** Characterized by "dew drop" vesicles on an erythematous base seen in hot and humid conditions; caused by retention of sweat in the superficial skin

**Presentation:** Produced by exercise in a hot environment

**Physical examination:** Found anywhere on the body

**Diagnosis:** Characteristic lesion

**Treatment:** Cold compresses

**Return to sport:** No restrictions

## Miliaria Rubra (Prickly Heat)

**Overview:** Deeper, erythematous, and pruritic papules often lasting longer than those seen in Miliaria Crystallina; again, caused by retention of sweat now in the living layers of epidermis or upper dermis

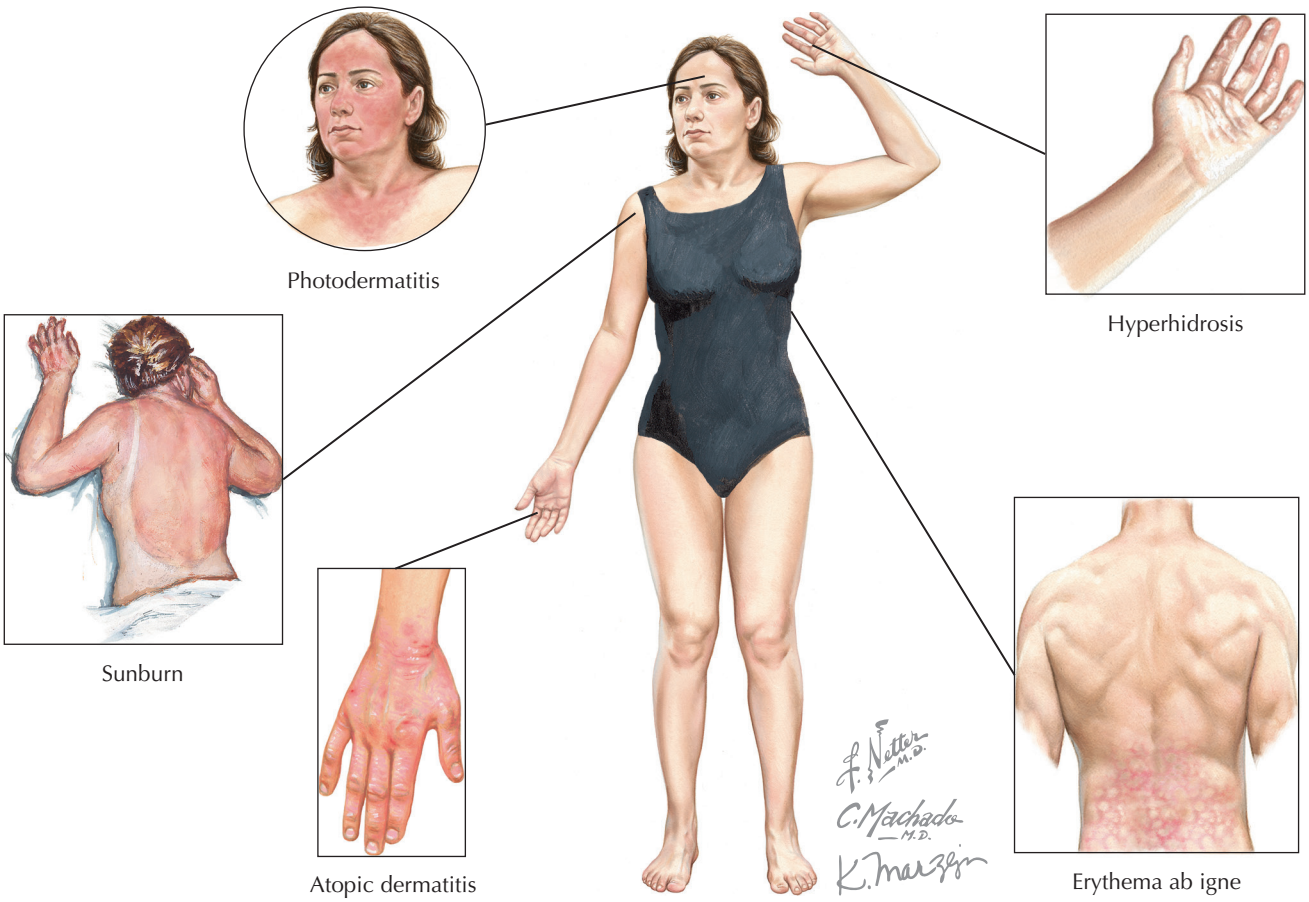



**Figure 40.7.** Common sun- and heat-related problems.

**Presentation:** Brought on by exercise in a hot environment; can evolve to Miliaria Pustulosa when Miliaria Rubra papules become pustules
**Physical examination:** Can be found anywhere on the body
**Diagnosis:** Characteristic lesion
**Treatment:** Cold compresses; discontinuation of exercise and resumption in cooler temperatures
**Return to sport:** No restrictions

## ALLERGIC REACTIONS AND DERMATITIS

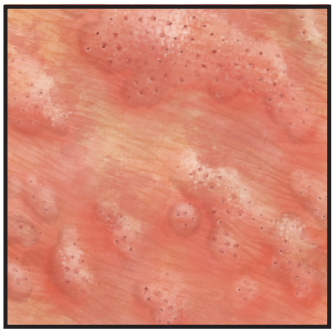
### Allergic Dermatitis

**Overview:** Caused by skin exposure to an allergen; ursinol, found in the sap of Toxicodendron plants, causing poison ivy is most common; other common allergens include nickel and elastic in sports equipment and tanning products in gloved sports; irritant contact dermatitis can be caused by calcium oxide seen in field-line markers when combined with water
**Presentation:** Poison ivy rash appears in linear rows approximating contact with the plant; other contact dermatitis are seen at allergen contact site
**Physical examination:** Can occur anywhere on body as erythema and later scaling, exudative plaques with pain, and/or pruritus; vesicles may form
**Diagnosis:** Characteristic rash and history
**Treatment:** With poison ivy, exposed skin, nails, fomites, clothing, and shoes should be cleaned immediately to remove the resin. Upon removal, the exposed area is no longer contagious. Treatment is with topical or oral steroids, nonsedating antihistamines, and rash coverage. With other allergens, immediately remove allergen-containing equipment and follow previous treatment. With irritant contact dermatitis, the skin should be flushed with soap and water to eliminate the irritant, followed by the previous treatment.
**Return to sport:** After resin removal or removal of allergen/irritant and coverage of lesion

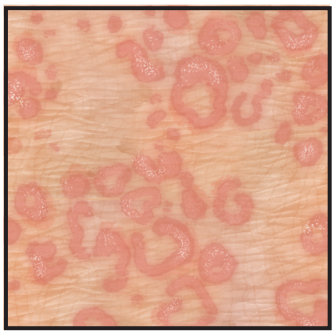
### Urticaria (Hives)

**Overview:** Can occur due to several triggers, including exercise, often seen in runners, cholinergic, often seen in response to increased body temperature, emotional stress, or physical exertion, aquagenic, often seen in swimmers, and cold urticaria, often seen in response to cold temperatures (Fig. 40.8)
**Presentation:** Presents as raised, pruritic, edematous, and erythematous plaques; these can continue to evolve in seriousness depending on exposure
**Physical examination:** Can occur anywhere on body—mostly on trunk and inner areas of the arms and legs
**Diagnosis:** Characteristic lesion and history
**Treatment:** Minor breakouts can be treated with antihistamines; systemic steroids are also used sometimes; evolving urticaria leading to anaphylaxis should be treated with epinephrine and airway and cardiovascular support; avoidance of triggers is preventative
**Return to sport:** No return if athlete is in anaphylaxis; can consider return if athlete is protected and treated with minor symptoms

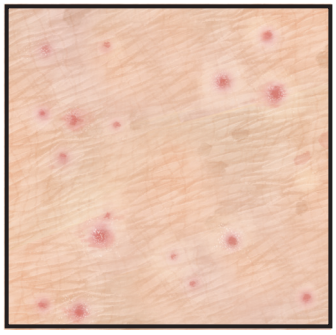
**Solar Urticaria:** Note the areas affected are those only exposed to the sun in this sleeveless shirt wearing man.



**Urticaria:** Pink edematous plaques with follicular accentuation caused by the dermal edema



**Annular and serpiginous urticaria:** This is a less commonly seen variant of urticaria.



**Cholinergic urticaria:** This form of urticaria can be induced by increasing the body temperature through exercise or submersion in a warm bath.

**Figure 40.8.** Urticaria.

## Stings

**Overview:** Insect stings should be planned for at sporting events
**Presentation:** Cause pain and pruritus at sting site along with swelling and erythema
**Physical examination:** Can be found anywhere on body
**Diagnosis:** Characteristic lesion
**Treatment:** Remove the stinger from the site without squeezing it to prevent more venom from penetrating the wound decreasing the reactivity at the site and systemically; removal of the stinger can prevent secondary infection. After cleaning, topical anesthesia can provide pain relief from an immediate sting; more concerning is systemic reaction to the sting. Anaphylactic reactions to the sting, including swelling of the lips or tongue, shortness of breath, or sudden hypotension, should be immediately treated with epinephrine. EpiPens should be a part of any medical kit, and the athlete should carry EpiPen on person in the future.
**Return to sport:** No restrictions

# TRAUMA

## Lacerations

**Overview:** Break in the skin barrier; more problematic with bleeding

Athletes cannot compete with bleeding wounds or blood soiled equipment or uniforms. With informed consent obtained in advance of the season, lacerations can be quickly and effectively closed and the athlete returned to play. Materials, including the use of a commercial blood removal agent, should be prepared in advance to allow for quick and effective cleaning and wound closure.
**Presentation:** Break in skin barrier that may or may not expose subcutaneous tissues
**Physical examination:** Seen anywhere on body
**Diagnosis:** Break in skin barrier, often with bleeding, and history of trauma is diagnostic.
**Treatment:** Universal precautions including use of gloves, and possibly a mask with shield, followed by anesthetizing the laceration. Cleaning and irrigation of wound with sterile saline to remove foreign debris. Do not damage the wound edges with irrigation. Betadine, or other cleansing agents, should be placed around the wound but not directly upon the wound itself because it can impede tissue healing. If time allows, sutures can be immediately placed during the competition. If not, and if adequate adherence of the wound edges can be obtained, the wound can be closed with steri-strips or glue and then sutured at halftime or after the competition, if necessary. Blood stain remover should be used to then remove blood from uniforms or equipment.
**Return to sport:** Closure of wound without active bleeding that is clean, dry, and covered and removal or cleaning of blood-stained uniforms or equipment.

# RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# CONNECTIVE TISSUE AND RHEUMATOLOGIC PROBLEMS IN ATHLETES

41

Mark E. Lavallee • Abbie L. Kelley

## CONNECTIVE TISSUE DISORDERS

### Marfan Syndrome

**Overview:** Inheritable autosomal-dominant genetic condition that affects the processing of fibrillin; it is caused by >400 mutations in the gene encoding fibrillin-1 (FBN-1), located on chromosome 15 at the q21 loci. Approximately 10% of patients with Marfan phenotype have no identifiable mutation in the FBN1 gene; mutations in TGF-beta receptor 2 and TGF-beta receptor 1 genes have been linked to these patients. Incidence is 1 in 3000–10,000 live births; an estimated 2,00,000 Americans have Marfan syndrome. Fibrillin, which is a major component of microfibrils, is found in large amounts in the aortic root > aorta > lens > joints > other connective tissues.

**Presentation:** Athletes with Marfan syndrome may (Fig. 41.1):

- Be tall (≥97th percentile) and thin
- Have long thin arms, legs, hands, and feet
- Present with sudden death (aortic root dissection)
- Complain of joint laxity (including joint subluxations and dislocations)
- Have paradoxical reduction in joint mobility, particularly in the elbow and digits
- Have joint pain
- Have scoliosis (60% have curvature >20 degrees)
- Have a pectus (chest wall) deformity (carinatum > excavatum)
- Have visual problems
- Have dental crowding
- Be drawn to sports where tall, slender build is an advantage (e.g., basketball, volleyball, rowing, and track)
- Famous athletes with Marfan syndrome include Flo Hyman (the United States [US] 1984 Olympic Silver Medalist in Volleyball—died of aortic dissection)

**Physical examination:** See Fig. 41.1

**Diagnostics:**

- Echocardiogram: aortic root dilation, aortic dissection, and valvular issues (e.g., mitral valve prolapse [MVP])
- Slit-lamp examination or ocular ultrasound: look for lens abnormalities
- Imaging of chest and abdomen: CT scan with IV contrast, MRI with IV contrast, and ultrasound
- Genetic testing: via blood or tissue samples for FBN-1 mutation; may be helpful if family members are tested
- Using diagnostic criteria (see Table 41.1)
  - The Revised 2010 Ghent Nosology emphasizes more on cardinal clinical features of Marfan syndrome (aortic root dilatation/dissection and ectopia lentis) and on testing for mutations in the FBN1 gene.
  - Application of diagnostic criteria to individuals aged <20 years requires special care because additional clinical features may present/develop later.

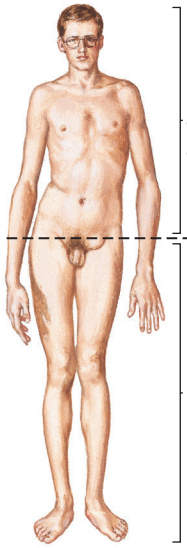
**Treatment:**

- **General**: Healthy lifestyle, including exercise and diet to control lipids and systemic blood pressure; Medic-alert bracelet/necklace stating the condition
- **Prevention**: Annual evaluation of eyes (ectopic lentis or lens dislocation), heart (valvular issues, aortic root dilatation, or dissection), and imaging of chest and abdomen (identifying/following aortic dissections, aneurysms, or dilatation)
- **Vascular:** Echocardiogram should be performed at the time of diagnosis and 6 months later to determine the rate of enlargement of the aortic root and ascending aorta. If stable over time, monitor annually for any changes in the aorta. Avoid strenuous activities that will increase intrathoracic pressure (e.g., powerlifting). If aortic pathology worsens, consider intraluminal prosthetic device versus elective open surgical replacement of the aorta. Use of a beta-blocker has been correlated with increased survival. The age at which beta-blockers should be initiated is still debated. Therapy targeting the renin–angiotensin system, such as losartan, may reduce aortic root dilatation rate in adults. Valve replacement surgery is also an option, but it is not without severe risks for those with aortic valve insufficiency and ascending aorta dilation.
- **Orthopedic:** Mild strength training to stabilize severe joint laxity; physical therapy (particularly joint and core strengthening, proprioceptive training modalities, and strengthening)
- **Psychology:** Provide empathy and psychological support as per the individual's and family's needs; genetic counseling is always strongly advised, as is putting the patient in contact with knowledgeable medical professionals and local/national support groups such as the National Marfan Foundation at www.marfan.org
- **Pain control:** Encourage smoking cessation. Control pain with oral, injectable, transdermal, and topical medications including acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, tramadol, transcutaneous electrical nerve stimulation (TENS) units, bracing, ring splints, and lastly, surgery.

**Prognosis:** In 1972, the mean age of death was 32 (range, 16–48) years. By 1995, the median cumulative probability of survival was 72 years.

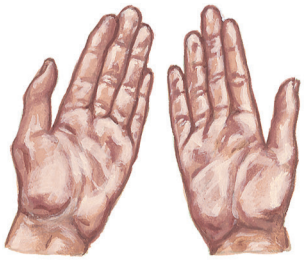
**Return to play:**

- 46th Bethesda American College of Cardiology recommendations (2015):
  - May participate in low and moderate static/low-dynamic competitive sports if they *do not* have one of the following: aortic root dilation, moderate-to-severe mitral valve regurgitation, or family history of aortic dissection at an aortic diameter of <50 mm.
  - Athletes with aortic root dilation may only participate in low-intensity competitive sports.
  - Avoid sports with bodily collision.
  - Same recommendation for aortic valve regurgitation
  - Prefer nonstrenuous, low-static, low-dynamic sports (e.g., golf, walking, billiards, riflery, and bowling).
- Encourage an active lifestyle.
  - Favor noncompetitive, isokinetic exercise performed at a nonstrenuous aerobic pace.
  - Avoid activities that involve high levels of isometric workloads (e.g., Olympic weightlifting, gymnastics, etc.).
  - Mild strength training (nonbreath-holding) if joint laxity is present; prefer multiple repetitions at lower resistance rather than maximal repetitions at higher resistance
  - Be careful in environments with rapid atmospheric pressure changes (scuba diving or flying in an unpressurized cabin) or rapid decelerations (car racing or skydiving).




Tall, thin person with skeletal disproportion. Upper body segment (top of head to pubis) shorter than lower body segment (pubis to soles of feet). Fingertips reach almost to knees (arm span-to-height ratio greater than 1.05). Long, thin fingers (arachnodactyly). Scoliosis, chest deformity, inguinal hernia, flatfoot

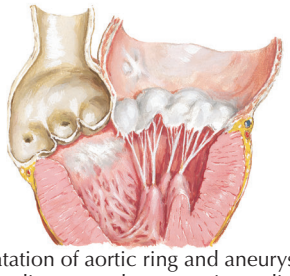
Ectopia lentis (upward and temporal displacement of eye lens). Retinal detachment, myopia, and other ocular complications may occur.



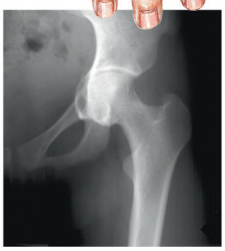
Arachnodactyly of hands

Walker-Murdoch wrist sign. Because of long fingers and thin forearm, thumb and little finger overlap when patient grasps wrist.

**Steinberg sign.** Tip of thumb protrudes when thumb folded inside fist. Thumb and index finger overlap when encircling opposite wrist.



Dilatation of aortic ring and aneurysm of ascending aorta due to cystic medial necrosis cause aortic insufficiency. Mitral valve prolapse causes regurgitation. Heart failure common



Radiograph shows acetabular protrusion (unilateral or bilateral).

Arachnodactyly of feet

**Figure 41.1.** Characteristics of Marfan syndrome.

## TABLE 41.1 GHENT NOSOLOGY

| Family History? | Criteria | Systemic Symptoms |
|---|---|---|
| Yes *(need at least 1 criterion for diagnosis)* | Ectopia lentis<br>Systemic score ≥7 points*<br>Aortic criterion (aortic diameter Z ≥2 when age ≥20 years, Z ≥3 when age <20 years, or aortic root dissection)* | Wrist **AND** thumb sign: 3 points (wrist **OR** thumb sign: 1 point)<br>Pectus carinatum deformity: 2 points (pectus excavatum or chest asymmetry: 1 point)<br>Hindfoot valgus: 2 points (plain pes planus: 1 point)<br>Pneumothorax: 2 points<br>Dural ectasia: 2 points<br>Protrusio acetabuli: 2 points<br>Reduced upper segment/lower segment ratio **AND** increased arm span/height **AND** no severe scoliosis: 1 point<br>Scoliosis or thoracolumbar kyphosis: 1 point<br>Reduced elbow extension (≤170 with full extension): 1 point<br>Facial features (at least 3 of the following 5 features: dolichocephaly [reduced cephalic index or head width/length ratio], enophthalmos, downslanting palpebral fissures, malar hypoplasia, retrognathia): 1 point<br>Skin striae: 1 point<br>Myopia >3 diopters: 1 point<br>Mitral valve prolapsed (all types): 1 point |
| No (need at least 1 criterion for diagnosis) | Aortic criterion (aortic diameter Z ≥2 or aortic root dissection) **and** ectopia lentis*<br>Aortic criterion (aortic diameter Z ≥2 or aortic root dissection) **and** a causal FBN1 mutation<br>Aortic criterion (aortic diameter Z ≥2 or aortic root dissection) **and** a systemic score ≥7<br>Ectopia lentis **and** a causal FBN1 mutation that has been identified in an individual with aortic aneurysm | |

*Diagnosis of Marfan syndrome can be made only in the absence of discriminating features of Shprintzen–Goldberg syndrome, Loeys–Dietz syndrome, or vascular Ehlers–Danlos syndrome **and** after TGFBR1/2, collagen biochemistry, or COL3A1 testing, if indicated.
Aortic Root Z score determined by height, weight, age, and aortic root (in cm) at sinuses of Valsalva.

- Control lipid levels, blood pressure, and blood sugar to decrease long-term injury to the vascular endothelium.
- Educate the patient regarding the risks of participation.
- Use protective eyewear whenever appropriate.

- Once orthopedic or ocular injury has healed and the injured area is adequately protected, the athlete may return to play. Support the patient with reasonable adaptive measures in order to participate.

## Ehlers–Danlos Syndrome (EDS)

**Overview:** Group of inheritable genetic conditions that affect connective tissue, particularly collagen in joints, vessels, skin, and internal organs; variable severity seen within each type and within the same family of pedigree; there are six types of Ehlers–Danlos syndrome (EDS), of which, 90% seem to fall into the first three categories (hypermobility, classical, and vascular). Incidence is approximately 1 in 5000 live births. The term *generalized* or *benign joint hypermobility syndrome* (GJHS or BJHS) was first used by Kirk, Ansell, and Bywaters in 1967. EDS hypermobility type was first described as a condition in 1946 and noted to be genetically based in 1970. The description of BJHS also uses the Beighton Scale and the same diagnostic criteria as EDS hypermobility type. Essentially, they are the same entity with only varying degrees of expressivity. A literature review seems to reveal no significant difference in the nosology between BJHS and aspects of EDS hypermobility type. BJHS seems to be a term preferred by those outside genetic circles (e.g., rheumatology, orthopedics, etc.) and EDS hypermobility type and Marfan syndrome are preferred by general medical and genetic groups. As of 2016, just after this chapter went to print, an international group, Ehlers–Danlos Society, published the NEW nosology, diagnostic criteria, treatment, and associated conditions in relation to EDS. The results will be available at www.ednf.org.

**Presentation:**

- **Hypermobility:** Most common type in North America; autosomal dominant inheritance with unknown underlying genetic abnormality: mild to severe joint laxity, most often involving multiple joints subluxating or dislocating, mild skin involvement, chronic joint pain with often normal imaging studies, Beighton Scale score of ≥5/9, and 22% risk of developing aortic root dilatation in lifetime; 12% of pediatric patients with EDS show aortic root dilatation on their first echocardiogram.
- **Classical:** Second most common type in North America; autosomal dominant inheritance, mutations found within the collagen genes COL5A1 and COL5A2, interact with type 1 collagen molecules during fibrillogenesis. Skin hyperextensibility, wide atrophic scarring ("cigarette paper scar tissue"), and joint hypermobility are the major criteria. Minor criteria include easy bruising; soft, doughy skin; tissue friability; molluscoid pseudotumors; piezogenic papules; hernia; epicanthal folds around eyes, and first-degree family member with EDS. Diagnosis must have one major and two minor criteria or three minor criteria. There is a 33% risk of developing aortic root dilatation in lifetime; 6% of pediatric patients with EDS show aortic root dilatation on their first echocardiogram; early osteoarthritis particularly in smaller joints.
- **Vascular:** Third most common type in North America; autosomal dominant inheritance, mutations in the COL3A1 gene; life-threatening (sudden death often from aortic/vascular or bowel rupture). Particular facial features include delicate nose, wide-set eyes, thin cheekbones, thin, translucent skin, very prominent veins on extremities and chest, skin friability, and mild to moderate joint laxity; a very significant risk (90% by the age of 40) of developing aortic pathology (e.g., root dilatation, aneurysm, and dissection).
- **Kyphoscoliotic:** Very rare; autosomal recessive inheritance, mutations in the PLOD1 gene: severe congenital hypotonia, progressive scoliosis, risk of ocular rupture, marfanoid body habitus, and diagnosis via urine test available
- **Dermatosparaxis:** Very rare; autosomal recessive, mutations in the ADAMTS2 gene: doughy, redundant skin, autosomal recessive, diagnosis via skin biopsy
- **Arthrochalasia:** Very rare; autosomal dominant inheritance, mutations in COL1A1 or COL1A2: congenital hip dislocation, atrophic scars, joint hypermobility, and diagnosis via skin biopsy

**Physical examination:** See Fig. 41.2

- Beighton scale (score: ≥5/9)
- Excessive subtalar motion
- Pes planus
- Pinchable skin in the relaxed palmer aspect of hand
- Pectus deformity: excavatum more common than carinatum
- Touch tip of tongue to nose
- Skin hyperextensibile to >3 cm at the mandibular angle
- Widened, atrophic scars (known as "cigarette paper scar tissue") (see Fig. 41.2)
- Piezogenic papules (often found near sole of feet)
- Subcutaneous spheroids (subcutaneous, firm, small, and mobile)
- Joint pain (acute or chronic, episodic)
- Joint laxity or hypermobility affecting multiple joints
- Joint subluxations and dislocations
- Family history
- Early osteoarthritis
- Postural orthostatic tachycardia syndrome (POTS)
- Chiari malformation
- Mast cell activation
- Hernias (e.g., ventral, umbilical, and inguinal)
- MVP
- Dental malalignment/gingival issues
- Sudden death (due to vascular or bowel rupture)

**Diagnostics:** Genetic testing, echocardiogram periodically, CT scan of chest and abdomen with IV contrast or ultrasound periodically, skin punch biopsy (4 mm) or blood serum put into a live medium for EDS and TGF-beta (e.g., *Chlamydia* culture) (currently only for classical and vascular types), and radiographs, and other imaging of affected joints

**Treatment:**

- **General:** Healthy lifestyle that includes exercise and diet to control lipids and systemic blood pressure; Medic-alert bracelet/necklace stating condition
- **Prevention:** Periodic evaluation of heart (e.g., valvular issues), chest/abdomen (e.g., identifying/following aortic dissections, aneurysms, or dilatation), and eyes (e.g., annual ophthalmologic evaluations in kyphoscoliotic type)
- **Vascular:** Monitor any changes in the aorta and avoid strenuous activities that will increase intrathoracic pressure (e.g., powerlifting); if aortic pathology worsens, consider intraluminal prosthetic device versus elective open surgical replacement of aorta; encourage smoking cessation.
- **Orthopedic:** Mild strength training to stabilize severe joint laxity; physical therapy, particularly joint and core strengthening, proprioceptive training modalities, strengthening, and unweighted exercise (e.g., pool therapy, harnesses, and total gym)
- **Psychology:** Provide empathy and psychological support as per individual's and family's needs. Genetic counseling is always strongly advised, as is putting patient in contact with knowledgeable medical professionals and local/national

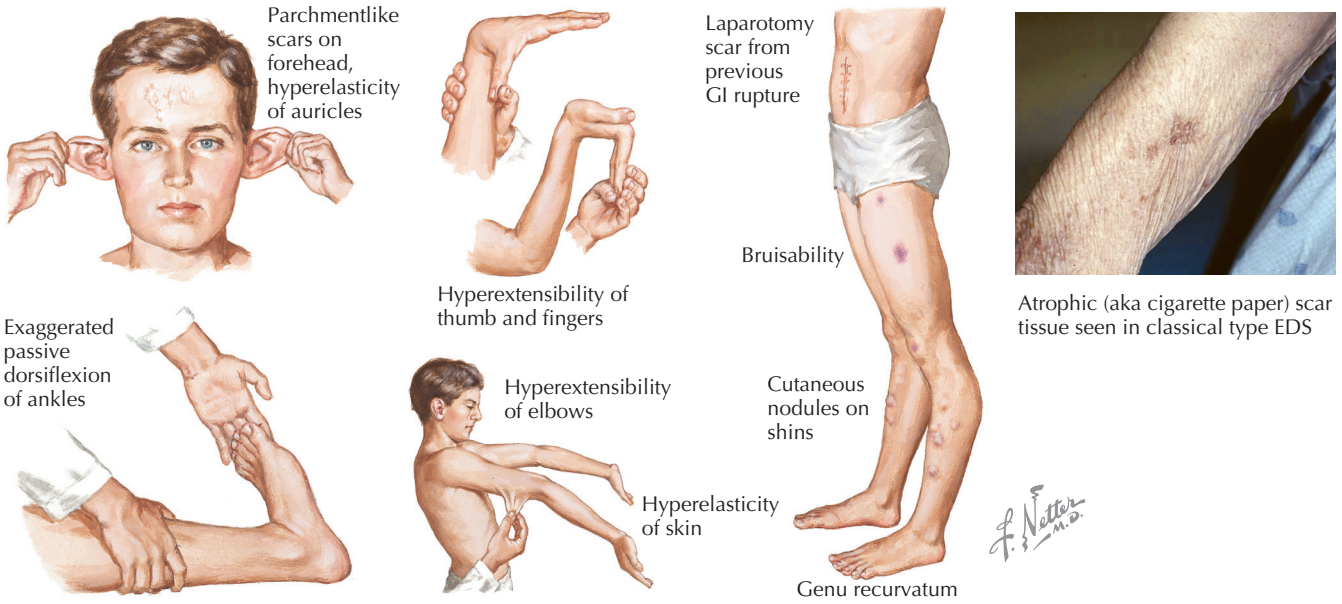



**Figure 41.2.** Characteristics of Ehlers–Danlos syndrome. (Photograph from Goldman L, Ausiello D, Arend W, et al., eds: *Cecil Textbook of Medicine*, 23rd ed. Philadelphia: Elsevier, 2007.)

support groups. Have the athlete visit the Ehlers Danlos National Foundation website at www.ednf.org.

- **Pain control:** Oral, injectable, transdermal, and topical medications including acetaminophen, NSAIDs, tramadol, TENS units, bracing, ring splints, ambulation-assist devices, and lastly surgery; use opioids only for acute trauma or as intermittent rescue medication owing to the risk of dependence from chronic use.

**Prognosis:** Proportion of patients with EDS who will develop pathology within their aortic root or aorta: 90% vascular type, 33% classical type, and 22% hypermobility type

**Return to sport:** Once orthopedic or integument injury has healed and the injured area adequately protected, the athlete may return to play. Support the patient with reasonable adaptive measures in order to participate. According to the 46th Bethesda recommendations (2015):

- Individuals with vascular type EDS may participate in low-static, low-dynamic sports if they do not have any of the following: aortic enlargement or dissection (or branch vessel enlargement), moderate to severe mitral regurgitation, or extracardiac organ system involvement that makes participation hazardous.
- Keep normal weight.
  - Underweight: Not enough muscle mass to stabilize loose joints
  - Overweight: Promotes early osteoarthritis
- Encourage an active lifestyle.
- Control lipids, blood pressure, and blood sugar to decrease long-term injury to vascular endothelium.
- Mild to moderate strength training is often helpful in joint stabilization.
- Encourage noncontact nontraumatic sports, particularly for those with skin fragility (i.e., classical type) and severe joint laxity (i.e., hypermobility type).
- Do not recommend high-dynamic sports that could increase intra-abdominal pressure and blood pressure owing to the existing risk of aortic dilatation or colonic rupture.

## Osteogenesis Imperfecta

**Overview:** Also known as *brittle bone syndrome*, osteogenesis imperfecta (OI) is an inheritable genetic disorder that affects collagen (type 1) (Fig. 41.3). A classification system is commonly used to describe how severely a person is affected by OI. The genetic defect (the *COL1A* gene) affects type 1 collagen, which is primarily found in the bone. A total of nine types of OI have been documented (most autosomal dominant; four types have autosomal recessive expressivity). There are two major forms of OI: Type I and Type II. In the Type I dominant (classical) form, the patient has extremely little or a poor quality of type 1 collagen. Type II dominant form has more severe manifestations, including long bone fractures and shortening and severe chest wall deformity.

**Incidence:** An estimated 20,000–50,000 individuals in the US have OI.

**Presentation:**

- Bones fracture easily. Most fractures occur before puberty.
- Normal or near-normal stature
- Loose joints and muscle weakness
- Sclera (whites of the eyes) usually have blue, purple, or gray tint
- Triangular face
- Tendency toward spinal curvature
- Bone deformity absent or minimal
- Brittle teeth possible
- Hearing loss possible, often beginning in early 20s or 30s
- May die in utero (Type II)
- Severe chest wall deformity (Type II)
- Often nonambulatory in wheelchair (Type II)

**Physical examination:** (Fig. 41.3)

- Dentinogenesis imperfecta (poorly formed teeth)
- Blue sclera
- Bone fragility
- Basilar skull deformity
- Pathogenic and multiplicity of fractures
- Adult-onset hearing loss
- Osteoporosis
- Joint hyperextensibility
- Short stature/limb shortening or deformity in severe forms

**Diagnostics:** Laboratory findings may show elevated alkaline phosphatase and hypercalciuria. Radiographs may show bowing deformities and multiple fractures/callus formation. Bone scan may show multiple areas of increased uptake as a result of multiple areas of occult or stress fractures. Dual-energy X-ray

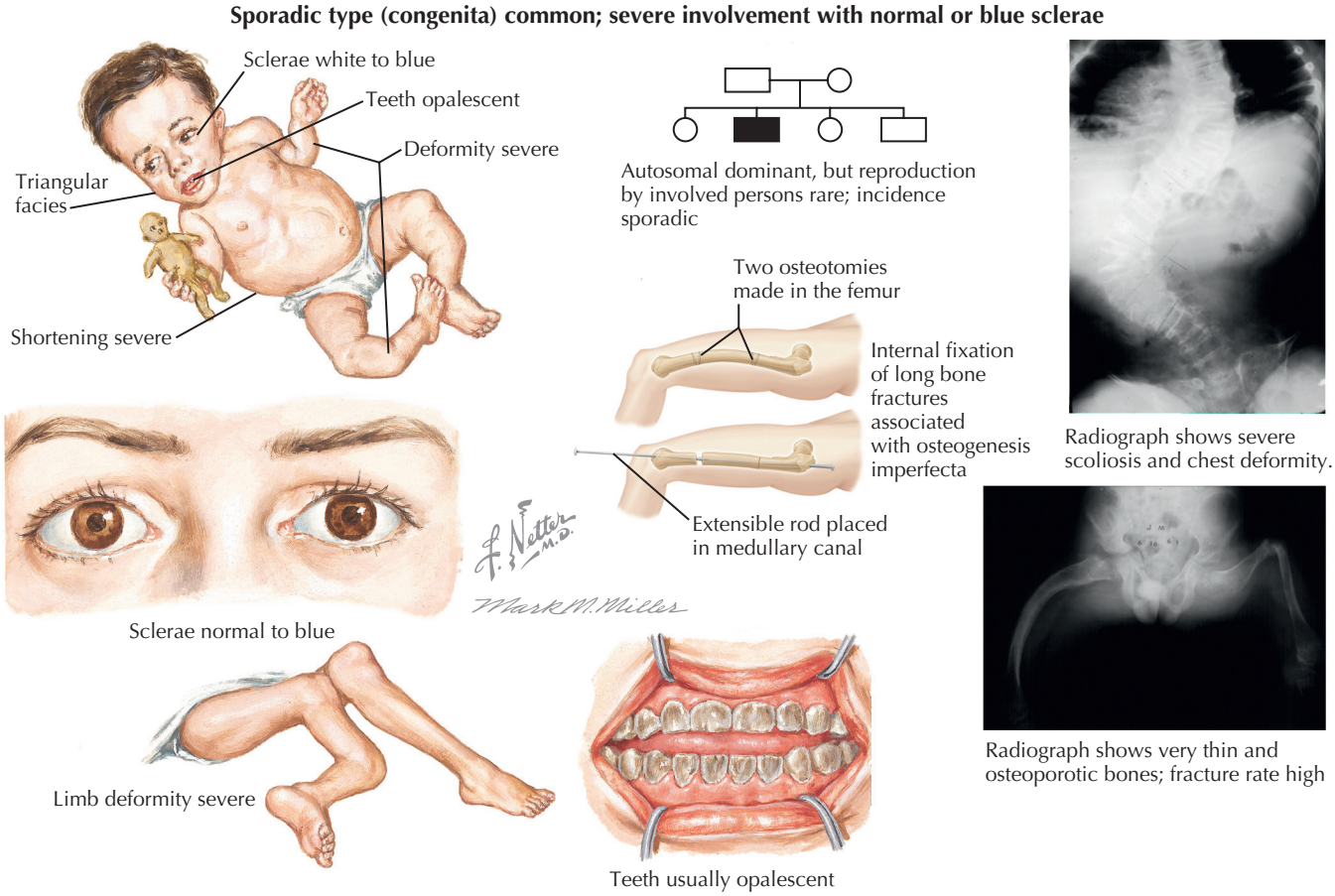



**Figure 41.3.** Characteristics of osteogenesis imperfecta.

absorptiometry (DEXA) scan may show premature decrease in bone density consistent with osteopenia or osteoporosis. Of skin-punch biopsy tests, both the collagen biopsy test and the DNA test are thought to detect almost 90% of all type I collagen mutations. A negative type I collagen study does not rule out OI. When a type I collagen mutation is not found, other DNA tests to check for recessive forms are available.

**Treatment:**

- **General**: Healthy lifestyle including exercise and diet
- **Prevention:** Protection or avoidance of contact or collision sports is paramount. Medic-alert bracelet/necklace stating condition and severity is recommended. Periodic evaluation of hearing, bone mineral density with DEXA scan, and spirometry to monitor for restrictive lung defects (secondary to rib and vertebral fractures)
- **Orthopedic:** Mild strength training to stabilize joint laxity; physical therapy may help (particularly after fractures) with pain control and improvement in function after prolonged immobilization. Often because of the nature of the fracture, open-reduction internal fixation is needed (see Fig. 41.3). Unweighted exercises may be beneficial (e.g., pool therapy, harnesses, and total gym). "Off-label" use of bisphosphonates has been advocated by some for between 2 and 5 years.
- **Psychology:** Provide empathy and psychological support as per individual's and family's needs. Genetic counseling is always strongly advised, as is putting patient in contact with knowledgeable medical professionals and local/national support groups. Have athletes visit the Osteogenesis Imperfecta Foundation website at www.oif.org.
- **Pain control:** Oral, injectable, transdermal, and topical medications including acetaminophen, NSAIDs, opioids, tramadol, TENS units, bracing, ring splints, ambulation-assist devices, and lastly surgery

**Prognosis:** Depends on the type of OI

In general, patients have a full life expectancy, but morbidity seems to be directly correlated to the number of fractures. Type II has a significant higher morbidity, and patients are often wheelchair-bound if they survive the perinatal period.

**Return to sport:** Absolutely avoid contact/collision sports. Certain high-impact sports may be contraindicated depending on severity of the condition. Joint and bone protection is crucial both in activities of daily life and in selection of athletic and recreational activities. No cardiovascular limitation noted by the 46th Bethesda Guidelines. Allowed once fracture(s) have both clinically and radiographically healed and matured

## SERONEGATIVE ARTHROPATHIES

### Ankylosing Spondylitis (AS)

**Overview:** A chronic, inflammatory rheumatic disease that causes arthritis of the spine and sacroiliac joint (Fig. 41.4); the chronic inflammation and irritation of the spinal joints (vertebrae) can eventually cause the vertebrae to fuse together, a condition known as "ankylosis."

**Incidence:** Approximately 350,000 Americans are estimated to have ankylosing spondylitis (AS). Approximately 1.8% of the US population will develop AS.

**Presentation:**

- Back pain: acute or chronic
- Usually presents in the 3rd–4th decade of life

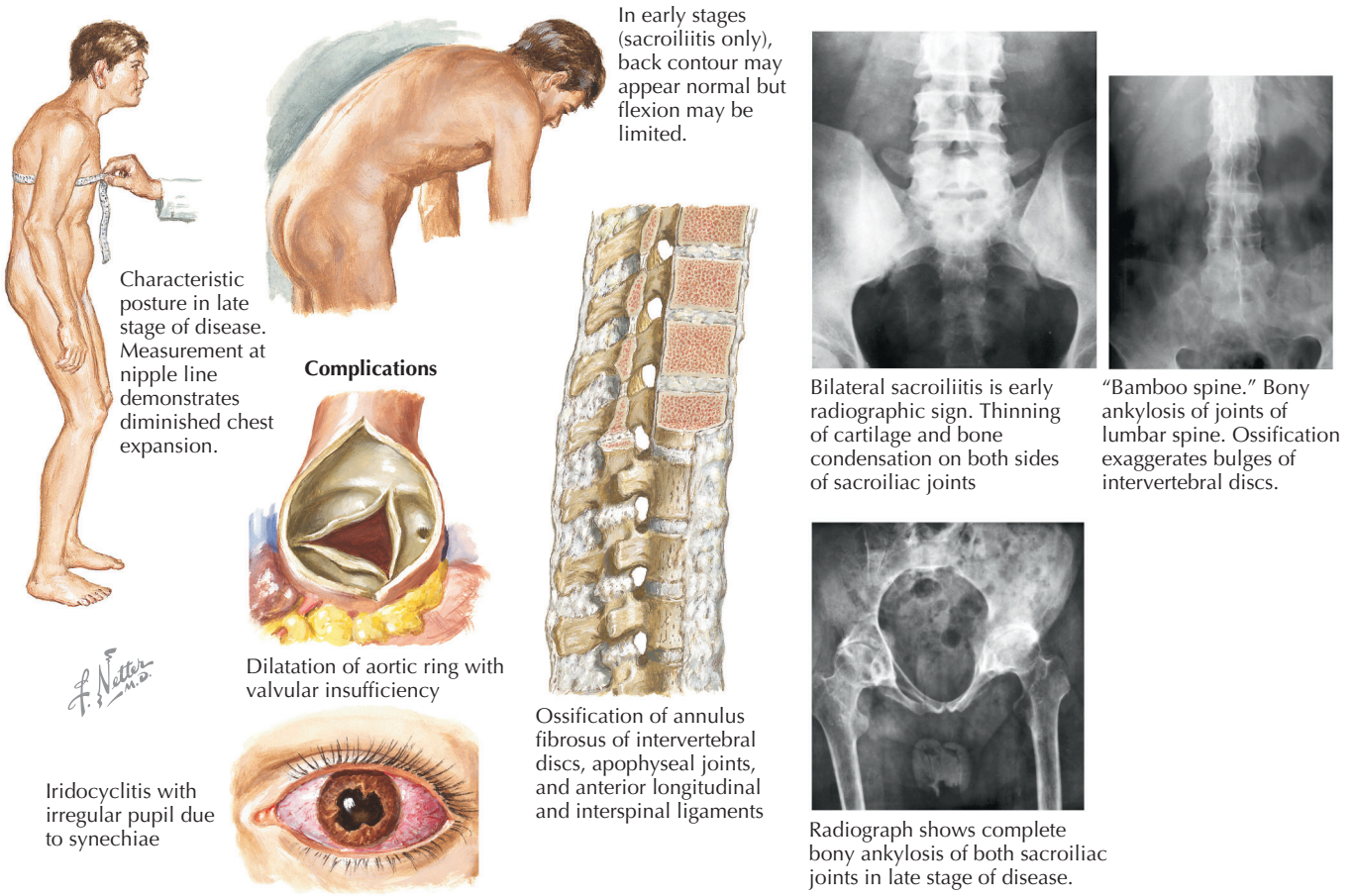



**Figure 41.4.** Characteristics of ankylosing spondylitis.

- Probable male to female ratio is 2 : 1 to 3 : 1, although recent survey in the US showed no significant difference in prevalence between men and women.
- Sacroiliitis
- Dactylitis ("sausage digits")
- Occasional pain/inflammation in other joints (e.g., hips, shoulders, knees, and ankles)
- Morning stiffness

**Physical examination:** See Fig. 41.4

- Pain with back flexion (lumbar/sacral area)
- Decreased range of motion in spine (cervical to sacral)
- **Lateral spinal flexion examination**: Patient stands erect with heels and back against the wall. The first measurement is the distance from finger tips to floor. Then, the patient is asked to laterally flex the spine without rotating or bending the knees. Second measurement is the distance between finger tips and floor. Difference of >10 cm is normal.
- **Schober test:** Measure forward flexion of the lumber spine. Patient is erect and a mark is placed 5 cm below the posterior superior iliac spine (PSIS), at midline at level of PSIS, and 10 cm above the PSIS over the spinous process. The patient is asked to bend forward without bending the knees. If the measurement between the two marks does not exceed 20 cm in length (start is 15 cm), the test is considered positive.
- Limited ability to fully expand chest with deep inhalation
- Ophthalmologic examination may show uveitis or iritis.
- Limited internal and external rotation of the hips
- Enthesitis
- Sacroiliac tenderness and positive FABER test

**Diagnostic:**

- Blood work: HLA-B27 is positive in 90% of AS cases. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are helpful for monitoring disease progression.
- Radiograph shows squaring of vertebral bodies, "bamboo spine," fusion of vertebrae
- Radiographic grading: modified Stokes Ankylosing Spine Score (SASS)
- MRI shows spondylitis (inflammation)/sacroiliitis.
- Indices for evaluating disease:
  - Bath Ankylosing Spondylitis Disease Activity Index (BASDAI)
  - Ankylosing Spondylitis Disease Activity Score (ASDAS)
  - Bath Ankylosing Spondylitis Functional Index (BASFI)
  - AS Quality of Life instrument (ASQoL)

**Treatment:**

- Discontinue smoking because it can accelerate lung scarring and decrease pulmonary function.
- TNF-blockers: Adalimumab, etanercept, or infliximab
- Drug-modifying antirheumatic drugs (DMARDs): Methotrexate or sulfasalazine
- Oral glucocorticoids: Limited data suggest high doses may have some benefits for very short-term therapy.
- Injection (prednisone): epidural versus facet
- Pain control: Aspirin, acetaminophen, NSAIDs, tramadol
- Physical therapy: Firm mattress; back extension exercises; stress core strength; maintain spine mobility and lung capacity; and improve posture

**Prognosis:** Variable

Most patients with limited disease do well, although a minority of patients develop severe skeletal symptoms or life-threatening complications. Approximately 1% will "burn-out" or go into remission.

**Return to sport:** Continuous flexibility training and core strengthening and mild to moderate exercise within limits of disease activity; swimming is preferred because it avoids jarring impact of the spine. Patients can participate in carefully chosen aerobic sports when disease is inactive. Aerobic exercise is generally encouraged because it promotes full expansion of the breathing muscles and opens the airways.

## Osteoarthritis

**Overview:** Most common joint disorder; approximately 5% of the population between ages 35 and 54 show signs of osteoarthritis in knee joint on radiographs; directly related to severity of occurrences of trauma, joint-related surgery, age, and activity wherein normal articular cartilage is damaged (Fig. 41.5). Currently, it is thought that metalloprotease enzymes (e.g., collagenase, stromelysin, and gelatinase) are active in the degradation of cartilage. Other mediators that seem to play a role in osteoarthritis are cytokines, other proteases, nitric oxide, calcium crystals, sex hormones, aging, and familial genetics.

**Presentation:** Early morning stiffness and joint pain that generally improves with activity (see Fig. 41.5).

**Physical examination:**

- Heberden nodules: joint enlargement of the distal interphalangeal (DIP) joint.
- Bouchard nodules: tendon nodules located around the proximal interphalangeal (PIP) joint (see Fig. 41.5)
- Stiff, enlarged joint, often monoarticular or asymmetric
- Decreased range of motion
- Antalgic movement of that joint (seen often in weight-bearing joints)

**Diagnostic:** History and physical examination; radiographs (weight-bearing preferred in lower extremity); bone scan will show moderate to severe arthritic changes in all joints of the body.

**Treatment:**

- Pain control (acetaminophen, tramadol, and NSAIDs)
- Physical therapy should address range of motion, core strength, mobility, modalities, mild strength training to stabilize joints, flexibility training, and home exercise program (HEP).
- Intra-articular injections: steroids and hyaluronic acid
- Weight loss if a patient is overweight
- Joint replacement surgery, when conservative measures fail

**Prognosis:** Varies among patients; depends on severity of the condition, number of joints involved, concomitant diseases, and activities (occupation/recreation)

**Return to sport:** As tolerated by pain and mechanical defects; long-term impact loading of affected joint(s) may advance the disease at a quicker pace

# SEROPOSITIVE ARTHROPATHIES

## Rheumatoid Arthritis (RA)

**Overview:** The most common chronic inflammatory arthritis, rheumatoid arthritis (RA) is thought to have a genetic susceptibility and be caused by a complex interaction between T-cell and B-lymphocytes, macrophages, and fibroblast-like synoviocytes. Bimodal expressivity of this condition as juvenile-onset versus adult-onset RA.

**Incidence:** Approximately 1% of the population in Great Britain and the US is affected by RA.

**Presentation:** Joint pain in multiple joints (usually symmetric), nontrauma-related, swollen joints, finger deformities or weakness (often episodic in nature), myositis/vasculitis, and drug-induced myopathy (e.g., glucocorticoids, statins, and antimalarials) (Fig. 41.6)

**Physical examination:**

- Metacarpophalangeal joint inflammation, particularly first
- Heberden nodules (more common in OA)
- Bouchard nodules (see Fig. 41.6)
- Tender PIP joints of the hand
- Fingernail: subungual hemorrhage or infarct (see Fig. 41.6)
- Synovitis of multiple joints
- Ulnar deviation of phalanges *(in long-term cases)*

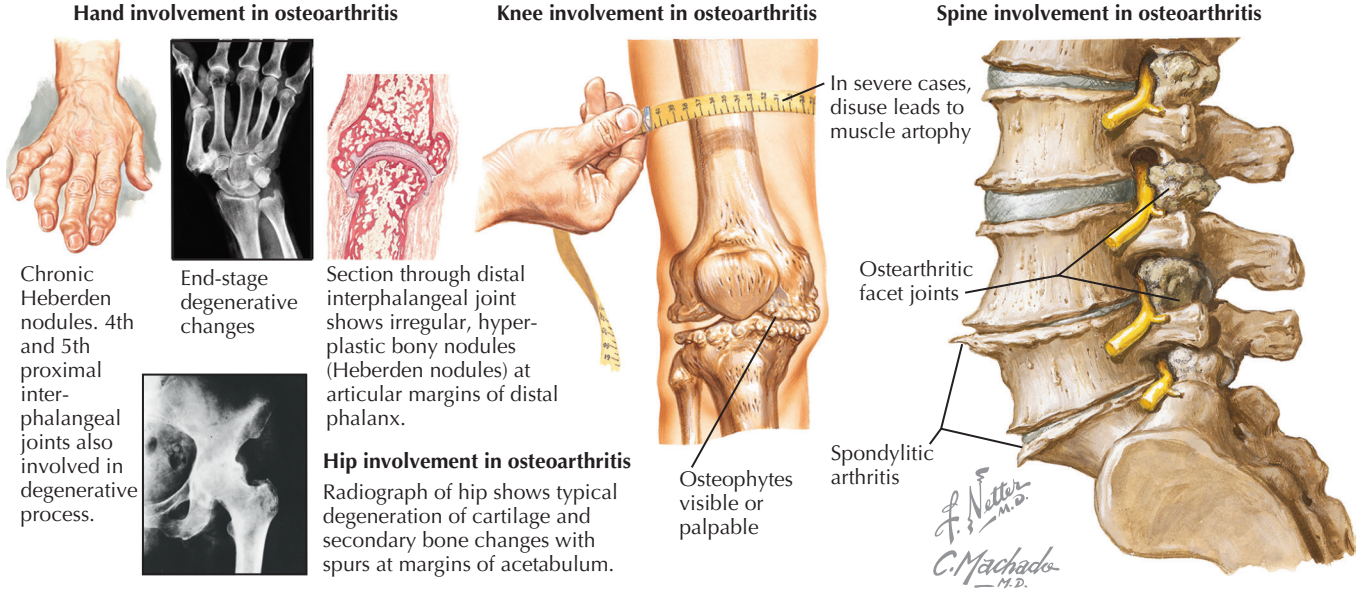



**Figure 41.5.** Characteristics of osteoarthritis or degenerative arthropathy.

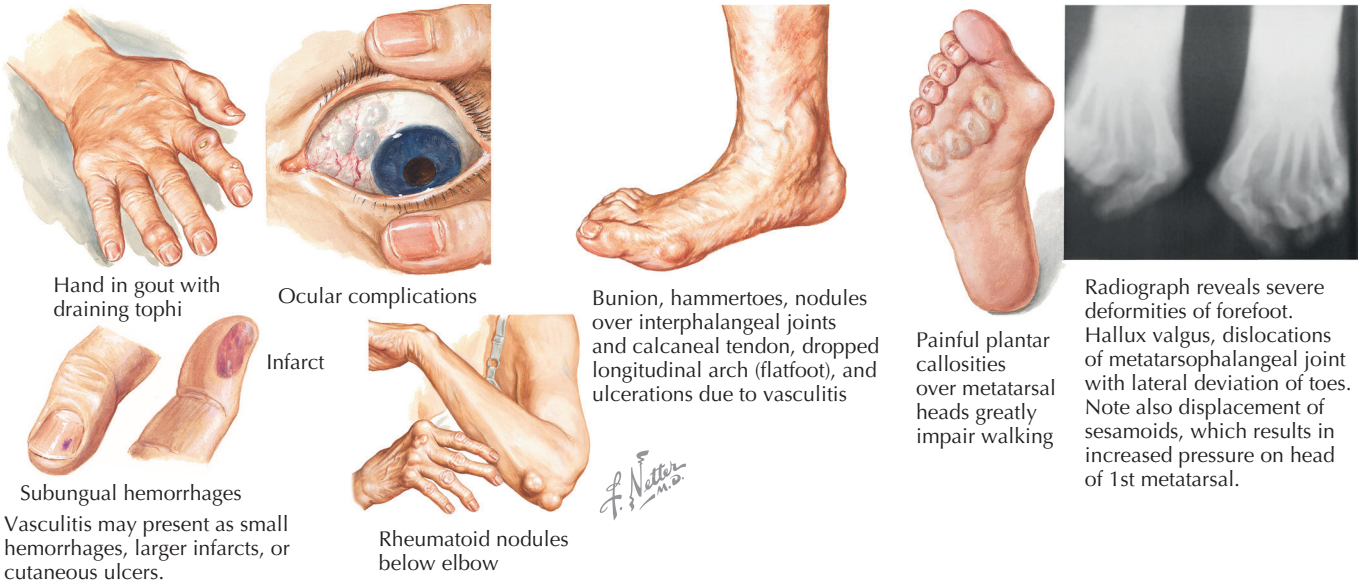



**Figure 41.6.** Characteristics of rheumatoid arthritis.

- Symmetric joint pain and swelling; destruction of joint, synovium, and surrounding soft tissue
- Swan neck deformity
- Eye involvement: Keratoconjunctivitis sicca and episcleritis (see Fig. 41.6)
- Lung involvement: Pulmonary toxicity secondary to methotrexate or gold salts
- Cardiac involvement: Pericarditis, myocarditis
- Foot deformities (see Fig. 41.6)
- Spine involvement

**Diagnostic:** History and physical examination and radiographic changes (joint space narrowing or destruction)

Serum laboratory examination: rheumatoid factor, sedimentation rate, anti-CCP ab, ANA, and CBC (anemia [normocytic hypochromic]) and neutropenia (consider Felty's syndrome)

The American College of Rheumatology Classification Criteria includes the following clinical features for diagnosis: inflammatory arthritis of ≥3 joints for >6 weeks, positive RA and/or anti-CCP, elevated CRP or ESR, and exclusion of other diseases with similar clinical features.

**Treatment:**

- DMARDs: methotrexate, leflunomide, sulfasalazine, and minoclycline
- Biologic DMARDS
  - TNF-alpha inhibitors: infliximab, adalimumab, etanercept, golimumab, and certolizumab pegol
  - Other monoclonal antibodies: abatacept and rituximab
  - Targeted, synthetic DMARDs: tofacitinib
  - Interleukin-6 inhibitor: tocilizumab
- Prednisone oral burst then taper
- Intra-articular injections: steroids and hyaluronic acid
- Physical therapy
- Physical activity and healthy lifestyle
- Orthotics for foot deformities/pain
- Joint replacement surgery, when conservative measures fail

**Prognosis:** Depends on severity, responsiveness to medications, and juvenile versus adult onset; in several cases, juvenile-onset RA may "burn-out" and become quiescent in early adulthood.

**Return to sport:** Avoid combative/collision/contact sports, particularly during the early phases of the condition as DMARDs are starting to work.

- Disease quiescent with complete functional recovery: moderate to vigorous activity (e.g., running)
- Disease quiescent with minimal to moderate crippling: mild to moderate activity (e.g., swimming and cycling)
- Asymptomatic with effusion and synovial changes in weight-bearing joints in active therapy: mild activity with participation at own tolerance
- Few evidence-based studies have shown that moderate athletic activity (e.g., swimming and cycling) had no negative impacts on deterioration of hand function and preserved hand function compared to nonactive controls.

## Systemic Lupus Erythematosus (SLE)

**Overview:** Systemic lupus erythematosus (SLE) is a chronic inflammatory immunologic disease that can affect multiple organ systems, including dermatologic, pulmonary, cardiac, renal, immune, musculoskeletal, and nervous systems, with the production of antinuclear antibodies (ANAs).

**Presentation:** SLE presents more often in young women than men. The organ systems that are involved early in the disease are the same organs systems that continue to be involved later in the disease process. Most patients present with fatigue, fever, myalgias, nonspecific abdominal pain and/or weight loss, and migratory asymmetrical arthralgias; the small joints of hands, wrists, and knees are the most frequently involved.

**Physical examination and diagnostics:** See Table 41.2

**Treatment:**

- Fever usually responds to NSAIDs, acetaminophen, or low-dose corticosteroids.
- Limit exposure to sunlight
- Difficult to differentiate between true SLE and drug-induced SLE. Review all recent medications/supplements.
- Infections may initiate or cause a relapse of SLE.
- Hydroxychloroquine or chloroquine
- NSAIDs and/or short-term use of low-dose glucocorticoids
- Other medications may include cyclophosphamide, methotrexate, azathioprine, mycophenolate, belimumab, and rituximab.

**Prognosis:** Variable, can be episodic with long periods of remission to rather progressive and debilitating; occasionally leads to significant morbidity and some mortality

**Return to sport:** Protect from sun exposure because this can worsen or prolong dermatologic manifestations such as rashes. Assess the patient for signs of renal involvement by looking

**TABLE 41.2 SYSTEMIC LUPUS ERYTHEMATOSUS CLASSIFICATION CRITERIA**

| Criteria | Clinical Criteria | Immunologic Criteria |
|---|---|---|
| American College of Rheumatology (ACR): must satisfy at least 4 of 11 criteria | Malar rash<br>Photosensitivity<br>Discoid rash<br>Oral ulcers<br>Arthritis<br>Serositis (pleuritis or pericarditis)<br>Renal disorder (proteinuria >5 g/24 hours or cellular casts)<br>Neurologic disorder (seizure or psychosis)<br>Hematologic disorder (hemolytic anemia, leukopenia, lymphopenia, or thrombocytopenia) | Elevated ANA titer<br>Antibodies (Anti-DNA, Anti-Sm, or Antiphospholipid) |
| Systemic Lupus International Collaborating Clinics (SLICC): must satisfy 4 of 17 criteria, including at least 1 of 11 clinical criteria and 1 of 6 immunologic criteria, OR have a biopsy-proven nephritis compatible with SLE in the presence of ANA or anti-dsDNA antibodies | Acute cutaneous lupus (malar rash or subacute)<br>Chronic cutaneous lupus (e.g., discoid rash, lupus panniculitis, chilblains lupus)<br>Nonscarring alopecia<br>Oral or nasal ulcers<br>Joint disease (synovitis or tenderness of ≥2 joints)<br>Serositis (typical pleurisy or pericardial pain for >1 day)<br>Renal (proteinuria >5 g/24 hours or red blood cell casts)<br>Neurologic (seizures, psychosis, mononeuritis multiplex, myelitis, peripheral or cranial neuropathy, or acute confusional state)<br>Hemolytic anemia<br>Leukopenia or lymphopenia<br>Thrombocytopenia | Elevated ANA titer<br>Anti-dsDNA antibody<br>Anti-Sm antibody<br>Antiphospholipid antibody<br>Low complement (C3, C4, or CH50)<br>Direct coombs test (in absence of hemolytic anemia) |

After excluding alternative diagnoses, SLE can be diagnosed using either ACR criteria or SLICC criteria. From Petri M, Orbai AM, Alarcón GS. Derivation and validation of the Systemic Lupus International Collaborating Clinics classification criteria for systemic lupus erythematosus. *Arthritis Rheum.* 2012;64(8):2677-2686.

for proteinuria, calculating glomerular filtration rate via plasma creatinine levels, and rarely, by doing a renal biopsy on a scheduled basis if patient suffers a flare. Discourage patient from participating during a flare that includes fever because of increased risk of endocarditis, vasculitis, and worsening synovitis. Baseline echocardiogram and subsequent follow-up echocardiograms prior to clearing for endurance or competitive sports with moderate to severe intensity to look for any valvular insufficiency. Patients with a long history of SLE should be screened before starting a strenuous exercise regimen because they are at a higher risk of developing coronary artery disease. Stress, surgery, and pregnancy have been known to trigger relapses, so caution is warranted during such events.

## OTHER RHEUMATOLOGIC CONDITIONS

### Buerger's Syndrome (Thromboangiitis Obliterans)

**Overview:** Nontraumatic, nonatherosclerotic inflammatory occlusion of small-to-medium-sized arteries or vasculitis. It often presents with pain and claudication of lower extremities and is associated with heavy smokers and men younger than 50 years; also seen in users of smokeless tobacco.

**Etiology:** Unknown, but possibly immunologic phenomenon with vasodysfunction and inflammatory thrombi

**Incidence:** The incidence of Buerger's is approximately 12.6 per 100,000 persons in the US. Incidence ranges from 1% to 5% in the population of Western Europe and approximately 80% among Ashkenazi Jews in Israel with peripheral arterial disease.

**Presentation:**
- Male-to-female ratio: 3 : 1
- Coldness, numbness, and cyanosis of hands and feet
- Cold weather exacerbation
- Multiple amputations and/or ischemic ulcers
- Arch pain (most common site of foot claudication)
- Pain at rest (at advanced stages)
- Three phases
  - Acute phase: Inflammatory
  - Intermediate phase: Progressive organization of thrombus
  - Chronic phase: Thrombus and fibrosis, no inflammation

**Physical examination:** See Fig. 41.7
- Mottled skin appearance
- Ulcers or gangrene on distal extremities
- Delayed capillary refill in extremities (2 seconds)
- Abnormal Allen test
- Tests for thoracic outlet syndrome (Wright's, Addson's, and Roos) may be positive
- Decreased or absent distal pulses
- 20%–50% will also have migratory thrombophlebitis
- To differentiate atherosclerosis from Buerger's
  - Buerger's usually affects both upper and lower extremities
  - Buerger's does not affect proximal arteries
  - Associated with tobacco use; improves when tobacco use stops
  - History of thrombophlebitis
  - Age <45 years

**Diagnostic:**
- Serum laboratory examinations (normal): ESR, CRP, ANA, complement, RF, Anticentromere Ab, Scl-70 Ab, coagulopathy panel, serum cotinine (metabolite of nicotine) level, and toxicology panel (cocaine, amphetamine, cannabis)
- Histopathologic examination of vessel identifies acute-phase lesions in a patient with an active tobacco history.
- Arterial angiography
  - Early findings: Consistent with claudication
  - Late findings: Show multiple fine "cork-screw"-shaped branches of distal arteries that end abruptly

**Treatment:**
- Eliminate contact with *all* tobacco products.
- Echocardiogram to screen for intracardiac thrombus.
- Common methods to assist patient quit smoking include hypnosis, nicotine replacement (via oral, inhaled, or transdermal), bupropion (Zyban), varenicline (Chantix), etc.

- Symptoms should improve if patient is compliant.
- Wear appropriate footwear and inspect feet daily.
- Avoid cold weather and vasoconstricting drugs.
- Iloprost decreases risk of amputation.
- Surgery for revascularization is sometimes considered if patient is noncompliant with smoking cessation. Results are often less than optimal.
- Amputation if gangrene or osteomyelitis occurs.
- Clinical trials for phVEGF 165 (vascular endothelial growth factor) and phosphodiesterase type 5 inhibitors (tadalafil and sildenafil) are underway and may show promise.

**Prognosis:** Good, if patient stops smoking

**Return to sport:** Once symptoms have begun to abate, then slow and progressive return to mild to moderate exercise is encouraged. Current literature review shows no evidence-based studies that recommend either sports participation or return to play.

Ischemic finger of a young male patient with Buerger's syndrome

Ischemic toe of a 28-year-old woman with Buerger's syndrome

**Figure 41.7.** Characteristics of Buerger syndrome. (Thromboangiitis Obliterans) (Photograph from Goldman L, Ausiello D, Arend W, et al. *Cecil Textbook of Medicine*, 23rd ed. Philadelphia: Elsevier, 2007.)

## Polymyalgia Rheumatica (PMR)

**Overview:** A rheumatic condition that is associated with giant cell (temporal) arteritis (GCA), polymyalgia rheumatica (PMR) is characterized by chronic aching and morning stiffness in shoulders, hip girdles, neck, and torso in older persons.

**Presentation:** Prevalence is approximately 700 per 100,000 in persons older than 50 years. Of those with PMR, 10% also have GCA. In addition to the shoulder and hip stiffness and achiness, patient complains of malaise, fatigue, weight loss and anorexia, and mild fever.

**Physical examination:** See Fig. 41.8

- Decreased active range of motion at shoulder, neck, and hips
- Normal muscular strength, though weakness/atrophy has been noted because of disuse
- Often shoulder, neck, and hip musculature is not tender.
- Tenosynovitis around carpel tunnel has been noted.
- Soreness in affected joints seems to be related to a bursitis/synovitis.

**Diagnostic:**

- Criteria, typical:
  - Be older than 50 years
  - Have bilateral, aching, morning stiffness in neck, shoulders, or hips for 30 minutes
  - Have ESR of ≥40 mm/hr
- *Differentiation from GCA:* PMR rarely expresses temporal tenderness, headache, jaw pain, visual loss, and noncranial ischemia (e.g., arm claudication)
- Hematologic: ESR is elevated, often >100 mm/hr, but up to 20% may have an ESR of ≤40 mm/hr.
- Elevation of CRP

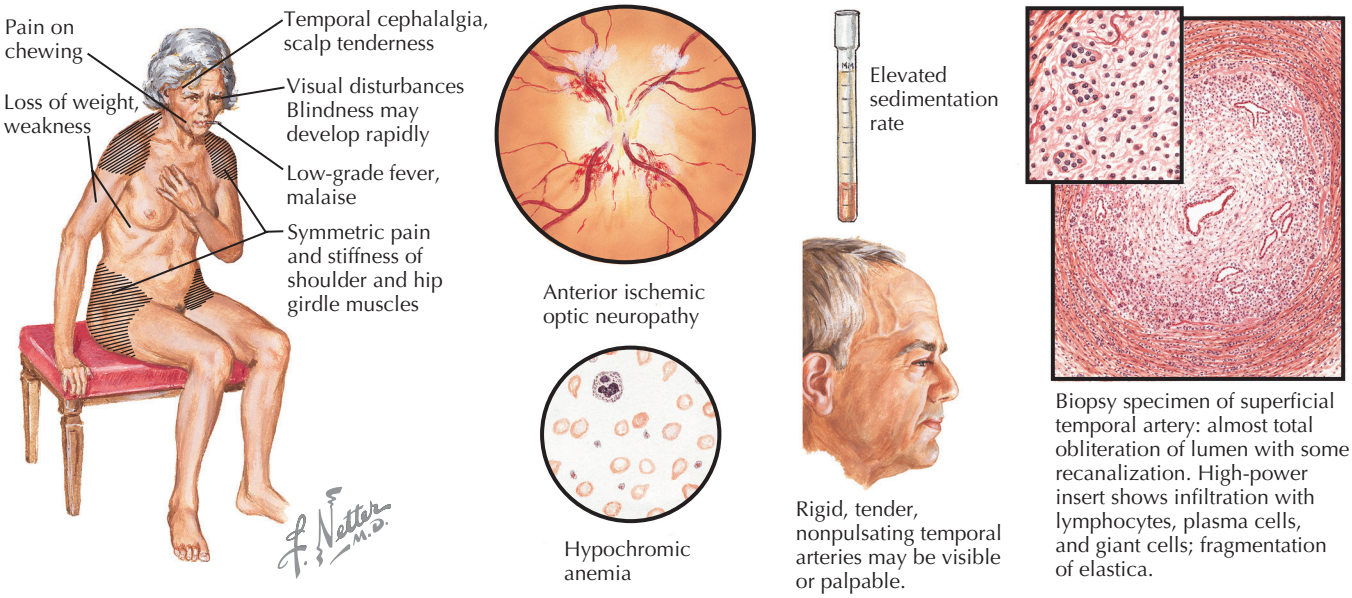



**Figure 41.8.** Characteristics of polymyalgia rheumatica and giant cell arteritis.

- Immunologic: PMR and GCA share the polymorphism on HLA-DR4. A secondary hypervariable region of the HLA-DRB1 gene has been associated with PMR. IL-6 may be elevated.
- Imaging:
  - Radiographs: rarely reveal any changes of inflamed joints
  - MRI: May show inflammation of tenosynovial sheaths or bursae
  - Ultrasound: Effusions of shoulder bursa
  - PET scan: No proven clinical value in the care of patients with PMR

**Treatment: Initial:** moderate doses of corticosteroids (7.5–20 mg daily); **Chronic:** lower doses of corticosteroids are used (note: *ESR, CRP, and IL-6 levels may return to normal once corticosteroid treatment is started);* **Additional agents:** methotrexate, infliximab show no proven benefit

**Prognosis:** In most patients, PMR runs a self-limited course over months to years and corticosteroids can be discontinued. Ongoing surveillance for development of GCA symptoms should be assessed.

**Return to sport:** Once a patient has been identified and appropriately treated, there are relatively few contraindications for participation outside of the issues associated with chronic steroid use (e.g., weight gain, glucose intolerance, osteoporosis, etc.).

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# MUSCULOSKELETAL INJURIES IN SPORTS

# 42

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## GENERAL CLASSIFICATION OF MUSCULOSKELETAL SPORTS INJURIES

Musculoskeletal sports injuries can be classified as **traumatic** or **overuse** injuries.

### Traumatic Injuries

**Description:** Result from specific episode(s) of trauma, whether recent (acute) or in the more distant past (subacute or chronic)

#### Bone

**Description:** Traumatic injury to a bone most commonly results in **fracture,** although rarely, another result occurs, such as **subperiosteal hematoma.**

**Descriptive terms:**

- **Closed fracture** is a fracture that does not produce an open wound in the skin.
- **Open fracture** is when an open wound in the skin communicates with the fracture site.
- Descriptive terms for direction of fracture line:
  - Fracture at right angles to the long axis of a bone is called **transverse.**
  - Fracture line at other angles to the long axis of a bone is called **oblique.**
  - Bone twisted apart creates **spiral** configuration of fracture.
- **Comminuted fracture** is when a bone is broken into three or more pieces.
- **Avulsion fracture** is a "pull-off" fracture; a piece of bone is pulled off by the ligament or by tendon attachment.
- **Greenstick fracture** is an incomplete fracture in children: one side of a bone is broken, whereas the other side appears bent.
- **Torus fracture** is localized buckling in the cortex of the bone, common in children.
- **Epiphyseal fracture** is a fracture that involves the growth center at the end of a long bone in children.

#### Joint

**Description:** Traumatic injury to joint and supporting structures (capsule or ligaments) often results in an instability episode referred to as **dislocation** or **subluxation.** Rarely, another result occurs from a direct blow, such as joint contusion or hemarthrosis.

**Classification:**

- **Dislocation** is complete displacement of joint surfaces so that they no longer make normal contact at all; important to distinguish **first-time** or **recurrent** dislocation
- **Subluxation** is partial displacement of joint surfaces, usually transient in nature; important to distinguish **first-time** or **recurrent** subluxation
- Dislocation or subluxation implies damage to ligaments or other supporting structures of a joint; important to ascertain injury to those tissues; discussed in the following section

#### Ligament

**Description:** Traumatic injury to a ligament is referred to as **sprain.**

**Classification:**

- **First-degree sprain:** Tear of only a few ligament fibers; mild swelling, pain, disability; no instability of joint created
- **Second-degree sprain:** Tear of a moderate number of ligament fibers, but ligament function is still intact; however, ligaments may be somewhat stretched. Moderate amount of swelling, pain, disability; slight to no instability of joint
- **Third-degree sprain:** Complete rupture of a ligament; severe swelling and disability; definite joint instability; instability may be classified as:
  - **1+** joint surfaces normally stabilized by ligament(s) displaced 3–5 mm from their normal position
  - **2+** joint surfaces separated by 6–10 mm
  - **3+** joint surfaces separated by >10 mm

#### Muscle–Tendon Unit

STRAIN

**Description:** Traumatic injury to muscle or tendon caused by **indirect force** (i.e., contraction of muscle itself) is referred to as **strain**

**Classification:**

- **First-degree strain:** Tear of only a few muscle or tendon fibers; mild swelling, pain, disability; can also be characterized by patient's ability to produce strong, but painful, muscle contraction
- **Second-degree strain:** Disruption of moderate number of muscle or tendon fibers, but muscle–tendon unit still intact; moderate amount of pain, swelling, disability; characterized by patient's weak and painful attempts at muscle contraction
- **Third-degree strain:** Complete rupture of muscle–tendon unit; may be at origin, muscular portion, musculotendinous junction, within tendon itself, or at tendon insertion; characterized by extremely weak attempts at muscle contraction

DEEP MUSCLE CONTUSION

**Description:** Traumatic injury to muscle caused by **direct force** may produce deep muscle contusion; typically affects quadriceps or brachialis muscles involved in contact or collision sports; may lead to **myositis ossificans** and therefore permanent loss of function

MYOSITIS OSSIFICANS

**Description:** Heterotopic bone formation caused by deep muscle contusion or strain, particularly after marked hematoma formation

**Common sites:** Quadriceps; biceps, triceps, brachialis; hip girdle; groin; and lower leg

**Risk factors:** Severe contusion; continuing to play after injury; massaging injured area; early application of heat; passive, forceful stretching; overly rapid rehabilitation; premature return to sport; re-injury of same site; individual propensity to heterotopic bone formation

**Calcification:** Follows injury by 3–6 weeks

- May continue to develop for ≥6 weeks
- May remodel or reabsorb over 3–12 months, particularly if close to musculotendinous junction

**Treatment:**

- Treat strain or contusion with basic athletic first aid (see Chapter 43, Comprehensive Rehabilitation of the Athlete) represented by the mnemonic **PRICES: p**rotection, **r**est, **i**ce, **c**ompression, **e**levation, and **s**upport; followed by progressive symptom-guided rehabilitation

- **Excision rarely necessary**
  - Warranted only in cases of persistent weakness or limited range of motion
  - Only after calcification matures (6–12 months)
  - High rate of recurrence if excised too early

#### EXERTIONAL RHABDOMYOLYSIS

**Description:** Breakdown of skeletal muscle cells with leakage of cellular contents, including myoglobin, creatine kinase (CK), and aldolase through damaged sarcolemma into serum as result of prolonged, heavy, or repetitious exercise

**Presentation:** Muscle pain and tenderness, muscle swelling, muscle cramps, and reddish brown urine

**Laboratory tests:** Urinalysis (urine dipstick positive for hemoglobin; microscopic exam reveals few to no red blood cells); elevated CK (normal: 200 U/L; subclinical to mild rhabdomyolysis: 600 U/L; most clinical cases: 10,000 U/L; and severe cases: reports 200,000 U/L); and elevated serum myoglobin (normal, 5–70 g/L; elevated only up to 6 hours after injury if renal function is normal)

**Etiology:** Intense exercise causes local tissue hypoxia, resulting in elevation of adenosine triphosphate (ATP) and consequent failure of sodium–potassium pump with potassium efflux and calcium influx; anaerobic glycolysis with lactic acid overproduction and, in severe cases, metabolic acidosis; sarcolemma permeability; and, in severe cases, cell death

**Common predisposing features:** Heat, humidity, dehydration, poor physical conditioning (may also occur in well-conditioned athlete with very intense or repetitive exercise), high altitude, recent viral infection, sickle cell trait, and hereditary defects in ATP synthesis

**Clinical course:**

- Most cases are **subclinical** and never diagnosed or **minimal** (visible myoglobinuria without muscle pain and spontaneous healing)
- **Mild-to-moderate cases** respond well to aggressive hydration; without nephrotoxic cofactors, dehydration, and acidosis, damage is usually not persistent; lasting systemic complications are rare; muscle has remarkable capacity for repair
- **Severe cases** are common in patients with dehydration or acidosis; they are characterized by **systemic complications:**
  - **Acute tubular necrosis and renal failure** caused by combination of renal hypoperfusion, acidosis, and myoglobin sludging in renal tubules
  - **Acute compartment syndrome** (rhabdomyolysis causes muscle swelling, which increases intramuscular pressure, causing vicious cycle of damage; in athletes with chronic compartment syndrome, increased compartment pressure may reduce tissue perfusion and cause rhabdomyolysis)
  - Hyperkalemia
  - Hypocalcemia
  - Hyperphosphatemia
  - Diffuse intravascular coagulopathy (DIC)
  - Cardiac dysrhythmia

**Treatment:**

- **Aggressive early hydration** to maintain renal perfusion and clear myoglobin, thereby preventing acute tubular necrosis and renal failure
  - May require 4–12 L of normal saline during first 24 hours
  - Diuretic (furosemide) may be necessary to maintain kidney function
- **Alkalinization of urine with bicarbonate**
  - Myoglobin is less nephrotoxic and more soluble in alkaline urine
  - Uric acid is more soluble and less likely to crystallize in alkaline urine
  - **Caveat:** Alkalinization with bicarbonate may increase precipitation of calcium in injured muscles, causing heterotopic bone formation. Safer approach may be oral acetazolamide, 250 mg, three times daily, if plasma bicarbonate level is >13–15 mEq/L.
- Correct hyperkalemia, hyperphosphatemia, and hypocalcemia. Avoid IV calcium, except to treat tetany, because of the risk of heterotopic bone formation.
- **Fasciotomy,** if necessary for compartment syndrome
- Treat DIC if it does not resolve spontaneously
- Dialysis if necessary

### Other Soft Tissues

**Description:** Traumatic injury to bursa with bursal swelling is referred to as **traumatic bursitis,** it is usually caused by bleeding into bursa. Traumatic injuries to other soft tissues include various **contusions** and **hematomas.** Lacerations may involve musculoskeletal tissues.

**Shearing injuries:** Avulsions, abrasions, or blisters

#### MOREL–LAVALLÉE LESION

**Description:** A posttraumatic, closed, degloving soft tissue injury in which the skin and subcutaneous tissues are separated from fascia superficial to the underlying muscle plane creating a space that fills with fluid and is susceptible to necrosis and infection.

**Presentation:** Diagnosis of Morel–Lavallée lesions is often missed or delayed. There may be a large swollen area where a hematoma develops often in the pelvis area. There is soft tissue swelling with or without ecchymosis. Typically, there is asymmetry of the skin contour and hypermobility. Underlying soft fluctuance is also present with minimal or absent tenderness. Often, chronic lesions will have decreased sensation and cracked areas on the skin.

**Imaging:** MRI is the preferred imaging modality of choice. In acute lesions, fluid collection will be a homogeneous collection that is hypointense on T1-weighted images and hyperintense on T2-weighted images. In chronic situations, the fluid will be hyperintense on both T1- and T2-weighted images (Fig. 42.1).

**Etiology:** A traumatic blow or sudden shearing force to any area with strong underlying fascia, most often around the pelvis or lower limb; the shearing force causes the thick layer of subcutaneous fat and skin to be ripped from its fascia underneath. As this occurs, the perforating vessels and lymphatics from the underlying muscle are torn and bleeding and lymphatic fluid leak into a newly created cavity that has difficulty draining. The pressure of this area can cause skin and underlying necrosis. High-energy mechanisms are responsible for >50% of lesions.

**Treatment:**

- **Acute:** In isolated lesions, aspiration and compression may be acceptable. With any signs of infection, fracture, or skin necrosis, a percutaneous or open incision and drainage should be performed in the operating room.
- **Chronic:** Asymptomatic lesions can be observed and conservatively treated. Symptomatic lesions can be treated with percutaneous or open incision and drainage and may need sclerotherapy and possibly deadspace closure.

## Overuse Injuries

**Description:** Account for >50% of injuries seen in sports medicine practices

**General overuse concepts:** If viewed as a function of Newton's third law of motion, athletic injury can be described as resulting from equal and opposite reactions, which, in turn, result in macrotrauma or microtrauma.

- Macrotrauma: Equal and opposite forces exceed strength of specific anatomic structure, and therefore, the structure fails (see Traumatic Injuries).

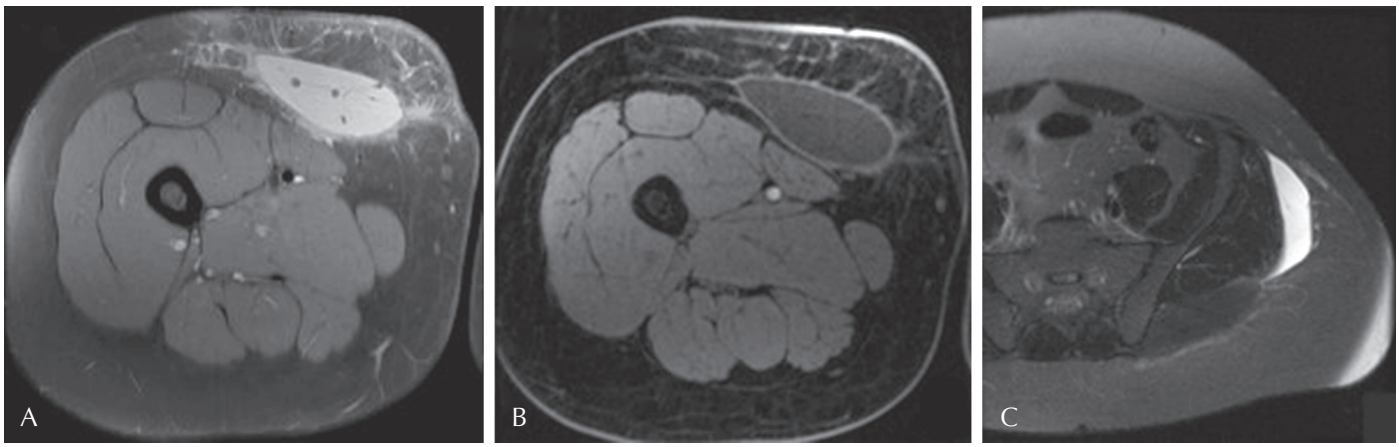



**Figure 42.1.** MRI demonstrating chronic subcutaneous fluid consistent Morel–Lavellee lesion on anterior lateral thigh. (From Greenhill D, Haydel C, Rehman S. Management of the Morel-Lavallée lesion. *Orthop Clin North Am.* 2016;47[1]:115-125.)

- Microtrauma: Microscopic subliminal injury from repeated activity; can be cumulative over time and result in inflammation; characterized by pain and dysfunction
- **Predisposition:** Equally important are intrinsic or extrinsic factors that predispose the athlete to overuse injury.
  - **Intrinsic:** Malalignment of limbs, muscular imbalances, and other anatomic factors
  - **Extrinsic:** Training errors, faulty technique, incorrect surfaces and equipment, and poor environmental conditions
- **Degenerative processes:** May influence traumatic injuries as well, but more commonly have effect on overuse injuries; normal degenerative processes occur in many musculoskeletal tissues with aging; may add to likelihood of certain injuries such as rotator cuff and Achilles tendon problems
- **General classification:** Overuse injuries are classified according to four stages, depending on pain:
  - **Stage 1:** Pain after activity only
  - **Stage 2:** Pain during activity, does not restrict performance
  - **Stage 3:** Pain during activity, restricts performance
  - **Stage 4:** Chronic, unremitting pain, even at rest

## Bones

**Description:** An overuse injury of bone may be stress fracture or apophysitis.

**Classification:**

- **Stress fracture:** Most often found in lower extremity, but can also be found in spine and upper extremity when it is subjected to weight-bearing (e.g., gymnastics, weight training)
- **Apophysitis:** In skeletally immature athletes, traction injuries can affect apophysis. Appear to result from repeated stress at tendinous insertion into bony growth center, followed by reactive bone formation; most common apophysitis is Osgood–Schlatter disease (see Chapter 51)

### Joints

**Description:** Overuse joint injuries almost invariably result from mechanical factors. Although they may create a condition that could be called "arthritis," it may be more valid when treating athletes to think of it as **synovitis.** Synovitis may be generalized, with swelling, warmth, pain, and occasionally, redness.

### Ligament

**Description:** There are a few examples of pure overuse injuries to ligaments. Theoretically, they may occur whenever a ligament is subjected to repeated stress. Examples include:

- **Medial elbow injuries:** Part of this spectrum may include overuse injury to medial collateral ligament of elbow, resulting from repetitive throwing with valgus loading.
- **Breaststroker's knee:** Probably most common example of pure ligament injury through overuse; typically involves medial collateral ligament of knee at femoral attachment, secondary to a breaststroke kick
- **Plantar fasciitis:** Technically a ligament connecting bone to bone, the plantar fascia is commonly involved in overuse syndromes of the foot (see Chapter 60: Foot Problems).

**Description:** Overuse injury of a muscle–tendon unit may be myositis, tendinitis, or tenosynovitis.

**Classification:**

- **Myositis** overuse injuries of muscle tissues are rather nondescript; can involve practically any muscle in the body
- **Tendinitis** is inflammatory reaction within the tendon tissue itself. Closely related to the concept of normal aging and degenerative changes within tendons (tendinosis), which may predispose to microtrauma. Common examples are bicipital tendinitis, lateral epicondylitis, and Achilles tendinitis.
- **Tenosynovitis (peritendinitis)** is an inflammatory change that involves tissue surrounding the tendon itself. Classic physical finding is crepitation or "dry leather creaking" sensation over the involved tendon as the tendon is moved through its sheath. Common locations include extensor tendons of forearm and tibialis anterior in lower leg.

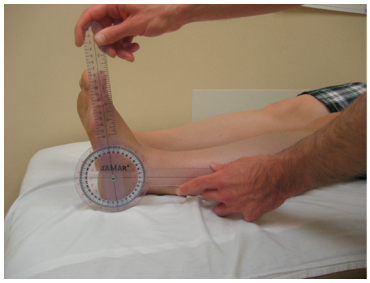
### Other Soft Tissues

**Description:** Most common overuse musculoskeletal injury involving other soft tissue is **bursitis.** Bursae lie between tissue planes and help to reduce frictional stress between those structures. Common sites for mechanical bursitis in athletes include subacromial bursa, greater trochanteric bursa of hip, and retrocalcaneal bursa in the ankle.

## GENERAL TREATMENT OF MUSCULOSKELETAL INJURIES

- Basic athletic first aid: Use the PRICES mnemonic (discussed earlier).
- **Nonsteroidal anti-inflammatory drugs** (NSAIDs) are commonly used in treating musculoskeletal sports injuries. Several different types and brands exist. Choice should always be tempered by known side effects (e.g., renal damage).
- **Physical modalities:** Cold, heat, ultrasound, iontophoresis, and electrical muscle stimulation are commonly used.

**Flexibility testing**



Heel cord flexibility

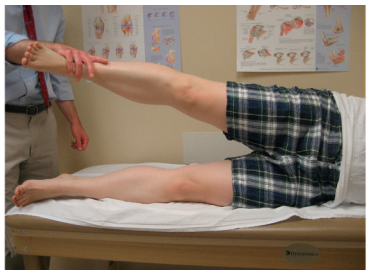
Hamstring flexibility

Quadriceps flexibility

Iliotibial band flexibility

**Strength testing**

Hip flexion strength



Hip abduction strength

Ankle dorsiflexion strength

Supraspinatus strength

**Figure 42.2.** Musculoskeletal Evaluation Techniques.

- **Therapeutic exercises:** Most important, but the most commonly underused means of treating musculoskeletal sports injuries; important to correct not only deficits that may result from injury, but also those that predispose to injury
- **Injection therapy:** Most commonly injected material is corticosteroid, with or without local anesthetic. **Studies have demonstrated a direct harmful effect of steroids on articular cartilage and a weakening effect on tendon** (see Chapter 56).
  - **Never inject corticosteroid into major load-bearing tendons** (e.g., patellar tendon or Achilles tendon); **doing so may hasten rupture.**
  - **Acceptable** to inject corticosteroid into muscular trigger points, bursae, and small, nonweight-bearing joints (e.g., acromioclavicular joint) and large joints (e.g., knee, shoulder); however, numerous injections in a normal large joint is not recommended; also acceptable to inject corticosteroid into **muscular** attachments into bone, such as lateral epicondyle (**total number** of injections should be limited); ligament attachments to bone where subsequent rupture of ligament would not be disastrous (e.g., plantar fascia attachment to calcaneus); **tendon sheath,** but not the tendon itself (e.g., for de Quervain's disease at wrist); and already degenerated joint in older athletes.
- **Braces, supports, and other devices:** Various products have been developed to aid in treatment of athletic injuries, ranging from simple compressive sleeves for various joints to expensive custom-made braces. They are discussed in chapters on anatomic parts and individual sports.
- Calcification excision: Rare

## SELECTED MUSCULOSKELETAL EVALUATION TECHNIQUES

### Flexibility Testing

**Description:** Flexibility is limited by length of muscle across joint. Lack of flexibility in two-joint muscles (muscles that cross two joints) is often indicated as cause of musculoskeletal problems. While assessing flexibility, consider whether the restriction seen is due to muscular tightness or other sources, such as lack of joint range of motion or pain.

**Heel cord flexibility:** Athlete sits with knee extended and is asked to actively dorsiflex the ankle. Measurement is made goniometrically (Fig. 42.2). Normal value is at least 10 degrees beyond plantigrade. This may also be done with the knee flexed to assess tightness within soleus (normal value is at least 20 degrees beyond plantigrade).

**Hamstrings flexibility:** Athlete lies supine with hip maintained at a 90-degree flexion and is asked to actively extend knee without repositioning the hip (see Fig. 42.2). Measurement is made goniometrically. Normal value is less than 10 degrees short of full extension.

**Quadriceps flexibility:** Athlete lies prone and knee is passively flexed by examiner. Normal value is full knee flexion without tilting of pelvis (see Fig. 42.2).

**Iliotibial band flexibility:** Athlete lies on the opposite side, near edge of the examining table, facing away from the examiner (see Fig. 42.2). Hip on the side to be examined is slightly extended and passively adducted by gravity. Normal is when the knee drops level or below the table level; also called *modified Ober's test*.

### Strength Testing

**Description:** Although there are various ways to assess strength, the authors prefer the manual muscle "break" test technique. Athlete generates maximal contraction of muscle in shortened range, and examiner applies opposite force in attempt to move athlete from testing position. Common muscle testing rule is not to apply forces across adjacent joints, but athletes are generally able to adequately support adjacent joints, thus allowing examiner to apply more force to the area in question. Strength is usually graded on a 0–5 scale (0 = zero, 1 = trace, 2 = poor, 3 = fair, 4 = good, and 5 = normal). Most athletic applications are in the

upper range of this scale and subjective in nature. Although more objective methods are available, the manual muscle test is the easiest to administer. Hip flexion, hip abduction, and supraspinatus strength tests are included because weakness may indicate a new or unrehabilitated condition more distal in the kinetic chain. Ankle dorsiflexion strength test is included because of a possible association with patellofemoral problems.

**Hip flexion strength:** Athlete sits at edge of the table with arms crossed. Athlete flexes hip and examiner performs a manual muscle "break" test (see Fig. 42.2). If a break occurs, observe whether the identified weakness is located in the hip or in the abdominal obliques.

**Hip abduction strength:** Athlete lies on the opposite side, facing away from the examiner. Athlete abducts hip and examiner performs a manual muscle "break" test (see Fig. 42.2). If a break occurs, observe whether the identified weakness is located in the hip or in the abdominal obliques.

**Ankle dorsiflexion strength:** Athlete sits with knee extended and is asked to dorsiflex the ankle. Examiner performs a manual muscle "break" test (see Fig. 42.2).

**Supraspinatus strength:** Athlete sits or stands with shoulders abducted to 90 degrees, horizontally adducted to 30 degrees, and in a "thumbs-down" position (fully internally rotated). Examiner performs a manual muscle "break" test, taking care to eliminate substitution from the trapezius (see Fig. 42.2).

## MYOFASCIAL PAIN SYNDROME

### Definitions

- **Myofascial trigger point:** Intensely irritable spot in muscle and/or adjacent fascia that stimulates and sends distress signals to the central nervous system.
  - Feels like an indurated nodule or a "ropey" taut band of muscle
  - Occurs only in characteristic anatomic sites
  - Each site has specific **"reference zones"** of radiating/referred pain or paresthesia. Reference zone pain is often the presenting complaint.
  - **May trigger a spasm–pain–spasm cycle** (discussed in later section)
  - **Active trigger point**
    - Symptomatic reference zone pain
    - Palpation reproduces both trigger point tenderness and reference zone pain
  - **Latent trigger point**
  - Tender on examination
  - No reference zone pain
- **Myofascial pain syndrome** (myofascial syndrome): Presence of one or more active trigger points with characteristic reference zone pain
- **Scapulocostal syndrome:** Clustering of trigger point spasms in the trapezius, levator scapula, and posterior cervical muscles

### Diagnosis

- **Knowledge of precise anatomic sites** of trigger points and reference zones; common trigger point sites:
  - Levator scapula, splenius capitis, trapezius, and sternocleidomastoid
  - Infraspinatus, supraspinatus, and rhomboids
  - Quadratus lumborum, gluteus medius, and tensor fascia lata
  - Biceps femoris, vastus lateralis, and adductor longus
  - Gastrocnemius/soleus
- **Initiating, precipitating, and perpetuating phenomena**
  - **Physical:**
    - Trauma (major/minor, old/recent)
    - Overuse (sports/exercise, work with repetitive motion, muscle cramps)
    - Inadequate warm-up
    - Cold exercise/work environment
    - Poor posture, poor body mechanics, anatomic abnormalities, and poorly designed or sized workstation (particularly computer worksite)
    - Disease (rheumatoid arthritis or multiple sclerosis)
  - **Mental:** fatigue, anxiety/stress, and depression
- **Palpation of trigger points:** "Rubbery" or "ropey"; indurated; tight; and exquisitely tender

### Pain–Spasm–Pain Cycle

- Trigger point activation
  - First pain
  - Then local muscle activation and fatigue
  - Thereafter, increase in pain spreads
  - Then, additional trigger points recruited
  - This causes more pain, which spreads further
- "Key" or "matrix" trigger point recruits "satellite" trigger points.

### Treatment

- **Stretch and spray:** Passive stretching using vapocoolant (fluoromethane) for distraction
- **Massage:** Deep friction or pressure (acupressure); manual, elbow, and dowel
- **Trigger point injection**
  - 0.5% procaine
    - Other local anesthetics may be myotoxic.
    - Dilute 2% procaine with three parts of normal saline.
  - "Needling" by inserting an 18-gauge needle without local anesthetic may inactivate trigger points.
  - Typically, corticosteroids do not provide additional benefits because trigger points contain no inflammatory cells.
- **Therapeutic exercise** improves strength and flexibility.
- **Ice** (heat may exacerbate trigger points)
- Ultrasound
- Muscle energy manipulation techniques

### Levator Scapula Syndrome

- Strain of levator scapula insertion with trigger point spasm of muscle body
- Treat trigger points as discussed earlier; may also need to treat muscle insertion with corticosteroid injection, iontophoresis, or phonophoresis

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 43 COMPREHENSIVE REHABILITATION OF THE ATHLETE

Kevin E. Wilk • Charles D. Simpson III • Robert A. Williams Jr.

## GENERAL PRINCIPLES

- The overall goal of rehabilitation is to enhance the recovery of injured tissues and avoid stresses that may prove deleterious to the healing process.
  - This is accomplished by understanding normal function, pathomechanics, and the healing processes of specific tissues.
- Current research and scientific evidence must establish guidelines for rehabilitation.
- Rehabilitation specialists must integrate the medical team's diagnosis and conduct a functional examination of the musculoskeletal system.

## PRINCIPLES OF REHABILITATION

### Overview

- A team approach consisting of the physician, therapist, athletic trainer, coaching staff, and the athlete is essential in the comprehensive rehabilitation of the athlete.
- Communication with the sports medicine team along with an accurate and differential diagnosis is the beginning of a successful rehabilitation process.
- Communication between the rehabilitation specialist and physician should concern the type of injury, surgical procedure performed, method of surgical fixation, results of any diagnostic tests, integrity and quality of the patient's tissue, and physician's patient-specific expectations, so the appropriate rehabilitation program can be designed and implemented.
- The rehabilitation specialist must determine specific functional impairments and specific structures involved by a thorough and systematic examination.
- The rehabilitation program must be designed based on the patient's unique response to injury and the athlete's specific functional needs.
- Program phases are designed to emphasize goals that are specific to the appropriate time frame of tissue healing at that particular point in rehabilitation:
  - Each phase will have specific goals that must be met, such as full range of motion (ROM), before progressing to the next phase.
  - Patients will reach milestones at different times, so criteria-based progression should be promoted over time-based progression.
  - Criteria-based progression assists with locating areas wherein the patient may be gradually improving and may thus need to be more heavily emphasized.
- Establishing a differential diagnosis based on involved structures and causes contributing to the lesion is a fundamental part in designing the rehabilitation program.

### Create a Healing Environment

- Clinicians must promote healing while being careful to not overstress healing tissues.
- The program must be progressive and sequential with each phase building on the prior one.
- Advancing a patient too quickly can result in inflammation, soreness, and potentially, tissue failure as opposed to controlled application of specific stresses, which can benefit healing tissues.

### Decrease Pain and Effusion

- The first goal in many rehabilitation programs is to decrease pain and effusion.
- Swelling stimulates sensory nerves and leads to an increase in the athlete's pain perception.
  - Pain and inflammation can work as muscle inhibitors, thus causing disuse atrophy the longer the effusion is present.
- Treatment options for swelling reduction include elevation, cryotherapy, high-voltage electrical stimulation, and joint compression.
- Patients with chronic joint effusion may benefit from using a knee sleeve or compression wrap to apply constant pressure while they perform everyday activities; such devices can minimize joint effusion.
- Patients with acute inflammation benefit from ice and elevation.
- Pain may play a role in muscle activity inhibition that is observed with joint effusion.
- Pain can be passively reduced through the use of cryotherapy and analgesic medications.
- Commercial cold wraps immediately after surgery can be extremely beneficial.
- Passive ROM (PROM) may also provide neuromodulation of pain during acute or exacerbated conditions.
- Therapeutic modalities such as ultrasound and electrical stimulation may be used to control pain via the gate control theory of pain.
- The speed of progression of rehabilitation, particularly weight-bearing and ROM, may affect pain and swelling; thus, any increase in pain and effusion in the involved joint should be monitored as the patient progresses and adds additional exercises (Fig. 43.1).
- New exercises should be carefully monitored to ensure the pace of rehabilitation is appropriate and the tissue is not being overstressed; this is particularly important with procedures involving articular cartilages.
- Persistent pain, inflammation, and swelling may result in long-term complications involving ROM, voluntary quadriceps control, and a delay in the rehabilitation process; thus, it is imperative that these symptoms be minimized.

### The Science of Rehabilitation

- An evidence-based rehabilitation approach should be utilized to best direct clinical care, which allows for a more predicable functional outcome.
- When progressing a patient through rehabilitation, consideration must be given to the healing tissue itself.
- Consider if the patient is ahead of schedule and has no complaints: Can the patient continue at an accelerated rate without compromising the long-term health of his/her tissue?
- Another consideration: Does someone returning at 4 months have better outcomes than someone returning at 6 months?
- Several characteristics must be considered when deciding the speed of rehabilitation:
  - The patient's age, genetics, nutrition, concomitant injuries, and unique healing characteristics can all affect the rehabilitation timeline.

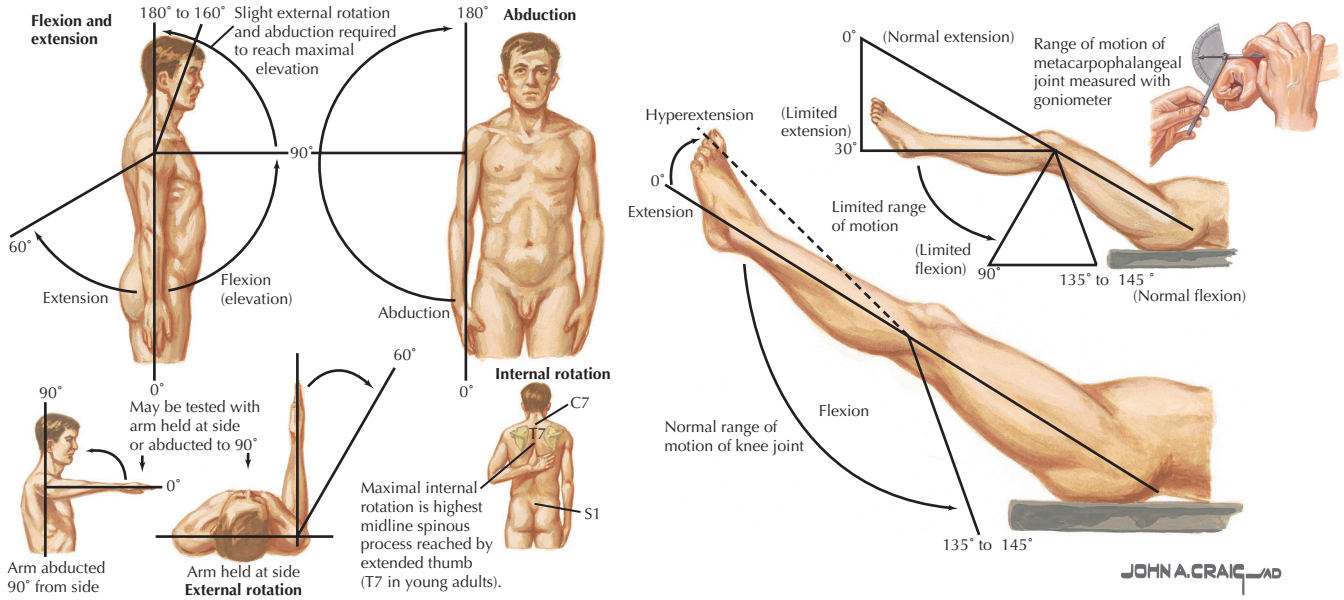


**Figure 43.1.** Range of motion.

- Injuries to the meniscus and or collateral ligaments can slow the rehabilitation process, for instance, following anterior cruciate ligament (ACL) surgery.
- Clinicians must also be aware of nonvisible concomitant injuries, such as bone bruises, that are associated with ACL injuries.

- Critical decisions have significant effects on metabolic activity of the injury site and the return to normal joint homeostasis.
- The risks and consequences of accelerated rehabilitation must be evaluated for each patient.
- The science of rehabilitation should be applied to all injuries and surgeries, particularly rotator cuff repairs, superior labral anterior posterior (SLAP) repairs, meniscus repairs, and related procedures.

## Prevent the Deleterious Effects of Immobilization

- Restriction of motion is often necessary in acute stages to promote tissue healing. ROM restriction can cause (see Fig. 43.1):
  - Quick loss of muscular girth and strength
  - Joint contracture
  - Loss of proteoglycan and weakening of articular cartilage
- "Motion is lotion for the joint."
- Deleterious effects of immobilization must be minimized and immobilization should be avoided in almost all cases.
- Current research indicates immediate controlled motion is critical to a successful outcome.
- PROM is often performed by a skilled clinician but can also be applied in the form of continuous passive motion (CPM).
- PROM can also be applied by an isokinetic device set in a PROM setting.
- CPM following surgery has several benefits, including avoidance of arthrofibrosis.

## Retard Muscular Atrophy

- Rehabilitation must also focus on retardation of muscular atrophy and facilitation of volitional muscle activity following injury or surgical procedure.
- Effusion can decrease voluntary control of surrounding musculature; this can affect the patient's ability to control his/her limbs and ambulate with a normal gait pattern.

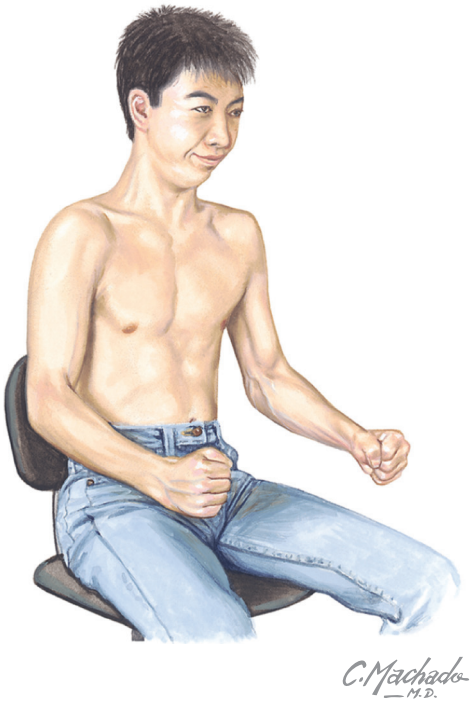


**Figure 43.2.** Isometric exercise.

- Exercises designed to enhance muscular volition begin with basic isometric contractions.
- Isometrics (Fig. 43.2):
  - Allow firing of the muscle fibers without joint motion
  - Are a safe and effective method of exercise during early rehabilitation
  - Are most often used at multiple static angles throughout the available ROM
  - Have been shown to be one of the most efficient forms of exercise to increase muscular tension and improve strength
- Muscle re-education with electrical muscle stimulation (EMS) may assist in restoring the patient's voluntary control of inhibited musculature.

- EMS is often concomitantly used during isometric and isotonic exercises to increase recruitment of muscle fibers during contraction.
- Several recent studies have found that patients that add neuromuscular electrical stimulation (NMES) to postoperative exercises have stronger quadriceps and a more normal gait pattern than nonusers.
- Biofeedback can also be used to enhance voluntary control of injured musculature.
- Clinically, NMES is used following injury or surgery while the patient performs isometric and isotonic extremity exercises.
- NMES is typically used before biofeedback when the patient presents acutely with the inability to activate the musculature.
- Once independent muscle activation is present, NMES may still be used to recruit additional motor units, thus resulting in greater strength gains.
- NMES is typically used 4–8 weeks after ACL surgery or following selected shoulder surgeries.
- Biofeedback is used for patellofemoral patients when they are unable to actively recruit their vastus medialis; the biofeedback causes the patient to concentrate on neuromuscular control.

## Restoration of Dynamic Stability

- Dynamic stability refers to the patient's ability to stabilize a joint during functional activities to avoid injury.
- Dynamic stability involves neuromuscular control and the efferent (motor) output to afferent (sensory) stimulation from the mechanoreceptors.
- Proximal stability should be established to allow for distal segmental mobility to occur within the kinetic chain.
- Dynamic stability of the glenohumeral joint is primarily achieved through interaction of rotator cuff muscles as they blend into the joint capsule.
- Contraction of the rotator cuff produces tension within the joint capsule, which centers the humeral head on the glenoid.
- Muscle weakness or strength imbalances of the posterior cuff muscles may have deleterious effects on shoulder mechanics.
- Emphasis should be placed on linking the upper extremity with the lower extremity. Exercises should link the scapula with the hip, hip with the knee, hip with patellofemoral joint, scapula with the glenohumeral joint, and the scapula with the elbow.
- Exercises to enhance dynamic stability are emphasized immediately following injury or surgery through the use of rhythmic stabilization drills.
- Alternating isometric contractions are performed to facilitate co-contractions of the anterior and posterior rotator cuff.
- Drills are progressed to include stabilization at end ROM and with the patient's eyes closed, particularly for overhead-throwing athletes, in whom dynamic stability is compromised during the throwing motion.

## Restoration of Proprioception and Neuromuscular Control

- Early proprioception and kinesthesia exercises are important for patients returning to sports because researchers have shown a decrease in these abilities following injury.
- Basic exercises designed to enhance the athlete's ability to detect the joint position and movement in space are performed to establish a baseline of motor learning for additional neuromuscular control exercises that will be integrated at a later time.

### Proprioceptive Training

- Proprioceptive training initially begins with basic exercises such as joint positioning and closed kinetic-chain weight shifting.

Mini-squats on a force platform that can provide objective feedback of the amount of weight distributed between lower extremities (Balance Trainer, Uni-Cam Inc., Ramsey, NJ).

**Figure 43.3.** Proprioceptive training.

- Joint repositioning drills begin with an athlete's eyes closed; the rehabilitation specialist passively moves the extremity in various planes of motion and then returns to the starting position.
  - The patient is then instructed to actively reposition the extremity to the location.
  - A therapist may increase the challenge to the patient's proprioceptive system by altering external stimulus such as vision and hearing.
- Weight shifting should be performed in the medial–lateral direction and in diagonal patterns.
- Mini-squats on a force platform to ensure equal weight distribution are beneficial (see Fig. 43.3).
- Advancement to minisquats on an unstable surface such as a tilt board may be used as the patient progresses.
- Several studies have shown that the wearing of an elastic bandage may have a positive effect on proprioception and joint position sense.
- As proprioception is advanced, drills to encourage preparatory agonist–antagonist co-activation during functional activities are incorporated.

### Dynamic Stabilization Drills

- Dynamic stabilization drills begin with single-leg stance on flat ground and unstable surfaces, cone stepping, and lateral lunge drills.
- The patient may perform forward, backward, and lateral cone step-over drills to facilitate gait training, enhance dynamic stability, and to train the hip to help control forces at the knee joint.
- Cone drills may be performed at various speeds to train the lower extremity to dynamically stabilize with different amounts of momentum.
- Lateral lunges are also performed, with the patient advancing from straight plane lateral lunges, to multiplanar/diagonal

**A.** Lateral lunges using a sport-cord onto an unstable surface.

**B.** Single leg balance on an unstable surface while incorporating alternating upper extremities movements with a weighted ball to alter the patient's center of gravity.

**C.** Single leg balance on a tilt-board while the patient tosses a ball against a rebound device. The rehabilitation specialist may create a perturbation by striking the board.

**D.** Rhythmic stabilization to promote co-contraction of the rotator cuff. The patient is asked to hold the arm steady while the therapist gives alternating forces to the extremity.

**E. Upper extremity lyometrics. a.** Chest pass using a trampoline and medicine ball. **b.** Single arm wall throws with the patient's arm at 90 degrees of abduction and 90 degrees of elbow flexion. **c.** Side to side throws using a medicine ball and trampoline.

**F.** Manual resistance during sidelying external resistance. The rehabilitation specialist resisted both external rotation and retraction of the scapula. Rhythmic stabilizations may also be performed at end range.

**Figure 43.4.** Dynamic stabilization drills.

lunges, to lunges with rotation, and finally to lateral lunges onto foam (Fig. 43.4A).

- CLX theraband training can be utilized to target the musculature of the hip and core. Lateral slide drills with concomitant shoulder proprioceptive neuromuscular facilitation (PNF) D2 patterns not only link the upper extremities with the lower extremities but also place emphasis on the core and hips.
- Concentration may be challenged by adding a ball toss to any of these exercises, which challenges preparatory stabilization.
- Single-leg balance exercises are progressed by altering a patient's center of gravity and incorporating movement of the upper extremity and uninvolved lower extremity (Fig. 43.4B).
- Perturbation training may also be incorporated into such exercises with single or double-leg balance exercises on a tilt board (Fig. 43.4C).
  - The patient performs an isometric hold of the tilt board with the knee flexed to 30 degrees while catching a light medicine ball.
  - The patient is instructed to stabilize the tilt board in reaction to the sudden outside force produced by the weighted ball.
  - The rehabilitation specialist can also challenge the athlete by providing manual perturbations by striking the tilt board with his/her foot to create a sudden disturbance in the static support of the lower extremity; this requires the patient to stabilize the board with dynamic muscular contractions.
- Exercises such as balance beam walking, lunges onto an unstable surface, and step-up exercises while standing on an unstable surface are also used to strengthen the knee musculature while requiring the muscles located proximally and distally within the kinetic chain to stabilize and allow coordinated functional movement patterns.
- Plyometric jumping drills are performed to facilitate dynamic stabilization and neuromuscular control (Fig. 43.4D).
  - Plyometrics use the muscle's stretch-shortening properties to produce maximal contraction following a rapid eccentric loading of the muscle tissue.
  - Plyometric training is used to train the extremities to produce and dissipate forces to avoid injury.

## Neuromuscular Control Drills

- The final aspect of rehabilitation regarding neuromuscular control involves enhancing muscular endurance.
  - Proprioceptive and neuromuscular control has been shown to diminish once muscular fatigue occurs.
  - Exercises such as bicycle, stair climbing, and elliptical machines are used to increase endurance.
  - High-repetition, low-weight resistance weight training can also increase muscular endurance.
- An additional strategy to protect the athlete from reinjury is to perform neuromuscular control drills at the end of treatment sessions after cardiovascular training.
  - This challenges the neuromuscular control of the knee joint after the dynamic stabilizers have been fatigued.
- Enhancement of neuromuscular control is equally important in the upper extremity.

- Efficient dynamic stabilization and neuromuscular control of the glenohumeral joint is necessary for athletes to avoid injuries during competition.
- Dynamic stabilization exercises for the upper extremity also begin with baseline proprioception and kinesthesia drills.
- Rhythmic stabilization is also incorporated to facilitate co-contraction of the rotator cuff and dynamic stability of the glenohumeral joint (Fig. 43.4E).
- Exercise involves alternating isometric contractions designed to promote co-contraction and basic reactive neuromuscular control (Fig. 43.4F).

- These dynamic stabilization techniques may be applied as the athlete progresses to provide advance challenges to the neuromuscular control system.
- As an athlete progresses through the program, it is necessary to train the upper extremity to provide adequate dynamic stabilization in response to sudden forces, particularly at end ROM (referred to as *reactive neuromuscular control*).

## Gradually Restore Muscular Strength and Endurance

- Gradually restore muscular strength after volitional muscle activity is achieved.
- Baseline levels of muscular strength are needed before the athlete can progress to the later stages of rehabilitation.
- Strengthening can be performed through various different methods of isotonic exercises.
  - Weight is gradually applied and increased as the athlete progressively improves strength.
- Isotonics can be used in the form of single- or multiple-joint exercises.
- Exercises can also be performed in either an open kinetic chain (OKC) or closed kinetic chain (CKC).
  - OKC exercise is defined as a movement wherein the distal extremity is not fixed, such as a leg extension.
  - CKC exercise is defined as an exercise wherein the distal extremity is fixed, such as a leg press.
  - These exercises both have a place in rehabilitation, although they have different effects on both muscular activity and biomechanics of the joint, as shown by multiple electromyographic activity studies.
- Muscular endurance is an important factor in rehabilitation programs.
  - Several activities related to athletics involve repetitive and microtraumatic events.
  - Training the musculature to endure these events is necessary to prevent injuries.
  - Fatigue has been shown to result in decreased proprioception and altered biomechanics of the joints, which may result in further pathology.

## Normalization of Soft Tissue Mobility and Flexibility

- Rehabilitation of soft tissues to restore tissue balance applies to both soft tissue around the joints, such as retinacular tissue surrounding the patella, and also muscular flexibility around each joint.
- Deviations in the balance of soft tissue forces will promote altered arthrokinematics and excessive forces to the joints.
- Muscular flexibility is vital to normal joint function because it allows the musculature to absorb force and align the joint in neutral position. For example:
  - Soft tissue tightness of the quadriceps muscle is common among people with patellar tendonitis and patellofemoral pain.
  - In the upper extremity, patients commonly experience tightness of the anterior structures, which can lead to several pathologies such as impingement syndrome.
- It is critical for the clinician to identify the causes for loss of motion and treat involved structures based on assessment.

## Emphasis on the Entire Kinetic Chain

- Rehabilitation must be focused not only on regaining strength and neuromuscular control of the affected joint but also on the surrounding areas. For example, neuromuscular control of the shoulder involves stability of not only the glenohumeral joint but also the scapulothoracic joint.
- Core stabilization drills are used to further enhance proximal stability with distal mobility of the extremities:
  - This is based on the kinetic chain concept, wherein imbalance at any point of the kinetic chain may result in pathology throughout.
  - Movement patterns, such as throwing, require a precise interaction of the entire body kinetic chain to be efficiently performed.
  - A multiphase approach is used, progressing from baseline core and trunk strengthening, to intermediate core strengthening with distal mobility, to advanced stabilization in sport-specific movement patterns.
- Imbalances of strength, flexibility, endurance, or stability may result in fatigue, abnormal arthrokinematics, and subsequent compensation.
- It is important to not neglect the uninjured extremity:
  - Numerous studies have indicated a crossover effect when the contralateral extremity is exercised, which may lead to improvements in proprioception and strength of the involved extremity.
  - The neuromuscular control system may have a certain amount of central mediating function receptive to bilateral training techniques.
  - When rehabilitating a patient with a joint injury, the rehabilitation specialist must consider having the patient perform either bilateral exercises or unilateral reciprocal exercises.

## Gradual Return to Functional Activities

- Following successful completion of the rehabilitation program, the athlete must begin a gradual return-to-sport program.
- Interval sport programs (ISP) are designed to gradually return motion, function, and confidence to the athlete after injury or surgery by slowly progressing through graduated sport-specific activities.
  - Goal of this phase is to gradually and progressively increase functional demands on the athlete.
- Criteria before returning to sport-specific activities are:
  - Full-functional ROM
  - Adequate static and dynamic stability
  - Satisfactory muscular strength and endurance
  - Satisfactory clinical examination
- Once criteria are met, a gradual return-to-sport activity is initiated in a controlled manner.
- Healing constraints based on surgical technique and fixation, as well as the patient's tissue status, should be considered.
- ISP is set up to minimize chance of reinjury and emphasize precompetition warm-up and stretching.
- There should be no set time table for completing the ISP because of individual differences.
  - Variability will exist based on skill level, goals, and injury of each athlete.
- The ISP is developed based on the specific sport and stresses observed during these athletic activities.

  - For example, overhead-throwing athletes must perform an interval throwing program that includes a limited amount of throws using a flat-ground long toss.
- Other goals are to maintain a patient's muscular strength, dynamic stability, and functional motion established in the previous phase.
  - A stretching and strengthening program should be performed on an ongoing basis to maintain and improve these goals.
- Rate of progression with functional activities is dictated by the patient's unique tolerance to activities.
  - Exercise must be performed at a tolerable level without overstressing the healing tissue—referred to as the patient's *envelope of function*.
- The athlete's return to sport-specific drills progresses through a series of transitional drills designed to progressively challenge the neuromuscular control system. Examples include:
  - Pool running before flat-ground running
  - Backward and lateral running before forward running
  - Plyometrics before running-and-cutting drills and, finally, sport-specific drills
- Integration of functional activities is necessary to train the injured patient to perform specific movement patterns necessary for daily activities.
- The intention of sport-specific training is to stimulate the functional activities associated with sports while incorporating peripheral afferent stimulation with reflexive and preprogrammed muscle control and coactivation.
  - Drills may be modified based on specific functional movement patterns that are unique to the patient's sport.
- Sport-specific training can include side-to-side shuffle, cariocas, sudden starts and stops, 45-degree cutting, 90-degree cutting, and various combination movements.
- Sport-specific patterns learned throughout the rehabilitation program are integrated to provide challenges in a controlled setting.
- Drills are performed to train the neuromuscular control system to perform during competition in a reflexive pattern.

## SPECIFIC REHABILITATION PROGRAMS

### Anterior Cruciate Ligament (ACL)

- Over a 20-year period, ACL surgery procedures have increased by 58% with 148,714 ACL surgeries performed in 2013 (PearlDiver Technologies. PearlDiver supercomputer database. Available at: http://www.pearl-diverinc.com/).
- ACL rehabilitation programs have dramatically changed over the past 20 years.
- Currently, a scientifically based program that takes into account patient's functional demands is critical to maximize outcomes.
- Emphasis is now on immediate ROM, full passive knee extension, immediate weight bearing, and activation of the quadriceps musculature through NMES.
- The rehabilitation program begins preoperatively through education of the patient and family on both the surgical procedure and rehabilitation progression.
- Preoperative goals include reduction of pain and swelling, restoration of ROM, gait normalization, and prevention of muscular atrophy.
- Postoperative rehabilitation begins 1–2 days after surgery.
- Initial sessions include the initiation of ROM activities and to assure patient that weight bearing with the use of crutches is encouraged.
  - Primary goals at this point are to reduce swelling, restore full extension, and activate quads.
- Expectations are that the patient should be able to bear full weight in a hinged brace without the use of crutches at 10–14 days after surgery.
- Full passive knee extension is attained as quickly as possible with gradual restoration of flexion ROM and patella mobility.
- OKC exercises with the use of NMES followed by incorporation of CKC exercises can be introduced 2 weeks after surgery.
- Machine weights may be introduced approximately 3–4 weeks after surgery, including leg press and multi-hip (abduction, extension, and adduction).
- Hip- and core-strengthening exercises are essential from a functional standpoint to prevent faulty biomechanical stresses on the knee. Activities should aim to control femur internal rotation, lateral trunk displacement, valgus knee collapse, and pelvic drop.
- CKC exercises and neuromuscular control drills may be progressed as tolerated to include perturbation training on unstable surfaces.
- Light plyometric jumping may be initiated 8–10 weeks after surgery.
- Functional activities such as jogging may begin shortly after plyometrics with progression to running and jumping at 10–14 weeks.
- Careful attention should be made to avoid overstressing the athlete during this phase; the development of patella tendonitis is possible, particularly if adequate quadriceps strength has not yet returned.
- Educating the female athlete on appropriate jumping and landing techniques should be included; this has been shown to reduce the incidence of future ACL injuries in the athletic population.
- Finally, the athlete can gradually return to cutting sports such as baseball, football, and tennis approximately 4–6 months after surgery, but a return to jumping sports (e.g., basketball and volleyball) should be delayed until 6–8 months after surgery.

### Superior Labral Anterior to Posterior (SLAP) Repairs

- Nonoperative rehabilitation is often successful in type I and II SLAP lesions with a sequential, multiphased program.
- Better understanding of the normal labral anatomy and healing constraints following a repair procedure will make a successful return to unrestricted function very likely.
- Successful return to the patient's prior level of function can be attained through communication by the surgeon with the rehabilitation team and patient; activity should be restricted until adequate healing response is obtained.
- Before initiating rehabilitation, a thorough subjective and clinical examination should be performed to identify the nature of the labral pathology and mechanism of injury.
  - This will aid the rehabilitation specialist after surgery while advancing the patient through the protocol.
- There are certain restrictions to a patient's rehabilitation process that can be unique based on the method of injury. For example:
  - Patients who have sustained labral injury after falling onto an outstretched hand would be encouraged to avoid closed-chain and weight-bearing activities.
  - Patients with traction injuries should avoid heavy biceps resistance activities, particularly those involving the eccentric phase of muscle contraction.
  - Overhead-throwing athletes with superior labral injuries should avoid excessive external rotation until an adequate healing time (at least 8 weeks) has passed.
- Mechanism of injury is a crucial factor to consider as the patient progresses through the rehabilitation program.

- Rehabilitation following a SLAP type II repair presents a significant challenge but should render the patient with good to excellent outcomes.
  - This type of labral lesion is commonly seen in overhead-throwing athletes with the biceps tendon detached from its glenoid rim attachment.
- Initially, the goal of rehabilitation is to control the forces placed on the healing tissue.
- Gradual passive and active-assisted ROM activities are performed below 90 degrees of flexion for the first 4 weeks (see Fig. 43.1).
  - Passive external rotation is allowed only to approximately 15 degrees in the scapular plane.
  - The patient is instructed to sleep in an immobilizer for 4 weeks.
- ROM activities are progressed beyond 90 degrees of flexion with full flexion at 6–8 weeks after surgery.
- External rotation ROM is progressed to 90 degrees of abduction at 4 weeks after surgery, and motion is gradually increased until completely restored (115–125 degrees) by approximately 8 weeks after surgery.
- Isometric strengthening activities are immediately performed to prevent muscle atrophy as a result of the immobilization.
- Rhythmic stabilization drills are also initiated with the patient in supine position and the shoulder in the scapula plane and neutral rotation.
- These activities promote dynamic stability and neuromuscular control of the humeral head within the glenoid while the scapula is stabilized by the table.
- Active joint-repositioning exercises following passive displacement are also used to enhance proprioception.
- External rotation/internal rotation tubing exercises are initiated during week 3–4 with the arm at 0 degrees abduction along with lateral raises, full can (scaption), and scapula stabilization drills.
- At approximately week 6–7, the Thrower's Ten program is initiated; this program places emphasis on external rotator and scapula strengthening.
- No isolated biceps strengthening should be performed for the first 8 weeks to protect the healing biceps attachment into the labrum.
- Isotonic strengthening can be progressed by 1 pound per 7–10 days as long as there is no increase in pain or soreness.
- More aggressive strengthening such as manual resistance exercises, PNF, and two-handed plyometrics may be added at 10–12 weeks following surgery.
- Functional activities such as one-handed plyometrics in the 90/90 position into a trampoline may be initiated approximately 2–3 weeks after initiation of two-handed plyometric drills.
- At week 12, machine weights such as lat pull-downs and seated presses may be incorporated.
- The interval hitting program may also be initiated at week 12; this comprises a gradual progression beginning with hitting of a tee, progressing to soft toss, and finally taking batting practice off a live pitcher.
- At week 16, the athlete may begin the interval throwing program.
  - Athlete begins at 45 feet and progresses to 60, 90, and 120 feet.
  - Following the 150-foot phase, the athlete may begin flat-ground throwing using pitching mechanics.
  - After completion of the flat-ground throwing step is when the athlete can begin throwing fastballs off the mound at 50% of the athlete's normal velocity.
  - Velocity and number of pitches are gradually increased.
  - The throwing program takes approximately 4–6 months depending on timing of the upcoming season, position played, or any concomitant procedures performed during surgery that may delay onset or progression of the program.
- Typically, return to play following a type II SLAP repair occurs at approximately 9–12 months following surgery.
- Rehabilitation should be based on the specific injury (mechanism or location), the surgery performed, and the ultimate functional goals of the patient.
- Gradual restoration of ROM, strength, endurance, and dynamic stability is critical, but it must be applied in a controlled manner to minimize stress on the healing tissue.
- The ultimate goal is to return the patient to prior level of function as safely and as quickly as possible without deleterious consequences.

## Ulnar Collateral Ligament (UCL) Reconstruction

- Overhead-throwing athletes are susceptible to numerous elbow injuries because of the strong forces that act on the elbow during the throwing motion.
- The ulnar collateral ligament (UCL) is the primary medial stabilizer of the elbow and is subject to high valgus stresses, particularly during the late cocking phase of the throwing motion.
- Injuries are caused by chronic stresses or repetitive microtrauma to the medial soft tissue structures that result from the athlete's attempts to stabilize the elbow joint.
- There exists a medial shear force of 300 N and a compressive force of 900 N.
  - The valgus stress that is applied to the medial elbow during the late cocking and acceleration phase of throwing is 64 N, which exceeds the strength of the UCL.
- Surgical reconstruction is employed to regain normal stabilizing function of the UCL, particularly the anterior bundle.
  - Autologous graft sources are typically either the palmaris longus tendon or the gracilis tendon.
  - Ulnar nerve transposition is often concomitantly performed at the time of reconstruction as well.
- Rehabilitation following UCL reconstruction is vital to fully restore normal function and return the athlete to competition as quickly and safely as possible.
  - Rehabilitation must be progressed to restore full ROM, strength, dynamic stability, and neuromuscular control while protecting the healing tissues.
- Rehabilitation generally commences before surgery through an educational component with the athlete and the family; this should include progression, brace use, and prognosis.
- Preoperative ROM measurements are generally obtained to gauge how much motion recovery after surgery should be expected.
- A general strength assessment is also performed of both the elbow joint and the shoulder joint musculature.
- Immediately following surgery, the elbow is placed in a 90-degree splint for the first 5 days; this allows for adequate healing of the UCL graft and soft tissue sling of the transposed ulnar nerve.
- The patient performs wrist ROM and gripping exercises to improve overall circulation of the distal upper extremity.
- After approximately 5 days, the athlete is placed in a hinged elbow brace, which is adjusted to allow 30–100 degrees of ROM.
  - Motion is increased 10 degrees in each direction per week until full ROM is obtained at approximately 4–5 weeks after surgery.
- Strengthening exercises progress from isometric contractions to isotonic exercises by week 4.
- The full Thrower's Ten program may be performed using light weights by week 6.

- Progressive resistance exercises for the elbow and shoulder dynamic stabilizers are emphasized through postoperative weeks 8–10.
- Wrist flexor and extensor stretches to increase flexibility are also incorporated at this time.
- Aggressive exercises involving eccentric and plyometric contractions are included, starting weeks 9–12.
- Two-handed plyometrics are added at week 10, and one-handed plyometrics are added approximately 2–3 weeks later.
- Interval throwing begins at approximately week 16.
- Return to competitive throwing usually occurs at approximately 8–10 months following surgery.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 44 PHYSICAL MODALITIES IN SPORTS MEDICINE

Russell G. Steves

## GENERAL PRINCIPLES

Modalities are best thought of as an adjunct to the body's own recovery process. It would be a mistake to think of them as all that any patient needs to rehabilitate. However, they do play a role in therapy, particularly in sports medicine, where any tool that hastens return to play is valuable. Some experts may claim that certain modalities are an integral part of healing and recovery from injury, although minimal evidence exists to support many claims.

## SUPERFICIAL HEAT

- Modality requires direct contact with skin.
- The greatest heating effect is achieved superficially.
- If maintained long enough, heat energy gets conducted into deeper structures.
- Increase in tissue temperature has the following beneficial effects:
  - Vasodilation
  - Increased blood flow to and from the site
  - Increased cell metabolism
  - Increased elasticity of collagen tissues
  - Decreased pain
  - Decreased muscle tone
  - Decreased muscle spasm

### Devices for Heat

- Devices conduct thermal energy across the skin to provide a rise in tissue temperature.
- **Moist heat packs**
  - Canvas covers filled with silica gel
  - Heated in hot water tanks (approximately 160°F)
  - Packs placed in insulating layers of towels or cloth covers
  - Covers have to be kept clean to prevent spread of skin disorders.
  - Heat dissipates quickly, usually within 15 minutes.
  - Easier to get the patient in a comfortable position
- **Dry heating pads**
  - Plug-in electrical or microwaveable varieties
  - Do not heat tissue as rapidly and comfortably as moist heat packs
  - Heat does not dissipate as rapidly so can be used for longer time
  - Skin can be burned by prolonged or overly intense heat exposure.
- **Whirlpools**
  - The body part immersed in a tub with a motor that moves water.
  - Water temperature usually ranges between 102°F and 110°F.
  - Lower temperatures used when more of the body is immersed.
  - More of the body part heated as it is surrounded by water
  - Water circulation keeps the temperature next to the skin constant and not dissipated into a body part.
  - Circulating water has a massaging effect.
  - Water is an excellent transmitter of bacteria into open wounds, so care must be taken to keep the wound and tank clean.
  - Whirlpool can be used for the debridement of superficial wounds.
  - Clean whirlpools thoroughly to prevent disease transmission.
- **Paraffin baths**
  - Mixture of wax and mineral oil melted to a liquid state (118°F–126°F)
  - Apply to a body part by dipping or brushing on.
  - Effective with irregularly shaped body parts such as hands and feet
  - Applying multiple coats and allowing to cool
  - Cooling wax solidifies and thereby transfers heat into tissues.
  - Low specific heat of wax allows for comfort at higher temperatures.

### Exercise for Heat

- Heat is a by-product of muscular work.
- The more intense the exercise, the greater and quicker the heating
- Heat is transmitted from muscle into other body tissues and is carried away by the bloodstream.
- Temperature increase occurs deeper in the tissues.
- A combination of exercise and superficial heat modalities results in the greatest increase in tissue temperature.

### Uses of Treatment

- Injuries no longer in the acute inflammatory phase
- Chronic pain
- Injuries resulting in decreased range of motion (ROM)
- Swelling
- In preparation for therapeutic exercise
- Injuries wherein the goal of treatment is to increase circulation

### Contraindications

- Acute injuries (risk of increased swelling)
- Uncovered open wounds
- Tumors
- Thrombophlebitis
- Nerve sensitivities

### Precautions

- Fair skin that burns easily
- Areas of decreased sensation
- Dermatologic problems and disease transmittal

## CRYOTHERAPY

- Cryotherapy involves the application of cold to affect changes in the body for therapeutic benefits. Various methods are used (see Methods of Cryotherapy).
- Each method attempts to draw heat from the body's tissues through the skin, which raises the temperature of the cold device rather than allowing the cold to penetrate the body. This

loss of thermal energy and subsequent decrease in tissue temperature results in:
  - Vasoconstriction
  - Decreased blood flow to the area
  - Decreased swelling
  - Reduction in inflammatory mediators and pain-producing substances
  - Decreased cell metabolism
  - Reduced elasticity in collagen tissues
  - Slowed conduction of nerve impulses; analgesia
  - Decreased muscle spasm
  - Decreased force production in muscle
- Because of these responses to cold application, cryotherapy is the most successful treatment modality for acute injuries. The reduction in pain, swelling, and inflammatory reaction helps the patient's healing process progress more quickly, which allows a faster return to activity. Other conditions that can benefit from cryotherapy include:
  - Injuries wherein pain is the predominant symptom
  - Postoperative conditions
  - Preexisting injuries (immediately following activity)
  - Problems wherein pain inhibits activity or therapy
  - Situations wherein anesthesia is desired
- Elevation of the injured body part to levels higher than the heart causes a decrease in vascular hydrostatic pressure. In addition, the force of gravity increases venous and lymphatic return, further reducing fluid collection in the injured area.
- The reduction in swelling at the time of injury decreases pain for the patient as well as the time taken eventually to return to full activity.
- The duration of cold application in acute injuries should not be a concern. Prolonged cold application, as occurs in the previously listed cryotherapy methods, has not been shown to result in a reflexive body warming. Longer cold application results in additional beneficial effects in acute injuries.

## Methods of Cryotherapy

- Ice bags
  - Crushed ice in plastic bags
  - Smaller ice cubes allow for greater surface coverage.
  - Less air in ice bag allows for more ice in contact with skin.
  - More conforming to the body part, which also results in greater cold effects
  - Longer application means deeper cooling
  - Application usually lasts for 15–20 minutes
- Reusable ice packs
  - Silica gel pack kept in a freezer
  - Allows for multiple uses
  - Requires an insulating layer between the pack and skin (wet towel) because packs can get colder than ice bags and result in skin irritation
  - Application is for 15–20 minutes
- Ice massage
  - Water frozen in paper cups and directly applied to skin
  - Peel paper from the cup to expose ice
  - Effective at cooling superficial tissues
  - Movement and pressure of application has similar benefits to massage
  - Limitations include smaller area of cooling and labor-intensive process
  - Application is for 10–15 minutes
- Chemical cold packs
  - Water and ammonium nitrate separated within a pack
  - Chemicals mix when barrier is broken by squeezing
  - Can be stored indefinitely and broken when needed
  - More expensive version of icing
  - Chemicals can irritate skin if bag leaks
  - Cold is not as long-lasting
- Ice immersion
  - Ice and water mixture in small container for lower legs and arms
  - Surrounds entire body part; helpful for joint injuries
  - Make immersion as cold as tolerable
  - With appendage immersion, there is less core temperature reduction
- Ice water circulating units
  - Commercially available units that combine cold and compression
  - Cold therapy can be applied for a longer duration; stays cold for a longer duration
  - Use in different locations
  - Commonly used after surgery or in chronic conditions; easier for multiple applications
  - More costly
- Cold-water immersion (cold baths)
  - Larger-sized tanks for immersion of more of the body
  - Used for cooling larger and deeper areas of the body
  - May use whirlpool action to keep colder at skin contact areas
  - Bath temperatures of 50°F–60°F recommended to achieve therapeutic benefit while avoiding too large a drop in core temperature
  - Used more frequently for muscle recovery than injury care
  - Application is for 10–15 minutes
  - Similar concerns as warm whirlpool in terms of open wounds and hygiene
  - Some evidence exists that cold-water immersion reduces delayed-onset muscle soreness, but insufficient evidence that this provides any other benefits
  - Exact physiologic rationale has not been shown. Possible mechanisms include anti-inflammatory effects, vasoconstriction, and decreased nerve transmission, all caused by cooling.
  - Systematic review: Bleakley C et al. *Cochrane Database Syst Rev.* 2012;2:1-136.
- Whole body cryotherapy (Cryosauna)
  - A device (chamber- or barrel-like) that exposes the body to extreme cold (<100°C) for short time periods (2–4 minutes) in order to ease muscle soreness after exercise.
  - These units use liquid nitrogen and refrigerated air to subject a person's skin while wearing minimal clothing (socks, shorts, gloves, and sports bras).
  - The mechanism of action is to drastically reduce the skin temperature and thereby produce a reflex vasoconstriction that will consequently result in posttreatment vasodilation, thereby washing out the components of muscle soreness.
  - Currently, research on the effectiveness of whole body cryotherapy is inconclusive. However, previous studies have revealed no evidence of adverse effects.
  - Whole body cryotherapy is most commonly used after strenuous workouts by elite athletes when muscle soreness and fatigue are present. In addition, certain benefits have been shown in patients having multiple sclerosis.
  - Evidence: systematic review in Costello JT et al. *Cochrane Database Syst Rev.* 2015;9:1-65.
- Vapocooling sprays
  - Chemical spray (ethyl chloride) topically applied on skin
  - Cools rapidly and evaporates quickly
  - Cools only superficially
  - Used whenever quick numbing is desired; trigger point treatment

## RICE

- Use rest, ice, compression, and elevation (RICE), particularly with acute injuries.

- Compression can take the form of:
  - Tightly applied ice bag
  - Ice bag applied over top a wetted ace wrap (to enhance thermal exchange)
  - Use of adhesive expandable tapes
  - Commercially designed device
- Compression works by:
  - Increasing heat exchange by squeezing tissues together and closer to the surface
  - Decreasing tissue spaces where fluid can accumulate
  - Decreasing blood flow so that less warm blood enters into the affected area
  - Increases contact areas of cold application on the skin

## WHEN TO USE HEAT VERSUS COLD

- It is not uncommon for a clinician to be confronted with a dilemma regarding when to use heat or when to use cold.
- Most simplistic guide to follow is, **if pain is the primary symptom, use cold; if stiffness is the chief complaint, use heat.**
- Injuries in the acute inflammatory phase warrant ice application. As swelling on an injury site increases, it becomes more painful and also takes longer to improve. Modalities that increase temperature will result in more swelling. The duration of the acute injury phase can be variable, but certainly not <24 hours after injury.
- Application of ice is effective as an anesthetic. When the patient has pain at rest or has high levels of pain, use some form of cryotherapy.
- Heat can be safely applied whenever the patient is out of the acute inflammatory phase.
- The application of heat before exercise can often make the workout more comfortable. The tissues are more elastic and flexible. Most bodily functions work more effectively at slightly higher temperatures, particularly those relating to athletic activity.
- After completion of a workout, particularly if the workout produces any pain in the injured area, ice is the appropriate treatment. This can reduce any inflammatory effects brought on by the activity.
- Certain chronic conditions may still benefit from cryotherapy. With the presence of a long-term injury, it is not always clear as to which will be the most effective modality. The clinician may have to use trial and error or revert to the aforementioned guidelines.
- When in doubt, apply ice. There is less risk of inappropriate application of ice compared with heat.

## ULTRASOUND

- Therapeutic ultrasound has thermal and nonthermal effects.
- **Thermal effects**
  - Increased tissue temperature
  - Increased cellular activity
  - Increased blood flow
  - Increased tissue extensibility
  - Reduced muscle spasm
  - Reduced pain
- **Nonthermal effects**
  - Acoustic streaming—movement of fluids along cell membranes
  - Cavitation—formation of gas-filled bubbles
  - Both these nonthermal effects are thought to facilitate tissue repair.
- Ultrasound is most effective therapy when there is a relatively small treatment area. Target area is 2–3 times the size of the device's sound head, called the *effective radiating area (ERA)*. Conditions that require treatment over a large area will result in longer treatment durations that may become extremely time-consuming for the clinician.
- Sound head must be kept moving (2 inches/second recommended) during the treatment. Accumulation of the thermal energy can become uncomfortable.
- Amount of ultrasound energy delivered is the dosage. This varies based on the desired treatment effect. As the dosage increases, thermal effects increase and become the limiting factor owing to the discomfort caused. Therefore, the clinician must select the amount of energy on the basis of treatment parameters (Table 44.1).
- Therapeutic ultrasound is often thought of as a deep heating modality, but it can also be used for heating superficial structures and can have nonthermal effects.
- Indications for ultrasound:
  - Conditions that benefit from increased heating include tendinitis, myositis, arthritis, sprains, strains, etc.
  - Relief of pain and muscle spasm
  - Acute conditions (using nonthermal settings)
- Contraindications for ultrasound: ischemic areas; deep vein thrombosis; anesthetic areas; active infections; tumors; external fixation devices; injury to eyes, heart, skull, genitals; over the trunk during pregnancy or menstruation; over stress fractures or osteoporotic areas.

**TABLE 44.1 ULTRASOUND TREATMENT PARAMETERS**

| | |
|---|---|
| **Frequency** | 1 MHz—penetrates to deeper tissues; up to 1 inch<br>3 MHz—affects superficial structures |
| **Intensity** | The higher the intensity, the greater the thermal effects<br>When goal is heating, select the highest comfortable intensity |
| **Duty Cycle** | Regulates the "on" time of the machine<br>The higher the percentage (up to 100%), the greater the thermal effects |
| **Duration** | Longer durations for larger treatment areas |

### Phonophoresis

- Phonophoresis involves the use of ultrasound energy to assist the diffusion of medication through the skin and into the target tissues.
- Corticosteroids are used to reduce inflammation; salicylates are used to relieve pain.
- Clear gels are better at conducting media than thick, white creams.
- Standard parameters for phonophoresis have not been established; use general ultrasound protocols.
- There is little understanding of how much medicine gets delivered or is required to have an effective outcome.
- Factors affecting how much medicine reaches tissues include:
  - Skin's water content
  - Patient's age
  - Skin composition
  - Skin thickness
  - Skin vascularity
- Phonophoresis may be attempting to work at crossed purposes. The use of pulsed ultrasound is to promote the body's progress through the inflammatory phase to the proliferation phase. Some of the most common medicines to drive through the skin are anti-inflammatories negating the effort to move through the

inflammatory phase. In addition, only high-intensity, continuous US has the energy to drive these medicines through the skin. This method of ultrasound is not reported to have proinflammatory properties.

### Ultrasound Bone-Growth Stimulators

- Ultrasound energy used to facilitate fracture healing
- Low-intensity, pulsed ultrasound used
- Devices made specifically for this purpose
- Application is 20 minutes/day
- Ultrasound energy thought to apply low-level mechanical force to reduce fracture healing time
- Few reports of adverse effects
  - Evidence is growing to show the benefits of low-intensity pulsed ultrasound in decreasing the healing time in fractures that are normally slow to achieve clinical union.
  - Evidence: Hannemann PF et al. *Arch Orthop Trauma Surg.* 2014;134:1093-1106. Ebrahim S et al. *Can J Surg.* 2014; 57:E105-118.

## ELECTRICAL STIMULATION

- Certain tissues of the body with higher water content, such as nerves, muscles, and cell membranes, are directly excitable by electrical current.
- Structures such as bones, cartilage, tendons, and ligaments may be affected by the electric fields caused by the current.
- Electrical muscle stimulation (EMS) is used for:
  - Pain reduction
  - Swelling reduction
  - Muscle spasm reduction
  - Facilitating muscle contractions
  - Minimizing muscle atrophy
  - Muscle strengthening
  - Facilitating fracture healing
  - Facilitating inflammation reduction
- In **direct current (DC)** electrical stimulation, one electrode is positive and the other is negative.
- **Alternating current (AC)** electrical stimulation, the more common application, can be either *monopolar*, with electrodes of unequal size (one designated "active," the other "dispersive"), or *bipolar*, wherein the electrode polarity changes many times per second and both electrodes are of equal size.
- EMS has been shown to stimulate sensory, motor, and pain nerve fibers given the appropriate stimulation parameters.

### Transcutaneous Electrical Nerve Stimulation

- Transcutaneous electrical nerve stimulation (TENS) is the most common form of EMS used for pain reduction.
- **High-rate (sensory) TENS**
  - Pulse frequency: 60–120 Hz
  - Phase duration: short (<150 µsec)
  - Intensity: highest tolerable without eliciting muscle twitch
  - Treatment goal: pain reduction through stimulation of large diameter (A-beta) nerve fibers; quicker-acting, shorter-duration pain relief
- **Low-rate (motor) TENS**
  - Pulse frequency: 2–10 Hz
  - Phase duration: long (200–300 µsec)
  - Intensity: highest tolerable muscle twitch
  - Treatment goal: pain reduction through stimulation of smaller diameter (A-delta) nerve fibers; slower acting, longer duration pain relief
- **Noxious TENS**
  - Pulse frequency: high or low
  - Phase duration: very long (>300 msec)
  - Intensity: painful; highest tolerable
  - Treatment goal: pain reduction through stimulation of smallest nerve fibers (C)
- Small, portable TENS units with current generated by batteries instead of wall outlets are used to help patients achieve and sustain pain relief for longer periods of time.

### Interferential Current

- Interferential current is another variation of EMS used for pain relief.
- Main advantage is deeper penetration
- Uses four electrodes (two pairs of slightly different medium frequencies)
- Arrange electrode pairs diagonally so that the currents interfere
- Resultant current is what determines treatment.
- Can be high- or low-rate TENS
- Configure electrodes so that effect is felt over desired area

### High-Voltage Stimulators

- High-voltage stimulators are also known as *high-volt galvanic stimulators.*
- Primarily used for pain modulation
- Use monophasic current of ≥150 volts.
- Higher voltage results in deeper penetration.
- Average current is low, so safe for patients.
- Phase duration is too short to mimic uncomfortable effects of DC.
- One smaller electrode is "active"; the other, larger electrode is "dispersive."
- Electrode polarity is interchangeable.

### Neuromuscular Electric Stimulation (NMES)

- Neuromuscular electric stimulation (NMES) is used for muscle activation and strengthening.
- NMES activates muscles by stimulating alpha motor neurons that often become inhibited with joint injury pain and swelling.
- Muscle activation can be thought of as lower-level strengthening.
- NMES current needs long phase duration to recruit as many motor units as possible.
- Current frequency of 35–50 pulses/second is needed to achieve tetany.
- The stronger the muscle contraction, the more rest incorporated into treatment cycle.
- Larger fast-twitch fibers are recruited first and more easily fatigued.
- By convention, use 5:1 rest-to-work time ratio.
- NMES for strengthening is more apt to be uncomfortable.
- Better strengthening effect if voluntary muscle contraction is superimposed over NMES stimulation.

### Iontophoresis

- Iontophoresis uses electric current used to move ions across skin barrier for reduction of pain and inflammation.
- It involves a DC generator with monopolar setup: one large dispersive electrode, one small active electrode.
- Clinician selects electrode's polarity depending on polarity of medication.
- Polarity of commonly selected medications:
  - Salicylate (–)
  - Hydrocortisone (+)
  - Lidocaine (–)
  - Dexamethasone (–)

- Mixture of lidocaine–dexamethasone most commonly used
- Lidocaine acts to reduce skin irritation at treatment site and can also reduce the amount of dexamethasone pushed through the skin.
- Now manufacturers make buffered electrodes that eliminate the need for lidocaine.
- Maximum current for each treatment should be 5 μA; maximum dosage 80 μA/minute
- For each treatment, set maximum dosage first, then highest tolerable current; treatment time will result from that.
- No standard number for iontophoresis treatments has been determined.

### Stimulation of Denervated Muscle

- In this treatment, denervated muscle from injury or disease is stimulated by DC.
- Current is directly applied to muscles to reduce atrophy.
- Alpha motor neurons can regenerate, as opposed to injured upper motor neurons in the spinal cord, which cannot regenerate.
- Because muscle cells are being stimulated directly and their capacitance is large, current phase duration must be very long (too long for AC).
- Stimulation is uncomfortable to patient because small diameter C nerve fibers that carry noxious pain are stimulated.
- Efficacy of DC stimulation of denervated muscle has not been proven.

### Microcurrent

- Microcurrent treatments use very low current (μA) to modulate pain, reduce swelling, and heal skin lesions.
- The current used is too low to depolarize nerve.
- Theoretically, subthreshold stimulation enhances adenosine triphosphate (ATP) production, which then affects cell function.
- Very little research exists to support claims of benefits of microcurrent.

### Electrical Bone-Growth Stimulators

- Electrical bone-growth stimulators attempt to introduce electrical field to aid fracture healing.
- They use an electrical field similar to that produced by normal body activities, thus stimulating bone growth in accordance with Wolff's law.
- Devices will have external electrodes placed transcutaneously using AC or implanted electrodes using DC.
- Effectiveness of electrical bone-growth stimulators has not been shown conclusively.

### Contraindications for EMS

- Heart/respiratory conditions: current through the chest and neck may disrupt normal functioning
- Application over carotid sinus, esophagus, larynx, pharynx, eyes, upper thorax, and temporal regions: all have enhanced nerve sensitivity and may be easily intolerable
- Pacemakers: current can interfere with device's function
- Pregnancy and menstruation: avoid current over abdomen, pelvis, or lumbar areas
- Tumors: current can stimulate growth
- Active infections
- Exposed metal implants: results in electrical shock
- Electronic monitoring equipment: interferes with device's appropriate function

## DIATHERMY

- Diathermy uses high-frequency electromagnetic waves to heat tissues; also known as *shortwave diathermy*.
- This treatment is chosen for its ability to penetrate deeper and over a larger area than other heating modalities.

### Methods of Application

- Capacitance technique
  - Body part placed between two electrodes
  - Resultant electric field cause structures in body that have a negative and positive pole (dipoles) to rotate
  - Dipole movement causes heat in the tissues
  - Muscle tissue has more dipoles and thus greater capacitance and requires more current to achieve desired effect.
  - Fatty tissue and skin tissue have fewer dipoles and so have greatest heating
- Inductance technique
  - Electromagnetic field generated by passing current through coiled wire
  - Coiled wire wrapped around patient or inside a drum
  - Patient not part of electrical circuit
  - Tissues such as blood vessels and muscle, which are better conductors, see more increase in temperature
  - Adipose and skin see less increase in temperature.

### Indications

- Same as for any other heating modalities
- Has larger effective treatment area and longer heat retention than other deep-heating modality and ultrasound

### Contraindications

- Acute inflammatory conditions
- Metal implants or jewelry
- Cardiac pacemakers
- Over the eyes or genitalia
- Pregnancy
- Tumors
- Open wounds
- Infection
- Peripheral vascular disease
- Water collection over the skin
- Areas of sensory loss

## LOW-LEVEL LASER THERAPY

- Low-level laser therapy (LLLT) makes use of light energy (laser) delivered to body's tissues for therapeutic benefit.
- Laser delivers its energy to a small target area.
- Therapeutic use of lasers is tightly controlled in the United States by the Food and Drug Administration (FDA) and approval has been granted only for:
  - Pain resulting from minor neck and shoulder problems
  - Carpal tunnel syndrome
- Medical lasers' classifications (1–4) are based on amount of energy produced and safety risks.
- LLLT devices are class 3b lasers
  - Medium power devices (5–500 mW)
  - Maximum power allowed: 90 mW
  - May be referred to as "cold lasers"
  - Hazard if viewed directly
  - Not normally a fire hazard
  - Cannot heat tissues >36.5°C
- LLLT devices use different gases to produce laser energy; different gases result in different wavelengths; longer wavelengths penetrate deeper into tissues.

- LLLT effects have been shown to occur to 15 mm.
- LLLT has multiple therapeutic uses. Treatment dosage depends on the intended therapy, and is defined by the machine's output power, which is not adjustable, the duty cycle, and the treatment time.
- **Precautions with the use of LLLT:**
  - Pain may occur the day following treatment, which is thought to be an activation of the tissue-healing mechanism.
  - Avoid use within 6 months of radiation therapy.
  - Patient dizziness may occur and with continued occurrence treatment must cease.
  - Avoid treatment to trunk during pregnancy and to unfused epiphyseal plates.
  - Avoid use with small children.
- Contraindications for LLLT include use over cancerous areas, over the eyes, and in areas of bleeding.

## MAGNETS

- It may be useful to understand what lies behind magnet therapy, although it is not an obvious therapeutic modality.
- Magnetic fields are commonly used for therapeutic benefit; magnetic fields used for therapy result from an AC source.
- Therapeutic magnets are low-power static magnets worn on the body. (Static magnets have a positive and negative pole.)
- Proponents state that magnets can be used to restore the body's natural magnetic field alignment, which becomes abnormal with injury or disease.
- The power of therapeutic magnets ranges from 500–1000 Gauss (G).
- Magnets over 1000 G are regulated by the FDA. (The power of an average refrigerator magnet is 4 G.)
- The power of magnets used for magnetic resonance imaging (MRI) is 15,000 G.
- The World Health Organization (WHO) has deemed magnets up to 20,000 G safe.
- There are two techniques used for the application of therapeutic magnets:
  - Unipolar magnet: one pole (usually negative) touches skin
  - Bipolar magnets: both poles touch skin and are arrayed in patterns to make optimal use of polar effects
- Little or no scientific evidence exists to demonstrate the therapeutic value of these low-power magnets. Reports of benefits are all anecdotal.
- However, evidence does exist demonstrating the value of compression garments in enhancing recovery from muscle damage and pain relief. The value of "magnets" may come from the compression of the garment.
- Evidence (systematic review): Hill J et al. *Br J Sports Med.* 2014;48:1340-1346. Marquez-Jimenez D et al. *Physiol Behavior.* 2016;153:133-148.

## DRY NEEDLING

- Therapeutic technique of introducing thin filiform needles through the skin to lessen pain and reduce muscle tension
- Most commonly the needles try to penetrate into muscle and connective tissue at areas of increased tension similar to trigger points. The technique is to ensure release of tension from these taut bands of skeletal muscles.
- Also called *sports acupuncture*, *myofascial acupuncture*, and *tendinomuscular acupuncture*. The technique should only be attempted by clinicians who have undergone specialized training, have practical experience, and understand the state regulations regarding dry needling.
- Contraindications include patients with local skin lesions, local or systemic infections, severe hyperalgesia, metal (nickel and chromium) allergies, abnormal bleeding tendencies, patients with vascular disease or varicose veins, and women who are in the first trimester of pregnancy.
- Adverse effects have been noted in patients in whom the needles have penetrated too deeply and punctured the underlying structures (pneumothorax).
  - Evidence: Amer Phys Ther Assoc. Description of Dry Needling in Clinical Practice: an Educational Resource Paper. Amer Phys Ther Assoc. Alexandria VA; 2013.

## EXTRACORPOREAL SHOCKWAVE THERAPY

- Therapeutic device that uses an electric charge to create a high-energy pulse (shock wave).
- A noninvasive technique whereby a shock wave is transmitted through the skin to the target tissue, providing pain relief and inflammation reduction.
- Exact mechanism of benefit has not been shown. Proposed benefit is that the mechanical disruption produced by the shock wave produces inflammation that initiates the body's normal healing processes.
- Shock waves are produced in two forms: focused and radial. Focused extracorporeal shockwave therapy (ESWT) has the point of highest energy at a small, specific point in the target area. rESWT has the highest energy at the device and then spreads out into the tissue creating a larger area of effect. No evidence exists that one method of ESWT is more effective than the other.
- ESWT has been shown to be safe. The only adverse effect noted is discomfort during the treatment procedure in certain patients. It has been suggested that patients with altered sensation at the treatment site should avoid this procedure.
- An optimum treatment protocol appears to be three treatment sessions at 1-week intervals.
- ESWT has been shown effective with plantar fasciitis, calcific tendinitis of the shoulder, Achilles tendinopathy, and patellar tendinopathy.
- Evidence of effectiveness (all systematic reviews):
  - Schmitz C et al. *Br Med Bull.* 2015;116:115-138.
  - Aqil A et al. *Clin Orthop Rel Res.* 2013;471:3645-3652.
  - Bannuru R et al. *Ann Intern Med.* 2014;160:542-549.
  - Al-Abbad H, Simon JV. *Foot-Ankle Int.* 2013;34:33-41.
  - Mani-Babu S et al. *Am J Sports Med.* 2014;43:752-761.
  - Speed C. *Br J Sports Med.* 2014;48:1538-1542.

## PULSED RADIOFREQUENCY

- Method of pain control using a device that generates a high-frequency alternating current in short high-voltage bursts introduced through a small (22 ga) needle electrode.
- Developed as a less destructive alternative to continuous radiofrequency treatments that attempt to induce necrosis in the target tissue (ablation).
- Used for pain relief in patients with cervical or lumbar facet joint pain, radicular pain, as well as other peripheral neuropathies. The mechanism of action is not clearly understood.
- Few adverse effects to pulsed radiofrequency have been reported.
  - Evidence (review): Byrd D Mackey S. *Curr Pain Headache Rep.* 2008;12:37-41.

## INTERMITTENT PNEUMATIC LEG COMPRESSION

- Device using full-leg sleeves that fill with compressed air to aid in blood flow and recovery from strenuous exercise.
- The garments have multiple compartments that fill with air and then release the pressure similar to a massaging effect.
- Mechanism of benefit attempts to create an external force that limits the space between the tissues to accumulate fluid. Once

the pressure is relieved, the fluid is thought to flow back into the tissues.
- More commonly used by elite athletes after strenuous exercise to aid in reducing muscle soreness and fatigue. Treatment lasts between 15–20 minutes. No adverse effects have been reported.
- Evidence (RCTs):
  - Cochrane DJ et al. *Int J Sports Med.* 2013;34:969-974 (10 male subjects)
  - Sands WA et al. *J Strength Cond Res.* 2015;29:1263-1272 (12 male/12 female athletes)

## SOFT TISSUE MOBILIZATION WITH INSTRUMENTS

- Commonly referred to as the *Graston technique*, *instrument-assisted soft tissue mobilization technique (IASTM)*, and *fascial abrasion technique (FAT)*.
- All techniques use instruments to apply force across the skin to detect and treat soft tissue lesions.
- Originated thousands of years ago as a traditional Chinese medicine therapy of scraping to create a light bruising called *gua sha*.
- In patients with pain and injury, myofascial adhesions are often present. This technique attempts to free such restrictions in addition to increasing local blood flow.
- Proprietary usage of different techniques exists with specific training and equipment.
- The tools (often metal or ceramic) are specifically designed to contour to various locations on the body where treatment is commonly applied.
- To date, the evidence on effectiveness is confined to case series and case studies. Some higher levels of evidence exist but have contradictory findings on effectiveness.
- Few adverse effects have been reported (other than temporary bruising and resultant soreness), although patients with open wounds should avoid these techniques.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


1. Baker KG, Robertson VJ, Duck FA. A review of therapeutic ultrasound: biophysical effects. *Phys Ther*. 2001;81(7):1351-1358.
2. Brosseau L, Casimiro L, Robinson V, et al. Therapeutic ultrasound for treating patellofemoral pain syndrome. *Cochrane Database Syst Rev*. 2001;(4):CD003375.
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5. Casimiro L, Brosseau L, Robinson V, et al. Therapeutic ultrasound for the treatment of rheumatoid arthritis. *Cochrane Database Syst Rev*. 2002;(3):CD003787.
6. Chou R, Huffman LH. Nonpharmacologic therapies for acute and chronic low back pain: a review of the evidence for an American Pain Society/American College of Physicians clinical practice guideline. *Ann Intern Med*. 2007;147(7):492-504.
7. Crawford F, Thomson C. Interventions for treating plantar heel pain. *Cochrane Database Syst Rev*. 2003;(3):CD000416.
8. Denegar CR, Saliba E, Saliba S. *Therapeutic Modalities for Musculoskeletal Injuries*. 2nd ed. Champaign, Ill: Human Kinetics; 2006.
9. French SD, Cameron M, Walker BF, et al. Superficial heat or cold for low back pain. *Cochrane Database Syst Rev*. 2006;(1):CD004750.
10. Khadilkar A, Milne S, Brosseau L, et al. Transcutaneous electrical nerve stimulation (TENS) for chronic low-back pain. *Cochrane Database Syst Rev*. 2005;(3):CD003008.
11. O'Connor D, Marshall S, Massy-Westropp N. Non-surgical treatment (other than steroid injection) for carpal tunnel syndrome. *Cochrane Database Syst Rev*. 2003;(1):CD003219.
12. Robertson VJ, Baker KG. A review of therapeutic ultrasound: effectiveness studies. *Phys Ther*. 2001;81(7):1339-1350.
13. Robinson VA, Brosseau L, Casimiro L, et al. Thermotherapy for treating rheumatoid arthritis. *Cochrane Database Syst Rev*. 2002;(2):CD002826.
14. Starkey C. *Therapeutic Modalities*. 3rd ed. Philadelphia: FA Davis; 2004.

# HEAD INJURIES

# 45

Siatta B. Dunbar • Margot Putukian • Christopher C. Madden

## GENERAL PRINCIPLES

- Head injuries in sports are comparatively mild compared with those in high-velocity motor vehicle accidents (MVAs), yet they remain significant and important injuries for team physicians to evaluate and manage.
- **Concussion** is the most common head injury in sports. Information is evolving regarding pathophysiology, diagnosis, natural history, and treatment of concussion in sports.
- Consider focal, vascular, and associated injuries (e.g., cervical spine, skull fractures, intracranial hemorrhage) when evaluating head-injured athletes.
- Cumulative injury, postconcussive syndrome, and other sequelae of head injury may contribute to significant morbidity.

### Epidemiology

- **Many head injuries go undetected,** especially in younger age groups.
- Athletes often underreport symptoms, contributing to the underreporting of injuries.
  - Athletes may take injuries lightly, viewing them as "part of the game."
  - Athletes may fear being removed from the game if they admit symptoms.
  - Head injuries may be evaluated on the sideline but subsequently not referred for further evaluation, or the athlete may fail to follow-up with the appropriate referral.
- According to the National Electronic Injury Surveillance System—All Injury Program from 2001 to 2009 a total of 2.6 million emergency department visits were due to head injury. Of these, 173,285 visits were by patients under 19 years of age who sustained injuries as a result of sports and recreational activities. Head injuries represent 6% of all sports and recreational injuries.
  - Of the 173,285 injuries, 10.6% were due to ATV accidents, 7.7% from soccer, and 7.2% from American football.
  - Comparatively, MVAs account for 14% of head injuries, falls for 41%, and assaults for 11% (CDC data 2006–1010).
- According to the Annual Survey of Catastrophic Football Injuries (1997–2012) in 2012, there were three cervical cord injuries and five brain injuries.
- The 4th International Conference on Concussion in Sport characterized concussion as a "subset of traumatic brain injury."
- Concussion is the most common head injury occurring in sports, accounting for 8.9% and 5.8% of injuries at the high school and college level, respectively.
- Data from the National Collegiate Athletic Association (NCAA) collected from 1998–1999 to 2003–2004 demonstrated that the incidence of concussion was 5.3/1,000 athlete exposures (AEs) in women's soccer compared to 3.9/1,000 in men's soccer, and 4.7/1,000 AEs in women's basketball compared to 3.2/1000 in men's basketball. Interestingly, these two sports have similar rules across genders (Table 45.1).
- Injury rates are comparable in sports with and without head protection.
  - The highest overall incidence of concussion occurs in wrestling, followed by men's football, men's hockey, women's field hockey, women's soccer, women's lacrosse, men's lacrosse, men's soccer, women's basketball, men's basketball, softball, women's volleyball, and finally, baseball.
- In sports with similar rules (soccer, basketball, baseball/softball), the mechanism of injury between sexes is different, with player contact to surface or equipment being more common in females than in males.
- Data suggest that women endorse more symptoms at baseline; however, evidence is inconclusive that there are balance/postural differences between men and women at baseline or post concussion.
- Exact reasons for gender differences are unclear, but they may include reporting bias, treatment bias, the effect of fluctuating sex hormone levels across the menstrual cycle, and biomechanics differences.
- **Look at epidemiology when considering injury prevention.** Before making rule or equipment changes, consider incidence of injury and how changes may affect the sport.
  - The use of a helmet in women's lacrosse and field hockey may decrease the incidence of facial lacerations, nasal fractures, or dental injuries but may not significantly affect the incidence of concussions. Increased aggressiveness on the part of players owing to added head protection may negatively affect the sport.
  - One study on ice hockey reported that 75% of 246 head injuries involve violence unrelated to on-ice activities (high sticking, deliberate pushing, or fistfights).
  - The proper enforcement of existing rules (avoiding head-to-head hits) is essential.
  - Use of helmets significantly decreases the risk of head injury in bicycling, baseball, skiing, snowboarding, and softball, without negatively affecting the sport.
  - There is no evidence that current helmets or mouth guards can prevent concussion. Helmets can decrease the incidence of skull fractures because of their protective plastic shell, and mouth guards can decrease incidence of tooth injury.

## TYPES OF HEAD INJURY

Head injuries occur across a large spectrum. Classifications such as diffuse versus focal or nonstructural versus structural are not absolute, but they permit the organized discussion of specific pathologies.

### Diffuse Brain Injury

#### Diffuse Axonal Injury (DAI)

- Involves diffuse axonal disruption in the white matter of the brain and brainstem
- Severity of injury determined by clinical course
  - Mild DAI: comatose for 6 to 24 hours; mortality rate is approximately 15%
  - Moderate DAI: comatose for more than 24 hours; often associated with basilar fracture; mortality rate is approximately 25%
  - Severe DAI: prolonged coma, severe disability, or persistent vegetative state common if patient survives; high mortality rate; death often caused by infectious complications, associated intracranial pathology, and other complications of prolonged coma

**TABLE 45.1 NCAA INJURY SURVEILLANCE SYSTEM DATA FOR 1988-1989 THROUGH 2003-2004**

| Head Protection Required | % of All Game Injuries | No Head Protection | % of All Game Injuries |
|---|---|---|---|
| Men's ice hockey* | 7.9 | Women's lacrosse* | 6.3 |
| Women's ice hockey | 18.3 | Wrestling | 3.3 |
| Men's lacrosse* | 5.6 | Women's soccer | 5.3 |
| Football* | 6.0 | Men's soccer | 3.9 |
| Spring football* (practice) | 5.6 | Field hockey | 3.9 |
| Softball | 4.3 | Women's basketball | 4.7 |
| Baseball | 2.5 | Men's basketball | 3.2 |
| Concussion injuries in games and practices per 1,000 athlete exposures | | | |
| Head protection required | | No head protection | |
| Football* | 0.37 | Wrestling | 0.25 |
| Men's ice hockey* | 0.41 | Men's soccer | 0.28 |
| Women's ice hockey | 0.91 | Women's soccer | 0.41 |
| Men's lacrosse* | 0.26 | Women's lacrosse* | 0.25 |
| Spring football* | 0.54 | Field hockey | 0.18 |
| Softball | 0.14 | Women's basketball | 0.22 |
| Baseball | 0.07 | Men's basketball | 0.16 |

*Mouth guard required.
Data from Hootman J, Agel J, Dick R. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42(2):311-319.

**TABLE 45.2 SIGNS AND SYMPTOMS OF CONCUSSION**

| Early (Minutes to Hours) | Late (Days to Weeks) |
|---|---|
| **Cognitive**<br>Confusion<br>Vacant stare<br>Slow to answer questions or follow instructions<br>Easily distracted<br>Inability to focus<br>Feeling "in a fog"<br>Disoriented: unaware of time/date/place<br>Slurred or incoherent speech<br>Memory deficits<br>Repeatedly asks same question (e.g., what happened?)<br>Retrograde amnesia (RGA): Cannot remember events before injury<br>Posttraumatic amnesia (PTA): Cannot remember events after injury<br>Loss of consciousness | Persistent low-grade headache<br>Lightheadedness<br>Poor attention and concentration<br>Memory dysfunction<br>Anomia (cannot think of word one wants to say)<br>Easy fatigability<br>Irritability and frustration<br>Difficulty with focusing vision<br>Photophobia<br>Phonophobia<br>Anxiety and/or depression<br>Sleep disturbance<br>Persistent cognitive deficits<br>Postconcussive syndrome |
| **Somatic**<br>Gross uncoordination: cannot walk straight line<br>Headache<br>Dizziness, disequilibrium or vertigo<br>Visual disturbances (blurry vision, photophobia)<br>Phonophobia<br>Fatigue<br>Nausea and/or vomiting<br>Seizure | |
| **Affective**<br>Emotional lability: may cry for no apparent reason<br>Irritability<br>Nausea and/or vomiting<br>Seizure | |

- Caused by **shear or tensile forces;** often results from falls or motor vehicle accidents
- **Presentation:** All patients present in coma, may exhibit decorticate or decerebrate posturing, severe posttraumatic amnesia, and cognitive deficits after awakening (moderate DAI); severe DAI often includes hypertension and hyperpyrexia (autonomic dysfunction), increased intracranial pressure (ICP), posturing, and herniation syndromes.
- **Treatment:** Supportive during coma; medical or surgical measures as needed for increased ICP and associated injuries

### Cerebral Concussion

- The Fourth International Conference on Concussion in Sport defined concussion as a "subset of traumatic brain injury" and is a "complex pathophysiologic process affecting the brain, induced by traumatic biochemical forces."
- Several common features that incorporate clinical, pathologic, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:
  - May be caused by direct impact to head, or elsewhere on body with "impulsive" force transmitted to head
  - Results in the rapid onset of short-lived impairment of neurologic function that usually resolves spontaneously.
  - May result in neuropathologic changes, but acute clinical symptoms reflect functional rather than structural disturbance
  - Results in a graded set of clinical syndromes that may or may not involve loss of consciousness. The resolution of symptoms typically follows a sequential course.
  - Typically associated with grossly normal structural neuroimaging studies
    - Advanced neuroimaging (e.g., diffuse tensor imagine, functional magnetic resonance imaging, magnetic resonance spectroscopy) may demonstrate abnormalities, although it currently remains primarily a research tool.
- Cerebral concussion is the **most common head injury in athletes;** isolated concussion has a low mortality rate.
- Caused by **acceleration/deceleration** (tensile), **rotational** (shearing), and **impact** (compressive) forces. Coup injuries often result from direct impact, whereas contrecoup injuries occur from accelerational/decelerational forces (e.g., athlete falls and strikes ground with head).
- **Presentation:** The hallmark of concussion is confusion; other signs and symptoms may occur immediately or several minutes later (Table 45.2).

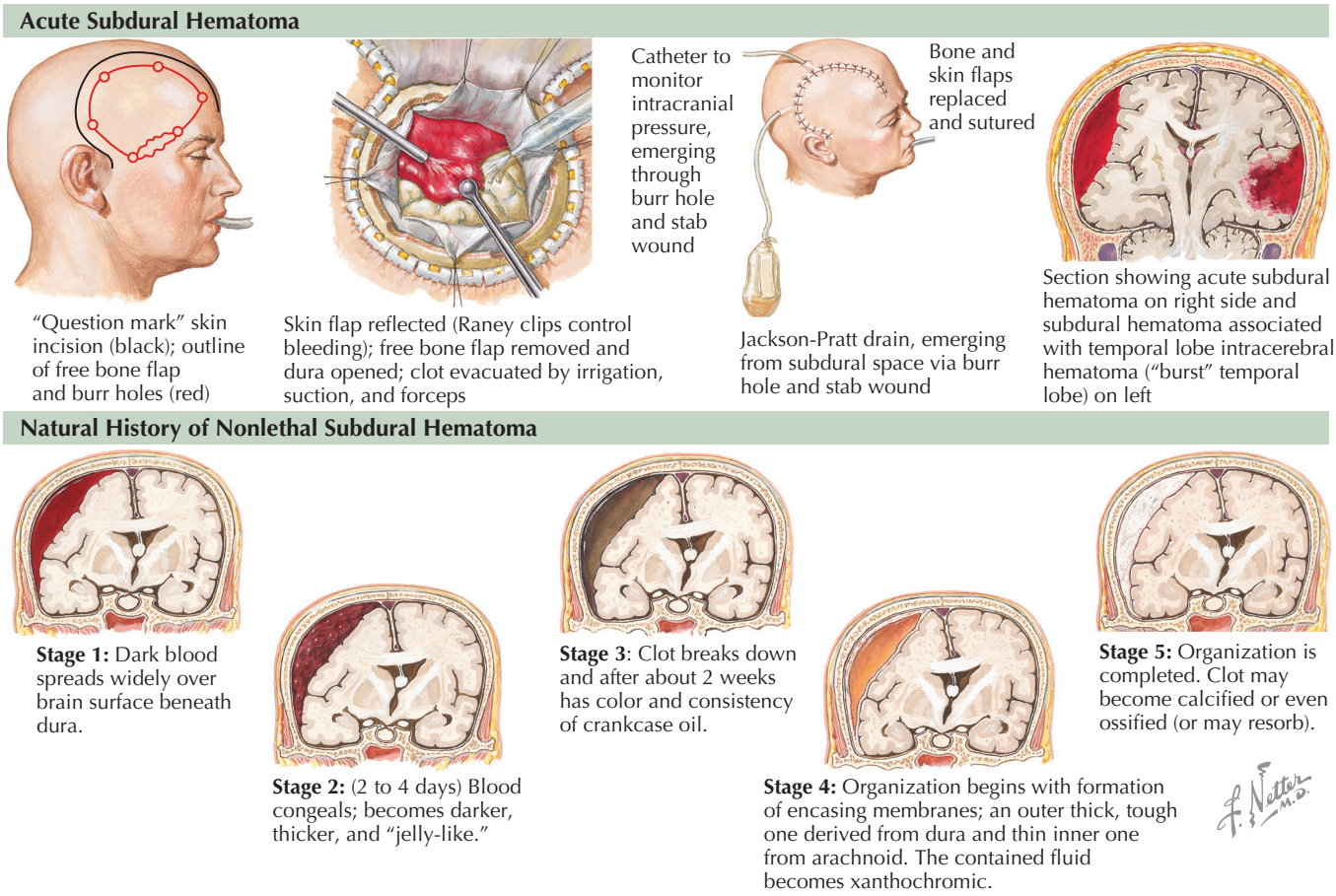



**Figure 45.1.** Subdural hematoma.

- **Treatment:** Rest (physical and cognitive), protection from further injury, and serial follow-up evaluations (see detailed discussion below)

## PATHOPHYSIOLOGY OF CONCUSSION

- Concussion is associated with neurochemical and metabolic changes with changes in glutamate, potassium, lactate, and glucose, as well as changes in cerebral blood flow.
- No current objective neuroanatomic or neurophysiologic measurements can be used practically and reliably to determine if an athlete has a concussion. See the Diagnostic Testing section.
- After concussion, the brain cells may be in state of **injury-induced vulnerability;** a second injury during this time of heightened vulnerability may produce additional deficits.
  - Injury-induced vulnerability is characterized by a **fuel need–fuel delivery mismatch.** The brain's need for glucose increases acutely (hyperglycolysis), and cerebral blood flow and oxidative metabolism are relatively reduced (sometimes called the disruption of metabolic autoregulation).
  - Increased levels of extracellular potassium probably activate adenosine triphosphate (ATP)-dependent sodium–potassium pumps, which increases energy consumption and adds to metabolic stress (e.g., need for glucose).
  - Glutamate (excitatory amino acid) levels increases extracellularly and may contribute to an increased flux of potassium.
  - Increased intracellular calcium may be related to regional reduction of cerebral blood flow.
  - Not known whether normalization of injury-induced neurometabolical and neurochemical abnormalities correlates with resolution of concussive signs and symptoms, but timeline of changes correlates with changes in neuropsychologic function

## Focal Brain Injury

### Subdural Hematoma

- Low-pressure venous bleed into potential space (subdural) between the arachnoid and dura mater; classified by time to clinical presentation (see Fig. 45.1).
  - **Acute:** within 24 hours; often associated with other intracranial pathology (e.g., contusion, axonal injury)
  - **Subacute:** 24 hours to 2 weeks
  - **Chronic:** 2 weeks or more
- Leading cause of death related to head injury; overall mortality rate of 35% to 50%; loss of consciousness implies poor prognosis
- Elderly and alcoholics at greatest risk because of increased space between brain (atrophy) and dura
- Caused by brain movement within the skull: acceleration–deceleration, rotational, shearing injuries
- **Presentation:** Decreased/altered level of consciousness, lucid interval followed by declining mental status and headache; patients may have pupil inequality, motor deficit (e.g., unilateral weakness or paralysis), or other indicators of brain swelling.
- **Treatment:** Usually prompt surgical evacuation; patient may be observed if prognosis is poor (e.g., prolonged loss of consciousness) or if elderly and asymptomatic (see Fig. 45.1).

### Epidural Hematoma

- High-pressure arterial bleed between inner table of skull and dura mater (epidural space); middle or other meningeal arteries often disrupted; 80% associated with a skull fracture in the temporoparietal region (fracture less common in children); occasionally caused by tear of underlying dural sinus (most common in posterior fossa) (Fig. 45.2)
- Other associated intracranial pathology common (e.g., subdural bleed, contusion)
- Mortality rate is low if diagnosed acutely; comatose state is associated with the highest mortality rate (<20%); mortality rate higher with associated injuries.
- Caused by a direct blow to head
- **Presentation:** Decreased level of consciousness followed by lucid interval, deteriorating mental status with eventual loss of consciousness, headache, confusion, sleepiness, nausea, vomiting. Only one-third present classically with loss of consciousness followed by lucid interval and focal deficits.
- **Late signs:** Ipsilateral dilated pupil, contralateral muscle weakness, coma
- **Lucid interval may last for several hours and lead to false reassurance and a missed diagnosis.**
- **Treatment:** Craniotomy/evacuation of hematoma

### Intracerebral Hemorrhage/Hematoma

- Bleeding from small-caliber arterioles within the brain parenchyma; frontal and temporal lobes are affected most often; may be accompanied by **brain laceration** (Fig. 45.3).
- Mortality rate low if patient is conscious before intervention; may approach 45% in unconscious patients
- Caused by tensile or shearing forces that stretch the brain (coup or contrecoup mechanisms of injury)
- **Presentation** varies with the size and location of lesions, as well as any associated pathology (e.g., contusion, postinjury edema); loss of consciousness (<50%), headache, confusion, nausea, vomiting, focal deficits (affected areas) (see Fig. 45.3). Symptoms may develop over hours or days
- **Treatment:** Many require emergent intervention to lower ICP and/or stop bleeding; depends on severity of clinical presentation, bleed, and associated pathology

### Subarachnoid Hemorrhage (SAH)

- Bleeding between the arachnoid and pia mater (subarachnoid space) into the cerebrospinal fluid (CSF); may be **traumatic** or **spontaneous**
- Traumatic SAH results in small tears of subarachnoid vessels; spontaneous SAH (and sometimes traumatic SAH) is often associated with intracranial aneurysms and arteriovenous malformations, and sometimes with hypertension and arteriosclerosis (Fig. 45.4).
- May be the most common abnormality after head injury (all causes); isolated traumatic SAH has low mortality rate; associated pathology (e.g., contusion, skull fracture) leads to less favorable outcome
- Caused by tensile or shearing forces
- **Presentation** depends on associated pathology and whether SAH is traumatic or spontaneous. Symptoms include headache (often described as the "worst ever"), photophobia, nausea, vomiting, dizziness, confusion, neck stiffness, and focal deficits (affected areas). Injury often complicated by posttraumatic cerebral vasospasm from 2 days to 2 weeks after acute bleed; causes neurologic deterioration and may be mistaken for second bleed.
- Must be differentiated from a "bad" migraine or meningitis. Lumbar puncture done only after funduscopic exam (to rule out papilledema) and CT scan. Xanthochromia of the CSF may be most specific finding with SAH.
- **Treatment** varies with individual case and pathology; may involve surgical intervention (e.g., clipping of aneurysm), medical management (e.g., calcium channel blocker for vasospasm), and conservative measures.

## Other Injuries

### Scalp Laceration

- The scalp has many layers and a generous blood supply. Large lacerations or avulsions may be a significant source of bleeding.
- All scalp lacerations should be examined for "step-off" deformities that indicate an underlying depressed skull fracture.
- Hemostasis may be obtained with direct pressure (if no skull fracture is present) followed by quick, careful closure of the wound, usually in single layer. Wounds should be debrided and irrigated well prior to repair.

### Skull Fracture

- Uncommon in athletics, but must always be considered
- Classified as linear or depressed (open or closed)
- Caused by direct impact; usually the force of impact is large enough to cause underlying brain injury
- Presentation varies with the type of fracture and associated brain injury.
- **Nondepressed linear fractures** may cause only localized pain and swelling; basilar fractures (linear) often occur in petrous portion of temporal bone, and may present with

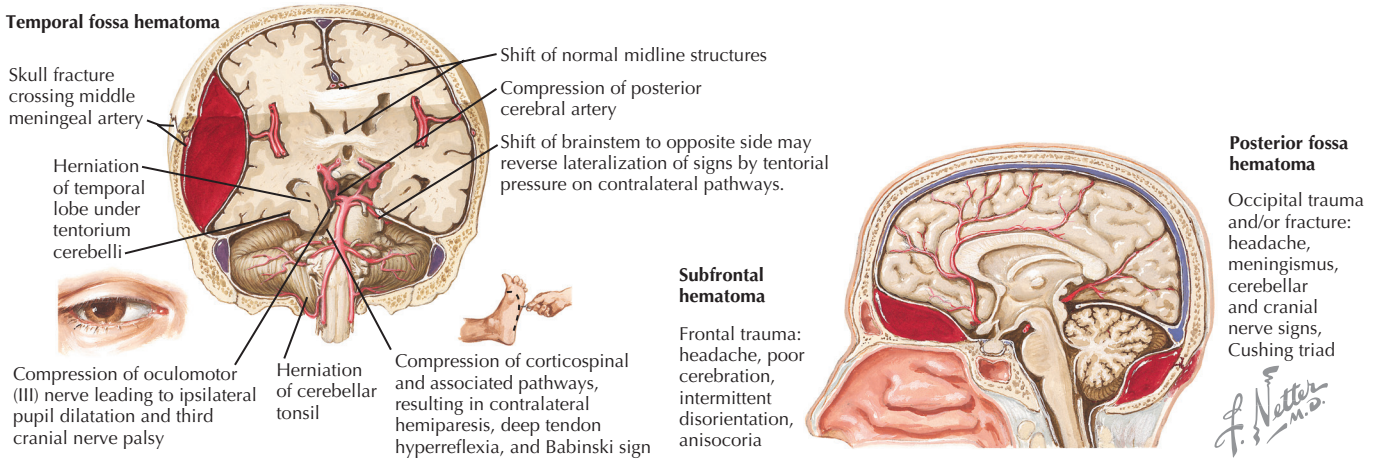



**Figure 45.2.** Epidural hematoma.

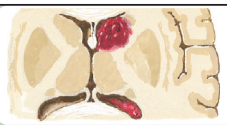
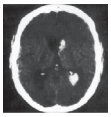
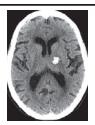
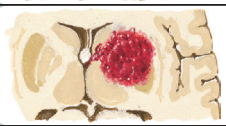
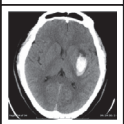
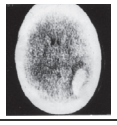
| Pathology | CT scan | Pupils | Eye movements | Motor and sensory deficits | Other |
|---|---|---|---|---|---|
| Caudate nucleus (blood in ventricle) |  |  | Sometimes ipsilaterally constricted | Conjugate deviation to side of lesion; slight ptosis | Contralateral hemiparesis, often transient | Headache, confusion |
| Putamen (small hemorrhage) | |  | Normal | Conjugate deviation to side of lesion | Contralateral hemiparesis and hemisensory loss | Aphasia (if lesion on left side) |
| Putamen (large hemorrhage) |  |  | In presence of herniation, pupil dilated on side of lesion | Conjugate deviation to side of lesion | Contralateral hemiparesis and hemisensory loss | Decreased consciousness |
| Thalamus | | | Constricted, poorly reactive to light bilaterally | Both lids retracted; eyes positioned downward and medially; cannot look upward | Slight contralateral hemiparesis, but greater hemi-sensory loss | Aphasia (if lesion on left side) |
| Occipital lobar white matter | |  | Normal | Normal | Mild, transient hemiparesis | Contralateral hemianopsia |
| Pons | | | Constricted, reactive to light | No horizontal movements; vertical movements preserved | Quadriplegia | Coma |
| Cerebellum | | | Slight constriction on side of lesion | Slight deviation to opposite side; movements toward side of lesion impaired, or sixth cranial nerve palsy | Ipsilateral limb ataxia; no hemiparesis | Gait ataxia, vomiting |

**Figure 45.3.** Intracerebral hemorrhage: clinical manifestations related to site.

hemotympanum, otorrhea, or rhinorrhea (CSF leak), periorbital ecchymosis (raccoon eye), or retroauricular ecchymosis (Battle sign) (Fig. 45.5).

- **Depressed fractures** are noted by palpating "step-off" beneath skull laceration (considered open if scalp is disrupted) (see Fig. 45.5). Some are associated with loss of consciousness, nausea, vomiting, and other neurologic deficits, depending on the extent of the underlying brain tissue injury.
- Open skull fractures have increased risk of infection and seizures.
- CT scan with bone window is more accurate than plain skull x-rays, and provides information about depressed skull fragments and associated intracranial pathology.
- Neurosurgical consultation is advised with all depressed skull fractures. Treatment is individualized and based on the specific location and pathology. Open depressed skull fractures often require prophylaxis for posttraumatic seizures and infection.

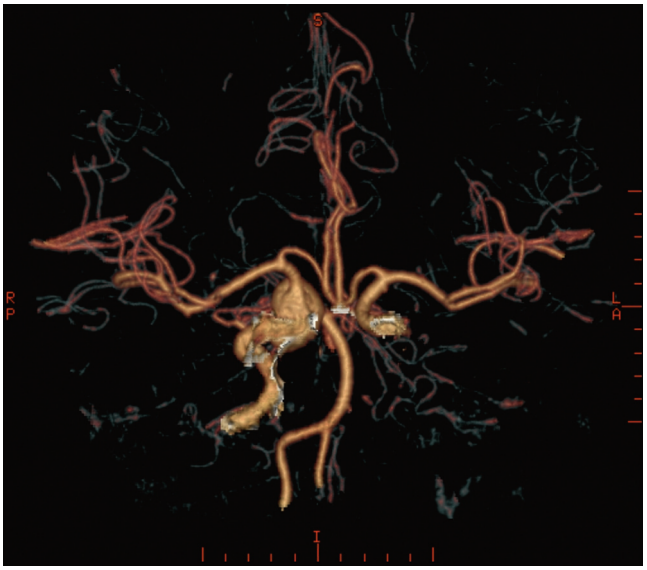
## EVALUATION

Evaluation and management of head-injured athletes must be complete, rigorous, and performed by trained personnel. Highly organized approach must be used.

### Early Evaluation of Head Injuries

- On the field and sideline
- **A**irway, **b**reathing, **c**irculation, **d**isability, **e**xposure (ABCDE)
- **Glasgow Coma Scale; evaluates best eye, verbal and motor response. Useful for predicting prognosis in severe head injury**
- **If athlete is conscious** and has no neck pain and a normal neurologic evaluation, remove to sideline for further evaluation and observation.
- **If athlete is unconscious** or confused or has a neurologic abnormality, skull fracture, or neck pain, protect the airway, immobilize cervical spine, and transfer to a hospital for further evaluation (e.g., cervical spine films, other imaging).
- **Evaluation of associated injuries**
  - Unstable cervical spine injury should be assumed until proven otherwise; ask about neck pain.
  - Maintain a high suspicion for vascular or focal injuries (change in level of consciousness, focal deficits, and other neurologic symptoms).
  - Assess for skull fracture (see the Skull Fracture section).
- **Thorough history at time of injury and focused neurologic exam with attention to mental status and cognitive functioning.**
  - Determine level of consciousness, and memory of event before (retrograde amnesia) and after (posttraumatic amnesia) injury.
  - Ask athlete to recount the specifics of events (e.g., game score, special plays, teammates), events before game, previous game score. Teammates and coaching staff may be useful in validating information.
  - Neurologic assessment: early cognitive, somatic, and affective signs and symptoms (see Table 45.2)
  - Orientation: time, place, date
  - Short-term memory, long-term memory, and concentration assessment
    - Examples include asking the athlete about teammates, coach, specific plays, home phone numbers, what and

CT Angio source image showing an aneurysm.



CTA 3-D reconstruction showing detailed anatomy of the aneurysm.

**Figure 45.4.** Subarachnoid hemorrhage.

who they ate with prior to the event, and what classes they attend on specific days.
    - Confusion may be quickly assessed by using questions proposed by Maddocks and colleagues: "Where are we? Which team are we playing? Who is your opponent? Which half is it? How far into the quarter is it? Which side scored last? Which team did we play last week? Did we win last week?"
    - Ask athlete to remember five items, count backward (digit spans, e.g., 1-4-2, 6-9-3-1 and so on up to six digits or more), name the months in reverse, give the number of dimes in a dollar, other simple tasks.
- Assess upper and lower extremity motor and sensory function, deep tendon reflexes.
- Assess for cranial nerve deficits.
- Assess cerebellar function: finger to nose, heel to shin, Romberg test, and tandem gait with eyes opened and closed.
    - Assess balance; may use computerized platform or clinical assessment. The modified Balance Error Scoring System (BESS) can be utilized.
    - Assess horizontal and vertical gaze as well as convergence.
- **History of prior concussions and risk factors**
    - Details of previous injuries, including alterations in level of consciousness, associated injuries, time lost from participation, and date of most recent concussion
    - Pay particular attention to repeat concussions with lesser impact forces and/or increasing duration of symptoms.
    - Those with a history of at least three concussions had a 3-fold greater chance of sustaining another concussion
    - Assessment of other modifiers: female gender, history of depression, ADD/ADHD or migraines, age (<18 years of age), intoxication, use of anticoagulants, hemophilia, inadequate postinjury supervision
- **Close observation and periodic reevaluation** are highly important. Signs and symptoms worsen with time.
    - Athlete's equipment (e.g., helmet, stick, and glove) as applicable should be held to prevent re-entry into game, and the coach should be made aware that the athlete is not available to participate.
    - Athletes may need to be serially evaluated after an injury, and a plan should be arranged with the athlete, parent, and/or other teammates/roommates such that the athlete can be watched for signs of deterioration and transported to an appropriate facility if need be.
    - If neuropsychologic testing (NPT) is available, consider performing postinjury NPT at least 24 to 48 hours after injury (as long as athlete is asymptomatic).

## Standardized Assessment Tools

- Developed to establish valid standardized, systematic sideline evaluation for the immediate assessment of concussion in athletes
- **Not meant to replace individual assessment** or more comprehensive formal clinical NPT; **do not diagnose concussion**

## Standardized Assessment of Concussion (SAC)

- Baseline testing recommended for comparison with postconcussion scores
- If appropriately used in addition to individualized assessment, may provide a good starting point for less experienced health professionals.
- Objectively assesses orientation, immediate memory, concentration, and delayed recall, but does not assess more complex neurocognitive functions
- **Advantages:** Practical and portable with some validity, takes about five minutes to administer; may be administered by the athletic trainer; three equivalent forms minimize practice effects.
- **Disadvantages:** Potential for inappropriate use to diagnose concussion and make return-to-play (RTP) decisions; sensitivity questioned by some as screening tool for concussion.

## Sport Concussion Assessment Tool (SCAT3)

- A standardized tool developed for use with both physician assessment of sports concussion and patient education; recommended for use by the Zurich 4th International Conference
- Combined multiple existing assessment tools into standardized tool, including SAC, Glasgow Coma Scale, symptom checklist, Maddock's, and modified Balance Error Scoring System (BESS)
- Similar uses, advantages, and disadvantages as SAC, but with additional memory questions, symptom diary, RTP guidelines, and patient education material

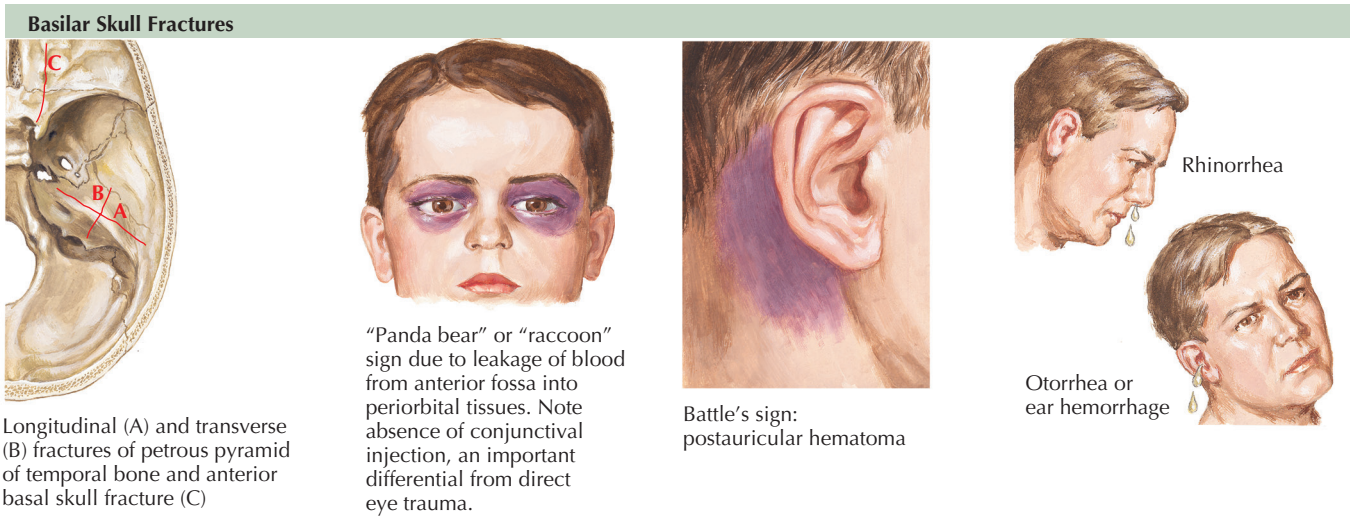



**Figure 45.5.** Skull fracture.

- When baseline assessments are available and can be compared with postinjury assessments, a drop in score of 3.5 points on the SCAT2 (same components as SCAT3) was demonstrated to have a sensitivity and specificity of 96% and 81%, respectively. When a baseline assessment is not available, sensitivity and specificity were 91% and 83% when a cutoff value was used.

### Transfer for Further Evaluation

- Suspected cervical spine injury
- Deteriorating mental status, deteriorating or persistent neurologic deficits (e.g., focal signs), unusual behavior or noticeable irritability, becoming lethargic and can't be awoken, worsening headache, persistent nausea, and vomiting
- Have difficulty with balance, weakness or numbness in arms or legs, and/or develop seizures
- High-risk condition (e.g., hemophilia, intoxication, anticoagulants)

### Delayed Evaluation

- Serial sideline, postgame, training room, or office
- All athletes with head injury should be reassessed serially as needed. Injury history is again essential. Family members often help validate history. Athletic trainer's notes and consultation may be useful.
- Same evaluation as on-field and initial sideline assessments
- Consideration of repeat or additional neuropsychological testing

## INJURY SEVERITY AND MANAGEMENT OF CONCUSSION

- Numerous concussion grading systems and RTP guidelines are available. **Many are empirical and lack scientific basis.**
- Early guidelines used loss of consciousness (LOC) as an indicator of injury severity, which has subsequently been shown not to be true.
- Newer guidelines, such as those published by the National Athletic Trainers Association, the American Medical Society for Sports Medicine, the Zurich 4th International Conference on Concussion in Sport, the American Academy of Neurology, as well as Team Physician Concussion Consensus Statement (TPCC), likely represent the most comprehensive concussion guidelines available.
- Diagnosis is based a range of domains: clinical symptoms (e.g., headache), signs (e.g., loss of consciousness, amnesia), behavior (e.g., irritability) and/or balance changes and cognition (e.g., slowed reaction times).
- The Team TPCC guidelines encourage determining the severity of a concussion only after all symptoms resolve and cognitive and neurologic exams normalize.
  - Severity of injury is based on nature, burden, and duration of symptoms.
- Zurich 4th International Consensus Statement substantiates an individualized RTP with progressive step-wise exercise program.
  - For RTP, emphasis is appropriately placed on individualized assessment, not on a rigid timeline.

### Return-to-Play Guidelines

- Newer guidelines all consistent; no same day return to play for an athlete diagnosed with concussion
- All agree that **symptomatic athletes should not be allowed to return to play.**
- The most recent Zurich Guidelines do not recommend a same day RTP under any circumstances for any athlete diagnosed with concussion.
- Conservative considerations with emphasis on a period of prolonged asymptomatic rest should be considered in athletes with a history of repeat concussions; in **young athletes;** and in athletes with a history of prolonged signs or symptoms following concussion, current or previous injury associated with disproportionately lesser force, and other risk factors.
- If delayed RTP, formal NPT and balance testing should be considered before RTP if available (see later discussion). If unavailable, serial evaluations with individualized progression of activity and conservative advancement are important.
- In some situations, if an athlete has symptoms after an acute period of time, physical activity may be considered as a component of treatment that may hasten recovery.
- The **timing** and **speed** of progression is controversial. Consider symptom-free interval prior to initiating and advancing progression if individual risk factors exist. Most important modifying factors include gender, age, prior history of concussion and temporal relationship, pre-existing headache, family history of headache in the adolescent athlete, ADD/ADHD, depression, or other comorbidities. Progression should occur at a slower rate for young athletes than in adults.
- No long-term data adequately assess RTP decisions.

### Additional Further Diagnostic Testing

- A computed tomography (CT) scan should be the first tool utilized if there is a concern about intracranial bleeding or fracture; better at detecting blood, fracture; as good as magnetic resonance imaging (MRI) at identifying surgical lesions.
- Electroencephalogram (EEG), MRI, and CT scan may present normally despite significant clinical symptoms and abnormalities in cognitive function.
- Positron emission tomography (PET), diffusion tensor imaging (DTI), and functional MRI scans may play roles; correlate with pathophysiologic data. Clinical applicability is not practical in most settings at this time; mainly research tools.
- Neuropsychological testing (see later discussion)
- Laboratory evaluation of athletes with significant head injuries should be individualized. Consider complete blood count, electrolytes, serum glucose, urinalysis, coagulation studies, toxicological and ethanol screens, and blood type and crossmatch.
- Cervical spine x-rays should be considered in all athletes with significant head injuries. Skull films may help localize and determine severity of depression of underlying skull fractures.

## NEUROPSYCHOLOGICAL TESTING (NPT)

- **NPT provides assessment and quantification of brain function** by examining brain-behavior relationships. Neuropsychologists are likely best trained to interpret NPT results.
- Tests measure a broad range of cognitive functions: speed of information processing, memory, attention and concentration, reaction time, scanning and visual tracking ability, and problem-solving abilities.
- Preliminary studies used in athletics utilizing paper and pencil testing demonstrate NPT as a useful tool in the assessment of concussion.
- Newer and more portable computerized test batteries are available, including: Immediate Postconcussion Assessment and Cognitive testing (ImPACT), CogState, Automated Neuropsychological Assessment Metrics (ANAM), and Headminder. These computerized tests are shorter and easier to perform than the standard paper and pencil tests. Controversy surrounds the optimal protocol of tests and most appropriate time for usage.
- Several studies using computerized NPT have shown the "value added" of NPT in addition to symptoms in demonstrating cognitive deficits in athletes after concussion.
- Recent studies have questioned the sensitivity and specificity of various computerized formats.
- Remains as "one piece of the puzzle" when managing concussion
- May be useful in assessment and recovery phases of head injury. Most clinicians advocate usage after acute injury and symptoms have resolved, compared with preinjury baseline assessment.
- NPT is currently used in the National Football League, the National Hockey League, Major League Soccer, US Soccer, and US Lacrosse, as well as at various college and high school programs in the assessment of concussion.
- NPT may detect acute and chronic head injury; it is more sensitive in assessing cognitive function than classic medical testing (neurologic exams using MRI, CT, and/or EEG).
- NPT provides additional useful information in the assessment of concussion and may supplement, but not replace, comprehensive individualized assessments. NPT is "only one tool in the toolbox."
- NPT may become more widely available for sideline use in the future, especially with the development of more sophisticated programs for laptop and hand-held computers.
  - Essential for consideration is how NPT is conducted and who is interpreting the results.

### Take Home Messages for Concussion

- Important to individualize treatment
- Any athlete suspected of having a concussion or other head injury should be removed from play and evaluated by a health care provider.
- No athlete should be returned to play the same day of a concussive injury.
- No athlete should be allowed to participate with acute symptoms.
- Any athlete with worsening symptoms, altered mental status, or other symptoms of intracranial bleeding should be transferred immediately to an emergency facility. Athletes with suspected cervical spine injury should be immobilized and transported immediately to an emergency facility.
- Severity of injury is more closely related to the burden and duration of symptoms, presence of amnesia, and prolonged confusion.
- Children and adolescents must be treated with more caution than adults. Available research demonstrates that it takes longer for the younger athlete to recover than their older counterparts.
- Once the athlete is asymptomatic, an individualized, gradual, step-wise return to play progression can be initiated.
- Treat each injury individually based on several factors including burden and duration of symptoms, age of athlete, gender, prior history of concussions and sport, mismatch between force of impact, and subsequent injury ().
- **No athlete who is symptomatic or has sustained a concussion on the same day should be allowed to return to sport.**
- "If in doubt, sit them out."

## HEAD INJURY COMPLICATIONS

### Second-Impact Syndrome (SIS)

- First described in 1973 and was initially thought to occur only in athletes under 18 years of age. Cases in college-aged athletes described the condition as "second impact dysautoregulation."
- **Controversy exists regarding whether SIS exists.**

- Rapid brain swelling and herniation after second head injury in athlete still recovering from initial head injury (during period of injury-induced vulnerability)
- Vascular congestion, increased ICP, and brain (uncal) and brainstem herniation probably result from loss of autoregulation of cerebral vasculature
- Second impact may be mild (e.g., a blow to chest or back that "snaps" the head). Athlete may initially appear dazed. Precipitous collapse, rapidly dilating pupils, coma, and respiratory failure ensue in seconds to minutes; end result is often death.

## Postconcussive Syndrome

- Characterized by persistent concussive symptoms for an extended period (often weeks to months)
- May be related to altered neurotransmitter function; severity and duration of symptoms may correlate with duration of posttraumatic amnesia
- Linked to depression in former professional football players with history of concussion
- MRI or CT and, if available, NPT should be considered for athletes with prolonged concussive symptoms.
- Treatment involves a multidisciplinary approach that may include psychotherapy, physical therapy, occupational therapy, speech therapy, vision therapy, vestibular rehabilitation, biofeedback, and medication (e.g., antidepressants, ADD medication, and anticonvulsants).

## Posttraumatic Seizure

- Three types of seizures may follow head injury: immediate, early, and late.
- **Immediate posttraumatic seizures (concussive convulsions)** are associated with no underlying structural or permanent brain injury, occur seconds after impact and involve a brief tonic phase, followed by bilateral myoclonic jerking. Seizures cease spontaneously and are followed by concussive symptoms; usually do not require anticonvulsant therapy. Recurrence uncommon.
- **Early** (<1 week) or **late** (>1 week) **posttraumatic seizures** may be partial or generalized. Many involve the temporal lobe, and most are associated with underlying brain pathology (e.g., contusion, hemorrhage, skull fracture) and require long-term anticonvulsant therapy. Seizures may recur in 20% to 25% of early and in up to 70% of late cases of posttraumatic epilepsy.
- **Differential diagnosis** for posttraumatic seizures includes idiopathic generalized epilepsy (poorly controlled or new onset), focal (partial) epilepsy associated with preexisting brain lesion or seizure focus, secondary epilepsy (e.g., drug-induced), convulsive syncope, and posttraumatic seizures.
- **Risk factors** for chronic posttraumatic seizures include depressed skull fracture, dural penetration, prolonged posttraumatic amnesia, acute intracranial hemorrhage, and early or late posttraumatic epilepsy.

## Chronic Traumatic Brain Injury (CTBI)

- CTBI (also called *chronic traumatic encephalopathy [CTE]* and dementia pugilistica) represents chronic and cumulative neurological dysfunction after repetitive head trauma. Observed most often in boxers, with more recent reports in professional wrestlers and American football players. Area of significant ongoing research
- Characterized by central nervous system dysfunction that may include cognitive impairment, ataxia, behavioral changes, parkinsonism, and pyramidal tract dysfunction
- Apolipoprotein E4 allele mutation has been noted to be a risk factor for CTBI in boxers.
- Varying degrees and durations of neuropsychological function have been observed after head injuries (both in sports-related and nonsports-related trauma).
- Recurrent head injury, cognitive dysfunction, and learning disability may be risk factors for reduced cognitive performance after head injury.
- Potential links to Alzheimer's disease, depression, and suicide have been recently reported.

**American College of Sports Medicine Consensus Statements**

- **Team Physician Consensus Statement: 2013 Update** available at http://www.acsm.org/docs/other-documents/team_physician_consensus_statement___2013_update-24.pdf (Accessed July 2016)
- **Concussion (Mild Traumatic Brain Injury) and the Team Physician: A Consensus Statement—2011 Update** available at http://www.acsm.org/docs/other-documents/team_physician_consensus_statement___2013_update-24.pdf (Accessed July 2016)
- **Sideline Preparedness for the Team Physician: A Consensus Statement—2012 Update** available at http://journals.lww.com/acsm-msse/Fulltext/2012/12000/Sideline_Preparedness_for_the_Team_Physician___A.24.aspx (Accessed July 2016)

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

American College of Sports Medicine Consensus Statements

- **Team Physician Consensus Statement: 2013 Update** available at http://www.acsm.org/docs/other-documents/team_physician_consensus_statement___2013_update-24.pdf (Accessed July 2016)
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- **Sideline Preparedness for the Team Physician: A Consensus Statement—2012 Update** available at http://journals.lww.com/acsm-msse/Fulltext/2012/12000/Sideline_Preparedness_for_the_Team_Physician___A.24.aspx (Accessed July 2016)

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# 46 NECK INJURIES

R. Lance Snyder

## GENERAL PRINCIPLES

Cervical spine injuries are most often seen in football and hockey but have occurred in wrestling, rugby, baseball, lacrosse, and mountain biking.

### Anatomy

- There are seven cervical vertebrae and eight cervical nerves.
- Spinal nerves exit above the vertebral body for which they are named; for example, the sixth cervical nerve exits at the C5–6 disc space.
- The cervical spine is divided into upper and lower segments. The upper segment includes C1 (the atlas) and C2 (the axis). The spinal cord occupies a little space because the canal is normal shaped. The atlas (C1) and the occiput account for 40% of cervical flexion. The axis (C2) has a finger-like projection, the dens, about which the Allis rotates; this accounts for 60% of cervical rotation. The key for C1 and C2 stability is the transverse atlantal ligament, which lies posterior to the body of C2 and connects C1 to C2. Distraction of this ligament can cause atlantoaxial instability (Fig. 46.1).
- The lower segment of the cervical spine includes C3–T1. The bony structure is relatively constant, with anterior column support provided by the anterior longitudinal ligament and the vertebral bodies in the discs. Posterior column support is supported by the posterior longitudinal ligament, facet articulation, facet capsule, interspinous ligament, and supraspinous ligament. The spinal cord occupies 75% of the canal at this level. Clinically, the space available for the spinal cord (SAC) ranges between 13 and 23 mm. The cord is stenotic when the available space is <13 mm. Typically, cord compression is present when the available space is <10 mm.

## HISTORY AND PHYSICAL EXAMINATION

### History

- First, the physician should enquire if and where the athlete has symptoms. Does the athlete have full movement and sensation of the extremities?
- The physician should then ask about the injury, including the direction of the athlete's helmet at the time of injury as well as the radiation of symptoms and their resolution.

### Physical Examination

- The athlete must be removed from the field in a safe and protected manner. Six people who have practiced the maneuver before injury, must logroll the athlete onto a spine board and safely transport the athlete to the sidelines. The helmet should not be removed, but the airway must be protected and maintained. The physician and trainers should be familiar with the removal of all types of facemasks. A power screwdriver is essential to accelerate facemask removal.
- A patient with pain must be examined with palpation.
- A fracture of the cervical spine may present with minimal pain, and the physician as well as the medical staff should be aware of this fact.
- Athletes who are suspected to have cervical spine injuries should be immobilized.
- If only radicular symptoms are present, the athlete should be examined for range of motion of the cervical spine, followed by a neurologic evaluation.
- Although this examination is not a complete and thorough workup, it should be performed at a later date.
- Strength is tested by comparing both sides at a scale of 0–5 (5/5 indicates full strength). The deltoids are checked with the corresponding C5 nerve root. The biceps correspond to C6 and the triceps correspond to C7.
- Sensation is checked with the thumb dorsally placed corresponding to C6, the long finger corresponding to C7, and the little finger corresponding to C8.
- Reflexes are then checked with the biceps reflex corresponding at the elbow to C6 and the triceps to C7.
- Spurling's test may be performed if fractures have been ruled out with the athlete's head being rotated and extended toward the affected extremity (Fig. 46.2).
- A positive Spurling sign is elicited when pain is exacerbated with this maneuver and is often caused by impingement of I nerve root with disc herniation or osteophytes.

## CERVICAL SPINE INJURIES

### Stingers or Burners

**Description:** This is a stretch injury of the nerve root.
**Mechanism:** Typically, hyperextension of the neck with lateral deviation of the head
**Presentation:** Typically, there is a stinging or burning pain into the shoulder arm or hand, which may be accompanied by weakness of the affected upper extremity. There may be numbness or tingling. Symptoms typically last from seconds to minutes but can last from days to weeks. The incidence of such injuries is 65% in college football athletes, with an 87% risk of recurrence.
**Differential diagnoses:** Cervical fracture or cervical herniation
**Diagnostics:** Cervical radiographs should be recorded in flexion and extension to rule out instability. Ideally, the T1 vertebral body must be included in the radiographs. Magnetic resonance imaging (MRI) may be required if symptoms persist.
**Treatment:** The athlete is rested until full strength has returned and sensation has returned to normal. Pad modification to prevent lateral deviation of the cervical spine may be beneficial. In addition, positional change may be beneficial as well (e.g., change from right to left guard).
**Prognosis:** The athlete is allowed to return to play when full range of motion and strength as well as sensation have returned. The return-to-play prognosis for such injuries is typically extremely good.

### Transient Quadriparesis

**Description:** Pathologic insult to the spinal cord, which may or may not be accompanied by transient hypoxemia to the spinal cord

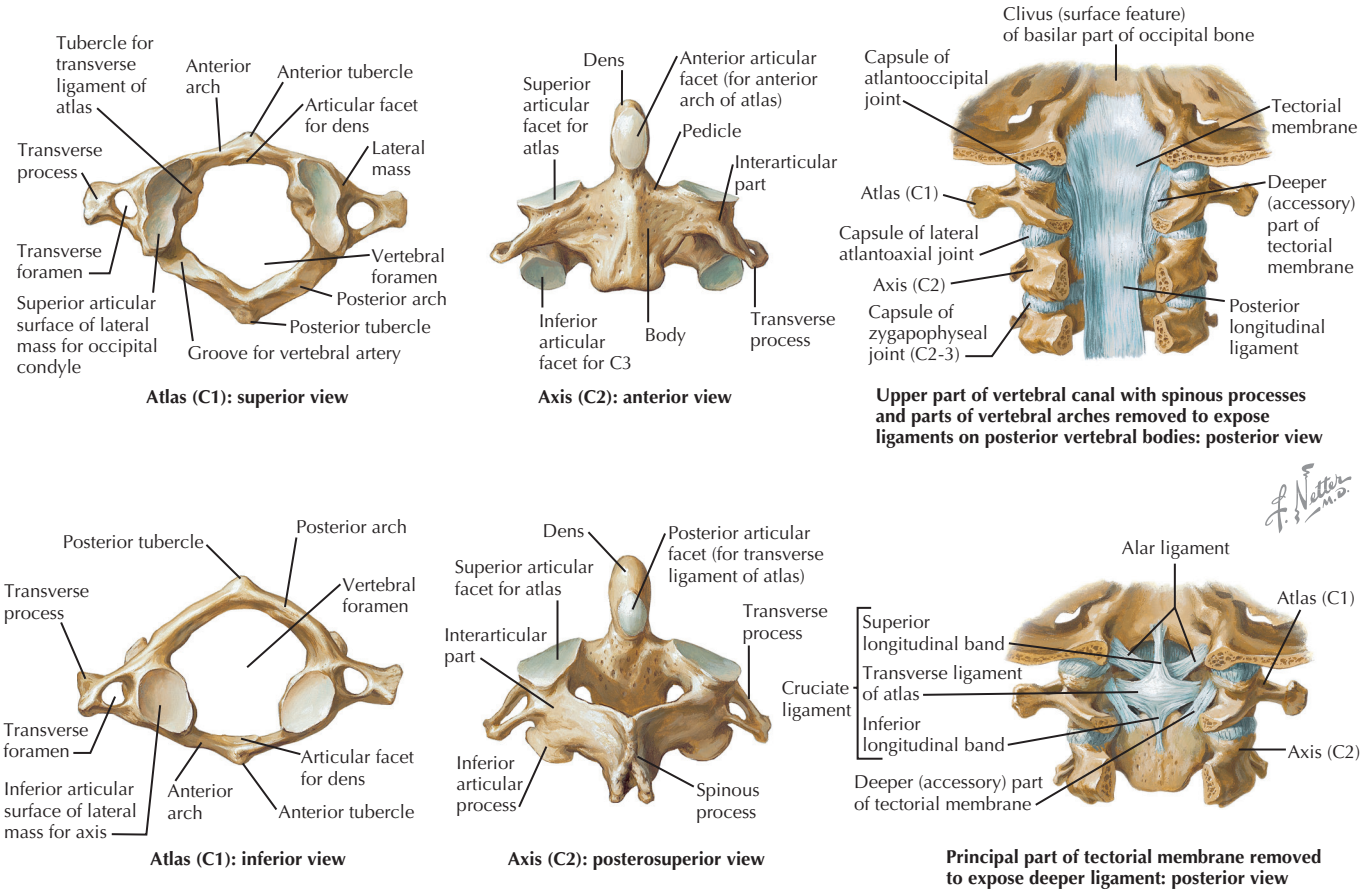



**Figure 46.1.** Anatomy of a vertebra.

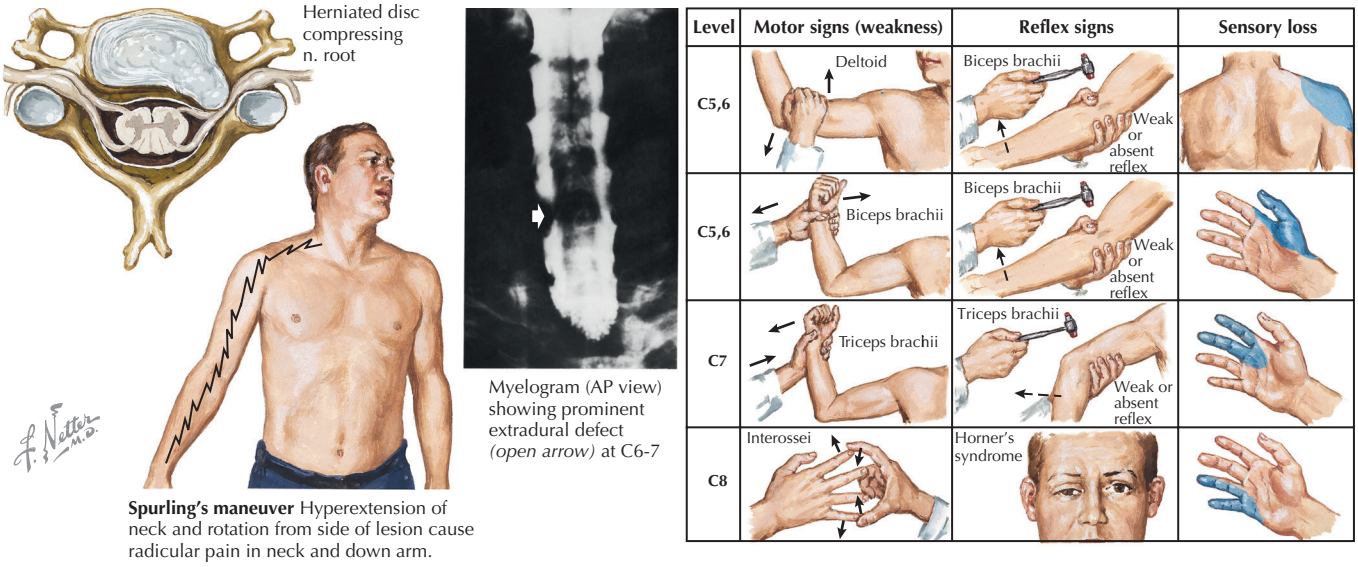



**Figure 46.2.** Cervical disc herniation: clinical manifestations.

**Mechanism:** With flexion of the cervical spine, the cord is pinched between the superior spinolaminar line and the superior aspect of the posterior lower vertebral body.

**Presentation:** An athlete will typically experience sudden attack of paralysis after a tackle or being struck. The paralysis may occur in all 4 limbs or be limited to the upper body. Weakness is typically short-lived, lasting only minutes, but can last from hours to days. Sensory changes may be present as well.

**Physical examination:** The airway must be cleared and maintained if there is loss of consciousness. A gross neurologic examination must be performed. The physician must check for upper motor neuron signs such as hyperreflexia. Reflexes of the upper and

lower extremity must be tested. Strength and sensation of the upper and lower body must be assessed as well.

**Differential diagnoses:** Cervical herniation, cervical fracture, and cervical fracture with dislocation

**Diagnostics:** Radiography

The port ratio is determined by comparing the space from the back of the vertebral body to the spinolaminar line with the width of the vertebral body. The port ratio should be >0.8; a value less than this indicates spinal stenosis as well as the risk of recurrence. AP and lateral radiographs should be obtained. A CT scan can help to delineate any fractures in the bony structures. An MRI will help delineate any pathologic damage to soft tissue structures.

**Treatment:** Careful observation of the athlete; serial observation and neurologic examinations should be performed. Diagnostic studies must be performed as well. There is not clear evidence regarding the use of steroids in such cases. Administration of a steroid regimen within 8 hours of neurologic damage has shown some improvement in certain cases. Typically, the dosage of methylprednisolone is 30 mg/kg bolus over 15 minutes and then 15 mL/kg over 23 hours.

**Prognosis:** Return to play is highly controversial. Several doctors believe that if there is no stenosis and no evidence of structural damage, athletes should be allowed to return to play; however, athletes must have regained full strength and sensation before returning to play.

## Cervical Disc Herniations

**Description:** Herniated disc material protrudes through a tear in the annulus causing compression on a root and rarely the spinal cord (see Fig. 46.2)

**Mechanism of injury:** Compression and rotation of the disc causes the annulus to tear. Pressure on the nucleus causes retropulsion through the tear and compression.

**Presentation:** A nerve root exits in the neuroforamen at the level above its vertebral body; therefore, a herniation at C5–C6 will cause compression of the sixth cervical nerve root. Athletes will likely experience corresponding radicular pain, which often radiates, along with weakness of the corresponding muscles.

**Physical examination:** A spurling sign may be present, which is pain radiating into the upper extremity when an athlete's head is extended and rotated toward the affected side. The athlete must be examined for sensory loss in a dermatomal pattern and checked for muscular weakness correlating to the corresponding nerve root.

**Diagnosis:** MRI is typically diagnostic and will indicate presence of a hard or soft disc between the affected vertebral bodies.

**Treatment:** Oral steroids or epidural injections are often beneficial. Anterior cervical discectomy and fusion or a foraminotomy of the knee may be needed to decompress the nerve root.

**Prognosis and return to play:** Athletes may be allowed to return to play when they have regained full function without any neurologic damage.

## Spear Tackler Spine

**Description:** Athletes experience ≥1 episodes of cervical neuropraxia; often seen in football athletes with the propensity to hit or tackle using the crown of the head. The NFL and the NCAA have banned the use of this technique; thereafter, the incidence of such catastrophic cervical spine injuries has correspondingly decreased.

**Diagnostic:** Radiographs may show cervical stenosis with a positive port ratio (<0.8). Moreover, degenerative abnormalities may also be observed on radiographs. Video analysis of athletes with the crown of the head and helmet intact is a common recent practice.

**Prognosis:** Athletes should not be allowed to return to athletic activity.

# FRACTURES

## C1 Fractures (Jefferson Fracture)

**Description:** Traumatic burst fracture of C1 (Fig. 46.3)

**Mechanism of injury:** Axial load

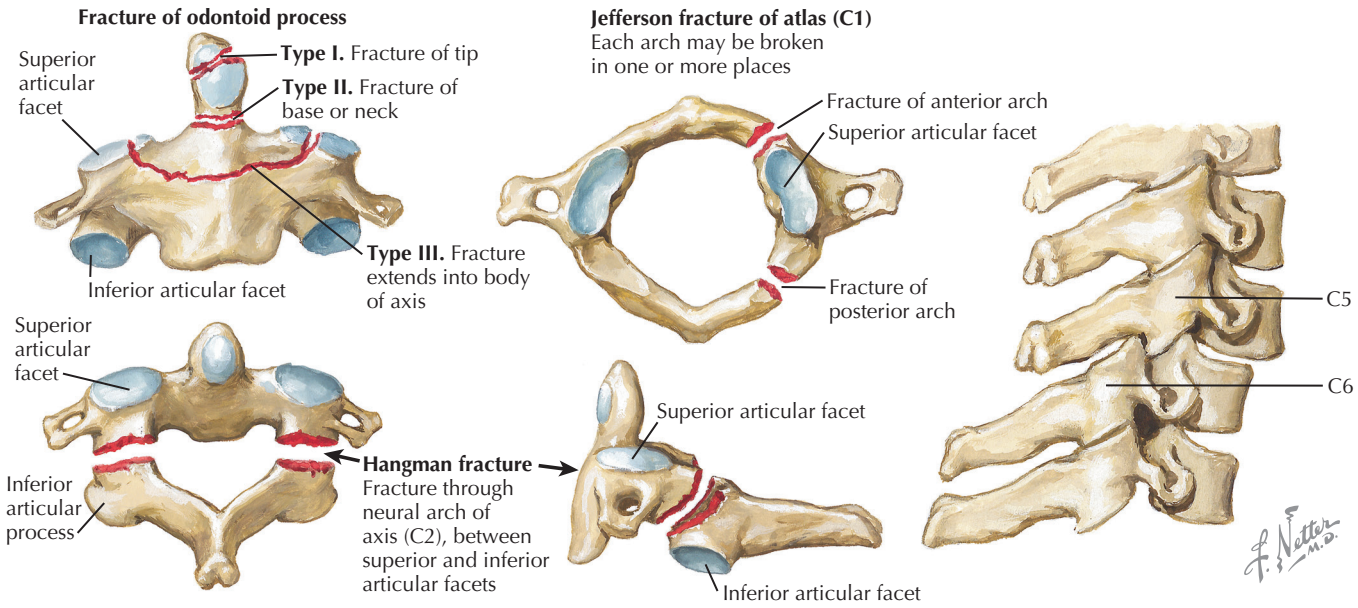



Figure 46.3. Fracture and dislocation of cervical vertebrae.

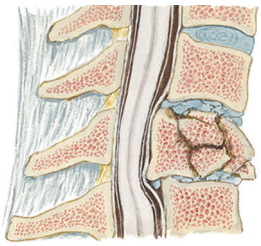
**Presentation:** Athletes often present with neck pain. Such an injury is unlikely to cause a neurologic injury because of the wide amount of space available for the spinal cord in this area.
**Physical examination:** Palpate for any tenderness. Check for lack of range of motion.
**Differential diagnosis:** Cervical strain
**Diagnostics:** Plain radiographs and CT scan are diagnostic.
**Treatment:** This is an unstable injury, and the athlete must be referred to an orthopedic spine surgeon or neurosurgeon.
**Prognosis and return to play:** The athlete will likely never return to play.

## C2 Fracture (Hangman's Fracture)

**Description:** Traumatic spondylolisthesis of C2 (Fig. 46.3)
**Mechanism of injury:** Axial load and extension
**Presentation:** Athletes will experience pain and may experience a sense of instability.
**Physical examination:** Palpation of the neck is performed, and range of motion is assessed. Such an injury typically does not result in paralysis or death because it actually widens the amount of area available to the spinal cord.
**Differential diagnoses:** Cervical strain
**Diagnostics:** Lateral radiographs can be diagnostic if there is displacement of the fracture. CT scans can further define the injury.
**Treatment:** The head should be immobilized, and the athlete must be referred to a spine specialist.
**Prognosis and return to play:** Poor

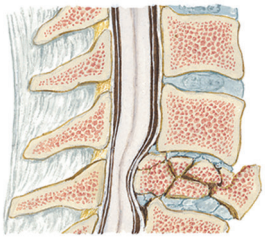
## Burst Fractures

**Description:** This injury involves a fracture of the vertebral body in the coronal and sagittal plane. There can be retropulsion of fragments, which often results in spinal cord damage (Fig. 46.4).
**Mechanism of injury:** When a pure axial load is applied to straighten the cervical spine, failure can result because strutting tissues cannot dissipate the force.
**Presentation:** Athletes commonly complain of neck pain, which may be the only symptom. There may be a root lesion accompanied by incomplete or complete paralysis.
**Physical examination:** Pain in the neck region. Often there is loss of motion in the cervical spine. There may be a sensory or muscular strength loss.
**Differential diagnoses:** Cervical strain
**Diagnostics:** Radiographs, CT scans, and MRI should be performed. A flexion and extension series should not be performed when there is an obvious fracture. The physician must be aware of the seemingly mild anterior teardrop fracture, which may indicate an unstable sagittal split in the anterior and posterior columns of the spine. Radiographs must include C1–T1 on the lateral films. An indication of fracture is loss of height (≥3 mm) of a vertebral body when compared with another vertebral body. An angulation of >11 degrees between the adjacent vertebral bodies is also a mark of instability. Measurements must be recorded between the vertebral bodies not just between the vertebral body and the inferior vertebral body line (Fig. 46.5).
**Treatment:** The ABCs of trauma management must be applied. Sports medicine team members mostly practice turning the patient from the prone to the supine position and transport the patient onto a spine board. This often requires at least 6 individuals because of the relatively large size of an athlete. Helmets should not be removed on the field, but face masks can be removed to gain airway access while the cervical spine is stabilized. The medical professional must be familiar with the head gear the athlete is wearing and have all the necessary tools, nonelectric and electric, to remove the face mask. Equipment differs from sport to sport, and this should be recognized. Anatomic differences between pediatric and adult athletes must be also taken into account. Once the athlete is stabilized, transport him/her to the emergency department for further treatment.

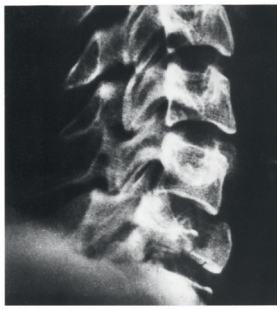


**Type III.** Fracture through entire vertebral body with fragmentation of its anterior portion. Posterior cortex intact but projects into spinal canal causing damage to cord and/or nerve roots.

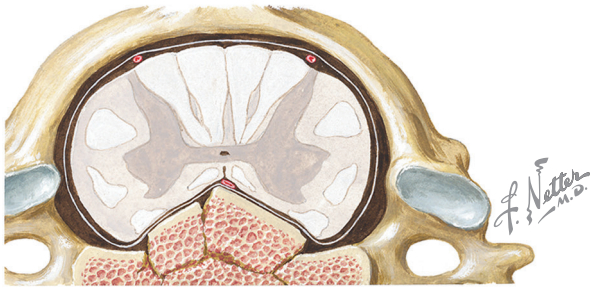
X-ray film: Type III fracture of C5



**Type IV.** "Burst" fracture. Entire vertebral body crushed, with intraspinal bone fragments.



X-ray film: Type IV fracture of C6

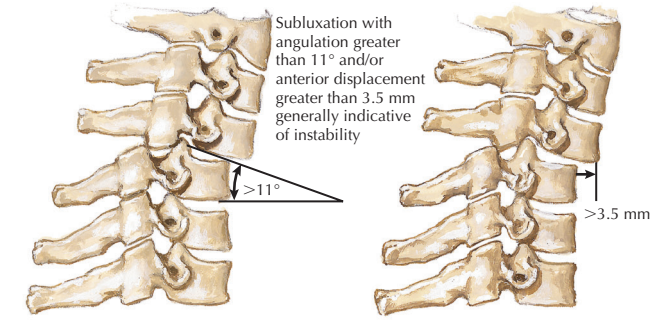



Dislocated bone fragments compressing spinal cord and anterior spinal artery. Blood supply to anterior two thirds of spinal cord is impaired.

**Figure 46.4.** Compression fractures of cervical spine.

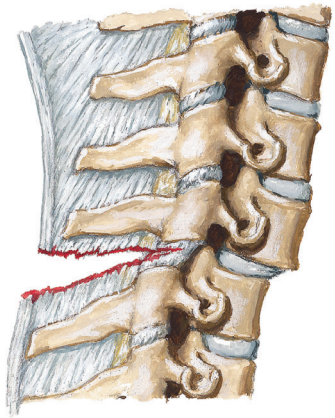
## DISLOCATIONS

**Description:** An injury wherein one or both facets are dislocated; an associated facet fracture may be present as well (Fig. 46.6)
**Mechanism of injury:** A flexion distraction injury
**Presentation:** Pain or tenderness in the cervical spine; pain with range of motion; muscular strength or sensation loss. There may be associated neurologic injury, which can include nerve root injury or a complete lesion.
**Differential diagnoses:** Cervical strain and cervical disc herniation



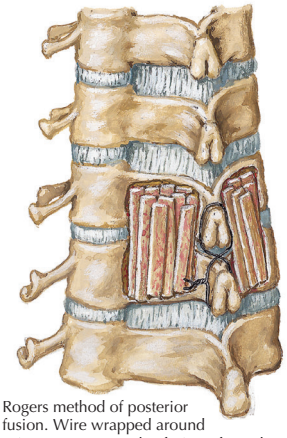

Subluxation with angulation greater than 11°

Anterior displacement greater than 3.5 mm



Tear of interspinal and supraspinal ligaments characteristic of anterior dislocation of spine.

Lateral radiograph shows severe kyphotic angulation in cervical dislocation.

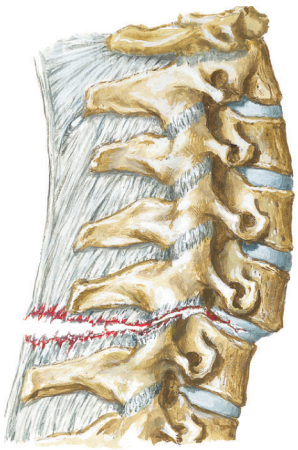


Rogers method of posterior fusion. Wire wrapped around spinous processes plus fusion of vertebrae.

Postoperative radiograph shows corrected alignment and fixation wire in place.

F. Netter M.D.

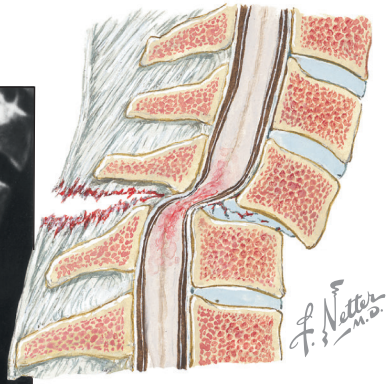
**Figure 46.5.** Subluxation and ligamentous instability of cervical spine.



Anterior dislocation of C5 on C6 with tear of interspinous ligament, facet capsules, and posterior fibers of intervertebral disc

X-ray film showing moderate (1st-degree) dislocation of C5. If there is no evidence of spinal cord injury, spontaneous healing may occur following reduction by traction and prolonged bracing.

X-ray film: 3rd-degree dislocation of C5



Severe spinal cord injury in 3rd-degree cervical dislocation. Any evidence of cord or nerve root damage is indication for prompt:
- **Reduction** by traction
- **Decompression** by disc removal
- **Fixation** by interbody fusion

**Figure 46.6.** Dislocation of cervical spine.

**Diagnostics:** Radiographs will usually show >25% displacement on the posterior vertebral body line from the adjacent posterior vertebral body line for unilateral facet dislocation. There may be >50% displacement for a bilateral facet dislocation. A >3.5-mm displacement of the posterior vertebral line of one vertebral body compared to the other indicates instability. CT scans should be performed as well, which will delineate fractures of facets and provide an overall indication of the force and treatment pattern. MRI will indicate the presence of myelomalacia or cord damage as well as presence of a herniated disc. A herniated disc is observed in up to 55% of cases with bilateral facet dislocation.

**Treatment:** On the field, the head and neck are mobilized until the athlete is safely transported to the hospital. The helmet should not be removed. An orthopedic spine surgeon or neurosurgeon should then be consulted. Reduction is typically performed with weight applied to Gardner–Wells tongs; this procedure is usually performed with the patient awake to ensure no worsening of any neurologic injury. The herniated fragment can retropulse into the spinal cord canal. Certain physicians propagate pre-reduction MRI to identify any herniated discs and reduce the likelihood of retropulsion.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 47 EYE INJURIES

David E. Olson • Robby S. Sikka • Jodi M. Blustin • Michael S. Broton

## GENERAL PRINCIPLES

- Over 42,000 sports- and recreation-related eye injuries were reported in 2000: of these, 72% occurred in individuals younger than 25 years and 43% in people younger than 15 years. 80% of injuries occurred in males.
- Approximately 1.5% of all sports-related injuries involve the eye or ocular adnexa; these injuries have a high morbidity rate.
- In the United States, basketball is the leading cause of sports-related eye injuries; in Europe and South America, soccer is the leading cause.
- Although eye protectors cannot eliminate the risk of injury, appropriate and well-fitted eye protection can reduce the risk of significant eye injury by as much as 90%.
- The American Association of Pediatrics and American Academy of Ophthalmology 2004 position statement on protective eyewear in young athletes categorizes sports by the risk of eye injury to the unprotected eye (Table 47.1). Although there is no ideal collecting system for data, the National Collegiate Athletic Association (NCAA) Injury Surveillance System (ISS) tracks injuries in college sports (Table 47.2).

### Mechanisms of Eye Injuries in Sports

- The severity of eye injuries can be positively correlated with the total impact force, rate of force onset, and kinetic energy of an impacting object.
- Ocular injuries fall into several broad categories (Table 47.3).
- Open globe injuries are full-thickness wounds to the eye wall (cornea or sclera) and result from a rupture or laceration. Sports that cause ruptured globes typically have a stick or projectile that fits into the orbit. A previous surgery or eye disease increases the risk of an open globe injury.
- Lacerations may be caused by objects that "slice" or penetrate the eye, which may lead to open globe injuries.
- Closed globe injuries are those that do not completely penetrate the cornea or sclera. These include lamellar lacerations, corneal abrasions, contusions, hyphema, or injury to the choroid, macula, retina, or optic nerve.
- Blunt injuries, typically causing contusions, globe rupture, or adnexal injury, account for most sports-related eye injuries. Contusions are usually caused by blunt objects smaller than the orbit (e.g., golf ball or finger). In addition, several objects will deform significantly on impact (e.g., soccer ball), producing a "knuckle" that will impact the eye (Fig. 47.1). With objects smaller than the orbit, there is generally a greater force transmitted to the internal structures of the eye, whereas with larger objects, there is an increased risk of orbital wall fracture and occult internal ocular injuries.
- Radiant energy or UV burn injuries are less common but may occur in activities that take place at a high altitude or on snow.

### Principles of Protection From Eye Injuries in Sports

- Protective devices work by deflecting the impact energy away from the eye and dissipating the energy over time and area. This is typically accomplished with either a lens or a mechanical grid (e.g., wire-framed face guard or mesh-fencing helmet).
- Inappropriate fit of protective gear can decrease the protection offered, placing the eye at an increased risk.
- The gear must be comfortable and not interfere with the performance of athletes.
- Contact lenses offer no protection. Athletes who wear contact lenses should wear one of these three options:
  - Contact lenses plus the appropriate protective eyewear
  - Polycarbonate lenses in sports frames that pass the appropriate ASTM standard
  - Over-the-glasses protector that conforms to the appropriate ASTM standard

### Certification and Selection of Eyewear

- Organizations that certify sports protective eyewear include the Protective Eyewear Certification Council (PECC), Canadian Standards Association (CSA), Hockey Equipment Certification Council (HECC), and National Operating Committee on Standards in Athletic Equipment (NOCSAE). The equipment approved by these organizations commonly bears their seal and should be selected when available (Table 47.4).
- ASTM International has written performance standards based on design and strength, upon which many of these organizations base their certification (see Table 47.4).
- For sports with no appropriate ASTM standards or certified equipment, the American National Standards Institute (ANSI) should be considered.

### Preparticipation Eye Examination

- Preparticipation eye examinations should include the assessment of visual acuity, visual fields, pupillary size and responsiveness, eye movements, and ophthalmoscopy.
- Documentation of anisocoria is imperative in order to determine if it is pre-existing or caused by an acute injury. Up to 20% of the population may have physiologic anisocoria of >0.4 mm. In physiologic cases, there will be no associated visual field defects or diplopia. The afferent and efferent pupillary light reaction will also be normal.
- Assess athlete's history for high degree of myopia, surgical aphakia, retinal detachment, eye surgery, infection, or injury. Also assess family history for retinal detachment, retinal tears, or diabetic retinopathy. All these conditions increase the risk of serious eye injury and thus require ophthalmologic consultation before participation in high- or very-high-risk sports.

### Visual Risk Factors

- Best-corrected visual acuity worse than 20/40 in either eye or spectacle correction for myopia or hyperopia >6 diopters; disease, degeneration, or structural weakness of the eye itself; thin sclera; history of retinal degenerative disease; and history of eye surgery that weakens the outer wall of eye, particularly cataract or refractive surgery. Athletes with such risk factors should be evaluated by an ophthalmologist before engaging in high- or very-high-risk sports.

**TABLE 47.1 RISK CATEGORIES FOR SPORTS**

| | |
|---|---|
| **High risk** | **Small, fast projectiles**<br>• Air rifle/BB gun<br>• Paintball<br>• Hard projectiles, fingers, "sticks," close contact<br>• Baseball/softball/cricket<br>• Basketball<br>• Fencing<br>• Field hockey<br>• Ice hockey<br>• Lacrosse (in men and women)<br>• Squash/racquetball<br>• Street hockey<br>**Intentional injury**<br>• Boxing<br>• Full-contact martial arts |
| **Moderate risk** | • Fishing<br>• Football<br>• Soccer/volleyball |
| **Low risk** | • Bicycling<br>• Noncontact martial arts<br>• Skiing |
| **Eye safe** | • Gymnastics<br>• Track and field |

Modified from Vinger PF. A practical guide for sports eye protection. *Phys Sports Med.* 2000;28(6):49-69. Committee on Sports Medicine and Fitness. Protective eyewear for young athletes. *Pediatrics.* 2004;113(3):619-622.

**TABLE 47.2 RELATIVE RISK OF EYE INJURIES AS REPORTED IN NCAA ISS**

| | *Annual Risk of Eye Injury* | |
|---|---|---|
| | **Men (%)** | **Women (%)** |
| Wrestling | 1.67 | |
| Basketball | 0.97 | |
| Field hockey | | 0.88 |
| Basketball | | 0.50 |
| Softball | | 0.50 |
| Soccer | 0.26 | 0.24 |
| Baseball | 0.20 | |
| Volleyball | | 0.12 |
| Football | 0.11 | |
| Ice hockey | 0.08 | 0.00 |
| Lacrosse | 0.06 | |
| Gymnastics | 0.00 | 0.00 |

Data from Dick R, Agel J, Marshall SW. National Collegiate Athletic Association Injury Surveillance System. *J Athl Train.* 2007;42(2):173-182.

- Disability from high corrective spectacle lenses can sometimes be mitigated by contact lenses; however, contact lenses themselves can be a risk factor.
- Functionally one-eyed athletes face an additional risk. A person is functionally one-eyed when loss of the better eye would result in a significant change in lifestyle owing to poor vision in the remaining eye.

**TABLE 47.3 RELATIVE FREQUENCY OF EYE INJURIES**

| **Most Common** | **Relatively Infrequent** | **Eye Emergencies** |
|---|---|---|
| Corneal abrasion | Chemical burns | |
| Corneal foreign body | Vitreous hemorrhage | Retinal detachment |
| Conjunctival foreign body | Retinal hemorrhage | Lens dislocation |
| Subconjunctival hemorrhage | Retinal edema | Blowout fracture of the orbit |
| Eyelid laceration | Hyphema<br>Injury to the lacrimal system | Optic nerve injury |

**Figure 47.1.** Large object causing injury to the globe.

- A child with vision worse than 20/40 should be considered functionally one eyed. Assessment of adults is more difficult because their judgment and values determine the visual impairment they are willing to accept. Special considerations are necessary for such athletes.
- The only sports absolutely contraindicated for the functionally one-eyed athlete are boxing and full contact martial arts because the risks of serious injury are high and there is a lack of effective eye protection. Wrestling and noncontact martial arts have a lower incidence of eye injury, but also do not have effective eye protection. They should be discouraged for functionally one-eyed athletes and banned for monocular athletes.

## Examination and Functional Testing After Injury

### History

- Mechanism of injury is important. Historical features such as type of trauma (blunt versus penetrating), the direction of force,

**TABLE 47.4 STANDARDS AND CERTIFYING ORGANIZATIONS FOR SELECTED SPORTS**

| Sport | Eye Protection | Standards | Certifying Organizations |
|---|---|---|---|
| Baseball | Polycarbonate or wire face guard attached to a helmet while batting; sports goggles with polycarbonate or TriVex lenses while on the field | ASTM F910 | PECC |
| Basketball | Sports goggles with polycarbonate or TriVex lenses | ASTM F803 | PECC |
| Field hockey | Full face mask for the goalie; sports goggles with polycarbonate lenses or wire mesh goggles while on the field | ASTM F803 | PECC |
| Football | Wire face mask and polycarbonate eye shield attached to the helmet | | NOCSAE |
| Ice hockey | Helmet with full face protection | ASTM F1587<br>ASTM F513 | CSA/HECC |
| Men's lacrosse | Helmet with full face protection | | NOCSAE |
| Women's lacrosse | Full face protection or sports goggles with either polycarbonate lenses or wire mesh goggles | ASTM F803 | PECC |
| Paintball | Full face protection | ASTM F1776 | PECC |
| Racket sports | Sports goggles with polycarbonate or TriVex lenses | ASTM F803 | CSA/PECC |
| Skiing | High impact–resistant eye protector | ASTM F659 | PECC |

From the American Academy of Ophthalmology (AAO) (www.aao.org).

size of the object, and whether eye protection was worn influence the type of injury.
- Relevant signs and symptoms include pain, decreased visual acuity, diplopia, flashers, floaters, and halos around lights.
- Remember that intraocular injuries or foreign bodies may be painless because the lens, retina, and vitreous have no pain sensation.

### Physical Examination

- **Inspection:** Look for signs of external trauma, bruising, fullness, or subcutaneous emphysema. Mild external trauma can be a sign of more severe internal ocular injury. Do not manipulate or forcibly open an eye if mechanism and examination cannot rule out a ruptured globe.
- **Visual acuity:** This is the single most important physical examination feature in evaluation of the eye. Visual acuity can be determined with a Snellen eye chart if one is available. Alternatively, have the patient read printed material from different distances. Changes in visual acuity are more important than absolute values. Any acute decrease in acuity necessitates immediate further evaluation and referral (Box 47.1).
- Confrontational visual field testing: Examiner tests visual fields in all four quadrants of each eye using his or her own eye as a control.
- **Ocular motility testing:** Check for the cardinal movements of the eye. Deficiencies in upward gaze may suggest entrapment with orbital blow-out fracture or neuro-ophthalmologic pathology. There will often be associated diplopia. Do not perform this evaluation if you suspect there may be a globe laceration.
- **Pupillary examination:** Initially, evaluate the pupils for uniform roundness and symmetry. Anisocoria may denote an injury along the pupillary pathways. Check direct and consensual responses. If a defect is found, a swinging flashlight examination may be performed to help localize the injury by quickly moving the light back and forth between the eyes.
  - Afferent pupillary defect, or injury to the retina or optic nerve (cranial nerve [CN] II), results in paradoxic dilation

**BOX 47.1 INDICATIONS FOR REFERRAL**

- Any loss of visual acuity
- Visual field cuts
- Pupil asymmetry or abnormal pupillary reaction
- Perception of flashing lights
- Orbit asymmetry
- Hyphema
- Laceration of eye or complex laceration of lids
- Orbital pain with movement of the eye
- Halos around lights
- Abnormal EOM
- Abnormal mass on inspection
- Diplopia

*EOM,* Extraocular movements.

when the light hits the ipsilateral eye. In this case, the consensual response would be intact because the efferent pathway (CN III) from the contralateral eye would be maintained. This is known as a *Marcus Gunn pupil*.
    - An efferent lesion would limit direct and consensual response in a lesion ipsilateral to the affected eye.
- Anterior chamber assessment can be accomplished by shining a light from the temporal side of the eye toward the medial aspect. Inspect the iris for a symmetric round opening and presence of hyphema.
    - Assess depth of anterior angle. Increased medial shadowing can suggest a narrow angle, though a special slit lamp examination is needed to visualize and fully assess the anterior angle.
    - Assess for a layer of blood that accumulates at the 6 o'clock position. Look at the iris for uniformity. The iris may prolapse into the wound producing an irregular pupil.
- **Ophthalmoscopy:** Often impractical on the sideline, but can add useful information for a more complete ophthalmologic examination; very useful in evaluation of acute visual loss
- **Red reflex:** Normal red reflex is evenly colored and without shadows.
    - Hyphema, acute swelling of the lens, and vitreous hemorrhage may lead to loss of this reflex.
    - Retinal edema occurs in contusion injuries to the globe as well as retinal detachments. This leads to interruption of circulation and may alter the intensity of the red reflex.
- **Fundoscopic examination:** Assess optic disk margins, cup-to-disk ratio, blood vessels, and retina. This is ideally performed after pupillary dilation.
    - Contraindications to pupillary dilation include (i) need for neurologic monitoring, including pupillary response, with significant head trauma; (ii) suspected acute angle glaucoma or narrow anterior chambers; and (iii) iris-supported ocular lens implants.
- **Neurologic:** Decreased sensation or numbness in the V2 distribution may accompany orbital floor fractures.
- **Special tests:**
    - **Fluorescein staining:** Instill anesthetic drops such as proparacaine hydrochloride 0.5% and have the patient blink. Fluorescein strips can then be moistened with sterile water and touched to the inferior cul-de-sac, with care taken to not brush the cornea. Use a cobalt light or Wood's light to examine for defects, which fluoresce yellow-green.
    - **Applanation tonometry:** Hand-held electronic tonometers are convenient for measuring intraocular pressure (IOP). Topical anesthetic drops should be instilled before use. Normal IOPs range 10–21 mmHg.
    - Pressure can also be estimated by palpating the globe and using palpated tension in the fellow eye for a rough comparison.
        - Care should be taken to avoid compression of a ruptured or perforated globe by this test.
        - IOP may be elevated with hemorrhage or swelling of orbital contents and may be decreased in certain cases of blunt injury to the globe.

## COMMON SPORTS INJURIES

### Corneal Abrasion

**Description:** Results from cutting, scratching, or abrading the thin protective surface of the anterior portion of the ocular epithelium. Disruption of the cornea near the central visual axis interferes with visual acuity. Patients often report a history of ocular trauma or foreign body sensation and subsequent acute pain. Aggressive eye rubbing can also cause injury. Predisposing factors include foreign body, contact lens, and previous history of corneal abrasion.

**Signs and symptoms:** Red eye, pain, photophobia, conjunctival injection, tearing, foreign body sensation, gritty feeling, and decreased visual acuity if central corneal area is involved; symptoms are worsened by blinking, rubbing, and light exposure

**Examination:** After fluorescein staining, corneal abrasions and foreign bodies will appear yellow-green against a blue background illumination. Topical anesthetic may be necessary to allow examination. Additional ocular injury must be ruled out, and any foreign bodies should be removed. Small conjunctival lacerations heal quickly and may mask any penetrating injuries of the globe.

**Treatment:**

- Healing is best facilitated by management of pain and controlling lid movement.
- Postinjury infection is uncommon; however, topical antibiotics may be useful in cases where contamination of the eye with debris has occurred, as well as in immune-compromised patients. Bacitracin, erythromycin, or gentamicin ointment has better lubricating properties than drops and is considered first-line treatment.
- In contact lens wearers, a gram negative/antipseudomonal antibiotic such as ciprofloxacin should be administered, and contact lens use should be discontinued until the abrasion is completely healed and the antibiotic course completed. **An ophthalmologist should follow these patients within 24 hours.**
- Patching is no longer recommended. Several randomized controlled trials (RCTs) have failed to show an increase in healing rate or improvement in pain with patching. Patching may cause increased pain, decreased oxygen delivery, and increased moisture, resulting in increased risk of infection.
- In large abrasions associated with significant pain and photophobia, topical nonsteroidal anti-inflammatory drugs (NSAIDs) such as Voltaren or Acular are modestly useful in reducing pain.
    - **Topical mydriatics have not been proven to be beneficial.** Mydriatics were previously thought to relieve ciliary muscle spasm. However, one RCT showed that pain was similar in patients using eye lubricant alone or combined with topical NSAID.
    - Topical anesthesia can be effective in relieving discomfort and allowing for an appropriate examination. It has long been believed that these medications should never be prescribed or used for a prolonged period due to in increased risk of corneal ulceration. However, a recent study showed that topical Tetracaine can be safely used with a frequency up to every 30 minutes for a 24-hour period. In this study, 116 total patients were studied, and the effectiveness of topical Tetracaine versus topical saline was compared. There was no increase in healing time with the use of topical anesthesia as determined by repeat fluorescein examination at 48 hours. Moreover, patients in this group reported significantly greater drug effectiveness compared to patients receiving saline drops. Based on this study, topical Tetracaine can be considered for a 24-hour period to allow an athlete to return to play the same day. Use should be considered on a case-by-case basis.
    - In general, avoid topical corticosteroid preparations except in complicated cases because they can encourage fungal and viral infections. In general, an ophthalmologist should be consulted if topical corticosteroid use is being considered.

**Prognosis:** Uncomplicated corneal epithelial injuries heal completely within 24–72 hours without scarring. Although frequently contaminated, they rarely become infected. **Recurrent epithelial erosion** is infrequent, but patients must be warned that this may occur.

## Foreign Bodies on Eye and Eyelid Surfaces

**Description:** May result from penetrating ocular trauma, which is the most common cause of blindness in teenage and young males. The size, shape, and momentum of the object at the time of impact affect the site of ocular penetration. Deeper penetrating objects can lodge within the orbit and cause retinal injury. Superficial corneal foreign bodies are much more common than deeply embedded foreign bodies. Corneal scarring or infection may occur.

**Signs and symptoms:** Same as for abrasions

**Examination:**

- Minute foreign bodies may require magnification to be appropriately visualized and removed.
- If a metallic foreign body has been embedded for hours to days, a rust ring may be present.
- Localization can be enhanced by fluorescein stain. Foreign bodies will appear yellow-green against the blue background illumination. Use of topical anesthetic may be necessary to facilitate examination.
- Both upper and lower conjunctival fornices should be carefully examined for presence of foreign body.
- Upper eyelid should be everted and the conjunctival surface inspected for the presence of foreign body.
- A positive Seidel test will help detect leaking aqueous or exposed vitreous. In this test, fluorescein ophthalmic strips are wetted with normal saline and then applied to the superior conjunctiva allowing the dye to flow over the cornea. The concentrated fluorescein is dark orange, but if it becomes diluted with aqueous, it turns bright green under blue light. The presence of an intraocular foreign body (IOFB) suggests globe penetration. The patient may be asymptomatic if the foreign body is below the epithelial or conjunctival surface. Over a few days, the epithelium may grow over small corneal foreign bodies, with pain reduction. If a corneal infiltrate is noted, an infectious cause should be considered. Foreign bodies can cause a small sterile inflammatory reaction around the foreign object. However, if a large infiltrate, ulceration, significant anterior chamber reaction, or significant pain is present, it should be managed as an infection.
- If there is concern for IOFB, helical computed tomography (CT) scan with 1-mm axial and coronal cuts or biomicroscope ultrasound can be considered. Referral should be made to an ophthalmologist for a dilated examination and management.

**Treatment:** For corneal foreign bodies, apply short-acting topical anesthetics. Removal of the foreign body using irrigation, sterile needle, and foreign body removal instrument should be performed; however, if there is a likelihood of penetration through >25% of the cornea, referral should be made to an ophthalmologist for surgical removal. Cotton-tip applicators are not appropriate because the large surface area may cause an epithelial defect.

- Always approach the foreign body tangentially and aim just beneath it to avoid accidental corneal perforation.
- Often, vigorous flushing with a forceful stream of irrigation solution, such as normal saline, from a squeeze bottle is enough to dislodge the foreign body.
- For metallic foreign bodies, a magnetic spud may be helpful.
- Topical antibiotic ointments such as bacitracin or Ciloxan should be prescribed to prevent infection.
- Rust rings may be removed by a well-trained physician with an Alger brush or automated burr. Rust rings should be visualized using a slit lamp.

## Eyelid Lacerations

**Description:** May occur in association with blunt trauma as well as from sharp objects and can result from the propulsion and shattering of eye protection equipment (Fig. 47.2).

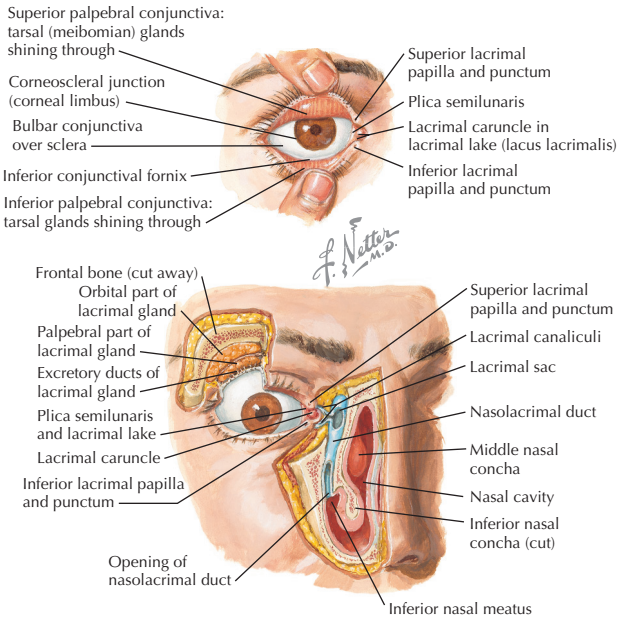


**Figure 47.2.** Orbit eyelids and lacrimal apparatus.

**Signs and symptoms:** Swelling, hemorrhage, and anatomical disruption of lids. Damage may be subtle and appearance may be normal.

**Examination:** Evaluation of normal anatomic relationship of lid margins and front surface of the globe as well as symmetry with uninjured side; opening and closing functions are specifically assessed, particularly to verify that lids can be spontaneously closed. Rule out the possibility of upper eyelid ptosis or lacerations in lacrimal drainage (canalicular) system. The globe must be thoroughly inspected for signs of damage.

**Treatment:** Lacerations require individual suturing of lid tissue layers and additional repairs specific to any injuries involving integrity of the lacrimal drainage apparatus. **Particular injuries requiring ophthalmology referral include:**

- Lacerations involving the upper or lower lid margin
- Lacerations suspected to involve the lacrimal sac or duct
- Lacerations with exposure of orbital fat
- Horizontal lacerations with ptosis and possible disruption of the tarsal plate
- Any laceration with avulsion of eyelid tissue

**Prognosis:** Because of the rich vascular supply to eyelids, healing is rapid and deformities are minimal in cases of minimal tissue loss and good anatomical approximation.

## Blunt Trauma to Orbit

Blunt injuries to the globe are the most common sports-related injury to the eye and may be associated with ruptured globe or hyphema. They result from facial blows directed toward orbital contents or from sudden pressure increases transmitted to the eye from surrounding orbital tissue.

### Ruptured Globe

**Description:** Occurs when the full thickness of the cornea or sclera is breached. There is potential for serious morbidity of the eye with this injury.

**Epidemiology:** A study of sports-related open globe injuries ordered the frequency of occurrence from most to least common as follows: fishing, hunting/shooting recreation, baseball/softball, golf, basketball, and racket sports.

**Mechanism of injury:** Rupture occurs when hard objects increase IOP, causing rupture at the weakest point. Globe lacerations

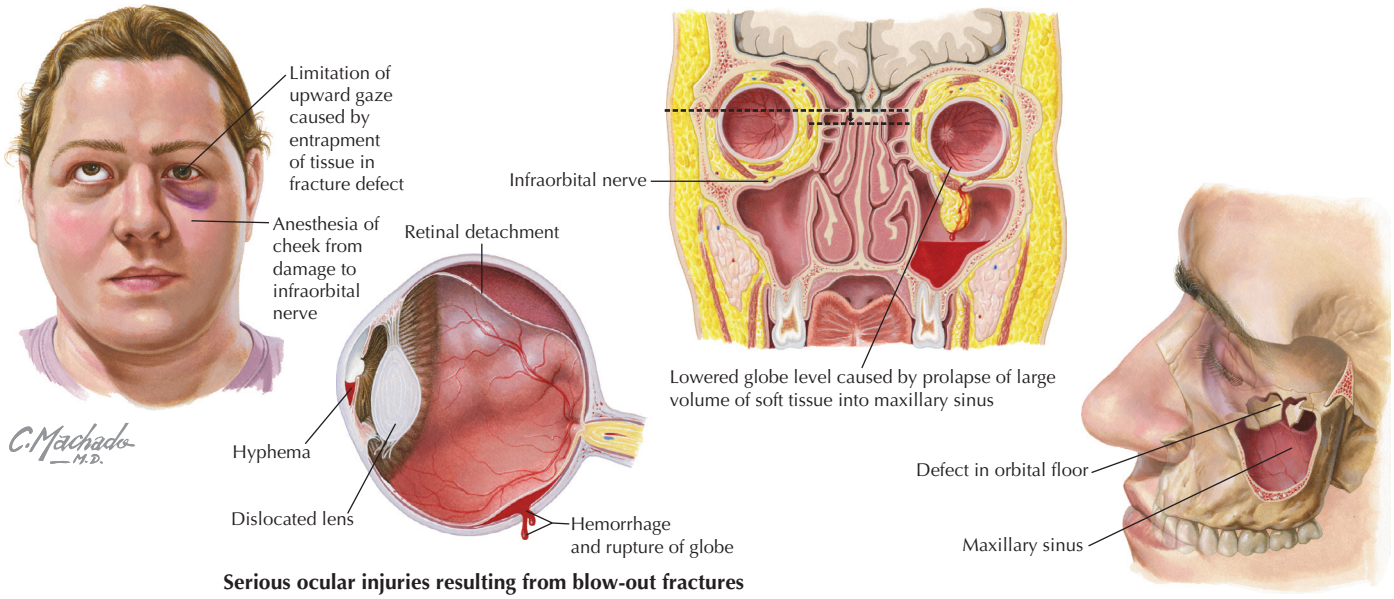


**Figure 47.3.** Orbital blowout fractures.

occur when sharp (e.g., sticks) or small hard objects (e.g., shrapnel, BBs, shattered eye protection) enter the globe at a high velocity.

**Physical examination:**
- Evaluate anterior segment of globe for signs of subconjunctival hemorrhage.
- The pupil should be round, central, and symmetric with the fellow eye.
  - Lacerations of the cornea frequently incarcerate iris tissue, causing distortion and displacement of the pupil.
  - Scleral lacerations also may displace the pupil because of herniations of the uveal tract through the defect.
  - Prolapsed uveal tissue presents as a dark brown or black mass, even in those with fair complexion and blue eyes.
  - Lacerations of the globe may result in subluxation of the crystalline lens.
  - Intraocular bleeding is a frequent complication, causing obscuration of ocular media and loss of the red fundus reflex.
  - IOP may be decreased.

**Treatment:** Emergent ophthalmology referral:
- Manage other trauma including other head or neck trauma.
- Patient should not be given anything orally.
- Keep a **rigid** shield over the affected eye at all times. Patches are not sufficient.
- Avoid ointments and other topical medications.
- Tetanus status should be updated.
- Other considerations to discuss with an ophthalmologist include narcotics, prophylactic antibiotics, and imaging. Imaging is often necessary to rule out associated foreign bodies.
- Penetrating objects that remain in place should be secured in place without removal and covered for protection. A Styrofoam cup may be a useful adjunct for this.

**Prognosis:** In patients with penetrating eye trauma, predictors of excellent final visual acuity (defined as 20/60 or better) are initial postinjury visual acuity of 20/200 or better, wound location anterior to the plane of insertion of the four rectus muscles, wound length ≤10 mm, and sharp mechanism of injury. Predictors of poor outcomes include initial visual acuity of light perception or no light perception, wounds extending posterior to rectus muscle insertion plane, wound length >10 mm, blunt or missile injury, associated hyphema, lid involvement, vitreous loss, and retinal detachment. Only 50% of children with ruptured globe injuries recover good visual acuity.

**Return to play:** After a ruptured globe injury, return-to-play decisions should include consultation with an ophthalmologist and include discussion of risks and benefits of participation as outlined earlier.

## Orbital Fracture

**Description:** An orbital blowout fracture occurs when blunt trauma to the eye or orbit is transmitted to the bony walls of the orbit, causing fracture. This may result in entrapment of contents of the orbit, including the muscles of the eye, leading to restricted gaze and diplopia (Fig. 47.3).

**Signs and symptoms:** A high index of suspicion for this injury should be present when an object larger than the ocular orbit strikes the eye. History should elicit the mechanism, change in visual acuity, pain, previous visual impairment, and use of eye protection. Additional symptoms suggestive of injury are diplopia, particularly with upward gaze, and ipsilateral nosebleed.

**Examination:**
- Rule out ruptured globe.
- Findings include:
  - Enophthalmos (posterior displacement of eye), which may have the appearance of relative ptosis
  - Periorbital ecchymoses/abrasions suggest significant mechanism to cause injury.
- Restricted vertical gaze occurs with orbital floor fractures that result in entrapment of the inferior rectus muscle, tethering the eye.
- Decreased sensation or numbness in V2 distribution can occur due to neurapraxia because V2 runs through the orbital floor/maxillary sinus roof.
- Coronal CT with 2-mm slices enables visualization of soft tissue densities, such as prolapsed orbital fat, extraocular muscle, and hematoma.
- Plain radiographs can have a false-negative rate of up to 50%.
- MRI does not visualize bone well.

**Treatment:** Surgical treatment is usually necessary only when there is actual interruption or herniation of orbital tissue. Diplopia may be transient after these injuries, and thus, surgical intervention should be deferred until a significant portion of

the contusion injury has resolved. Conversely, in cases with little contusion injury and obvious interruption or herniation of orbital tissue, there is no need to delay definitive repair.

**Prognosis:** Varies depending on degree of injury; prognosis is best when orbital tissue damage is minimal. Prolonged tissue entrapment and inflammation can result in fibrosis and contractures, which can lead to permanent functional disabilities.

### Hyphema

**Description:** Refers to the presence of blood in the anterior chamber that can result from a tear in peripheral iris vessels. Anteroposterior compression of the globe leads to expansion of the globe with damage to the blood vessels of the iris or ciliary body. There is a high frequency of concomitant injury, including blunt or penetrating trauma. Projectiles that strike the exposed portion of the eye are a common cause of hyphema. Baseball and softball players are commonly affected. Hyphema frequently occurs in microscopic quantities and consequently, can be easily overlooked.

**Signs and symptoms:** Because many hyphemas are small, visual acuity may be unaffected. There may be a mild injection of globe with moderate transient discomfort and photophobia. In addition to hyphema, other injuries associated with contusion injuries may also be found, including papillary paralysis, pupillary contour irregularities, and tearing of the uveal tract.

**Examination:** Slit lamp examination is necessary to identify any turbid microscopic hyphema before it has had time to settle out in the anterior chamber; however, careful penlight examination can identify hyphema in acute settings. IOP should be measured. Of hyphema patients, 25% have other ocular injuries, including vitreous or retinal hemorrhage or dislocated lens.

**Treatment:**

- Immediate referral to an ophthalmologist
- Manage as if a ruptured globe were present
- Shielding of the eye should be accomplished with a protective metal shield for 2 weeks.
- Physical activity should be severely restricted with bed rest for 4 days followed by light activity for 2 weeks. Topical cycloplegics (atropine sulfate twice daily for 2 weeks), and topical corticosteroids should be given. Salicylates and NSAIDs should be restricted. Acetaminophen can be given for pain relief. Topical beta blockers, such as timolol, are often used if there is associated increased IOP. In nontraumatic cases, athletes should be assessed for coagulopathy and treated accordingly.
- Examine for 3 consecutive days to monitor for re-bleeding or IOP elevation. Recurrent bleeding is most frequent during the initial 5 days after injury and occurs in 4%–6% of cases. Increased IOP may lead to glaucoma. Patients with sickle cell trait or disease are more susceptible to this complication. **Increased IOPs may accompany hyphemas of any size.**
- Corneal blood staining is possible.
- Sickle cell status should be established in all African-American patients or Hispanic patients who have an IOP >21 mmHg.
- Patients should be re-evaluated 2 weeks after the injury even if there are no other problems.

**Prognosis:** Excellent, particularly if blood clears rapidly, and there is no recurrent bleeding or other injuries. Usual duration of an uncomplicated hyphema is 5–6 days.

- **Recurrent bleeding may lead to permanent loss of visual acuity.**
- Most microhyphemas can be treated on an outpatient basis, unless re-bleeding or IOP is uncontrolled. If the hyphema occupies greater than one third of the anterior chamber, IOP is elevated beyond 30 mmHg, or both, hospitalization is recommended.

### Vitreous Hemorrhage

**Description:** When vitreous hemorrhage is caused by trauma, significant force is involved and additional injury to the eye is generally present. It can result from blunt trauma, shaking, or proliferative retinopathy. Blood obscures the light path through the vitreous cavity of the eye and reduces visual acuity.

**Signs and symptoms:** Assessing and documenting the patient's vision before symptoms of hemorrhage are crucial. Patients may report seeing "floaters, visual haze, smoke, shadows, or cobwebs." More severe hemorrhages may result in the sensation of dark streaks that break up into numerous, minute black spots, or vision reduction to only light perception. Isolated vitreous bleeding is not associated with symptoms such as pain or discomfort.

**Examination:** Measure visual acuity and pupillary response; on fundoscopic examination, fundus detail is often blurred or disappears entirely and may be seen as a "black reflex." On slit lamp examination, fresh blood is identified readily by adjusting the slit beam to a tangential position and viewing the anterior vitreous directly behind the lens.

**Treatment:** Generally conservative, although severe cases may require surgical removal of blood and vitreous. Such procedures are often performed at the time of repair of associated ocular injuries.

**Prognosis:** Guarded

### Retinal Hemorrhages and Detachment

**Description:** Can result from direct trauma to eye by transmission of force to the retinal surface; this produces immediate loss of visual function within the detached segment. In the absence of treatment, the entire retina eventually becomes involved, and total retinal detachment develops. Can be caused by retinal instability seen with violent exercise performed in conditions of decreased oxygen saturation or elevated venous pressure from Valsalva maneuvers; such findings have been documented in activities such as mountain climbing and weightlifting. **Retinal detachment** produces immediate loss of visual function within the detached segment. Any form of blunt or perforating trauma can produce retinal detachment. Indirect trauma, such as severe head injury, myopia, and vitreous traction, are risk factors.

**Signs and symptoms:** Assessing and documenting the patient's vision before hemorrhagic symptoms develop is crucial. Multiple asymptomatic areas of retinal hemorrhage and edema are not uncommon, particularly if affected areas are confined to the peripheral retina. Involvement of the macula results in decreased visual acuity or distortion of visual perception. Patients often report positive scotoma at the edge of the visual field or seeing "floaters, visual haze, smoke, shadows, or cobwebs." As detachment progresses, the patient may describe visualization of "lightning flashes" or "flying sparks." An enlarging scotoma may be seen as a waving black curtain encroaching on central vision. Severe hemorrhages may result in the sensation of dark streaks that break up into numerous, minute black spots, or vision reduction to only light perception.

**Examination:** Visual field and acuity measurement; fundoscopic examination reveals both flame-shaped hemorrhages and round-blot hemorrhages or may show a "black reflex." On slit lamp examination, fresh blood is readily identified by adjusting the slit beam to a tangential position and viewing the anterior vitreous directly behind the lens. As the retina becomes more elevated, it is necessary to add additional convex or plus lenses to the fundoscopic viewing port to maintain sharp focus on internal retinal surface. It is necessary to dilate the pupil widely to visualize a detachment during its early stages because detachments begin in the far periphery.

**Treatment:** Symptomatic

Surgical intervention is almost invariably necessary after actual separation of the retina has occurred. If surgery is not indicated,

then close observation for 1–2 weeks allows time for spontaneous clearing of mild hemorrhage, but it may take several months for complete vision to return. Upright positioning for sleep may enhance settling of the hemorrhage. Emergent consultation is required if hemorrhage results from trauma.

**Prognosis:** Varies depending on location, extent, and severity of involvement; a chance for recovery of good central vision is generally poor if retina becomes detached in the area of macula

### Dislocated Lens

**Description:** Results from tearing of lens zonules and loss of support in its normal position; may result in subluxation or movement of the lens away from the site of injury, causing it to decenter slightly but remain in a relatively normal position. More extensive damage may entirely displace the lens, causing it to fall either into the anterior or posterior chamber.

**Signs and symptoms:** Visual acuity is affected by even the slightest shift in position of the crystalline lens:

- Lens decentering causes irregular astigmatism with poor near vision secondary to loss of accommodation power and decreased distance visual acuity.
- Complete dislocation results in aphakia.
- Shifts of lens position, although slight, can also cause loss of iris stability and resulting tremulousness with slight ocular movements or vibrations (iridodonesis).
- Monocular diplopia

**Examination:**

- Visual acuity may be initially reduced but can frequently be corrected by change in refraction.
- Slit lamp examination may reveal iris undulations (iridodonesis) at the pupillary margin after rapid eye movements.
- Strabismus is not uncommon (secondary to amblyopia).
- Pupillary dilatation can aid in assessment of lens position and evaluation of the retina.
- Enophthalmos with facial myopathic appearance may be seen in patients with Marfan syndrome.
- IOP should be assessed because lens dislocation can result in secondary glaucoma.
- If Marfan syndrome, homocystinuria, or other collagen vascular diseases are suspected, cardiac workup should be performed including a cardiac echocardiogram. Check serum and urine levels of homocysteine or methionine for homocystinuria. Genetic-associated lens dislocation is usually bilateral.

**Treatment:** Variable and may necessitate surgical removal of the lens; immediate referral should be made to an ophthalmologist if this injury is suspected

**Prognosis:** Varies with extent of injury

### Chamber Angle Recession

**Description:** Generally results from blunt trauma to the globe, causing a sudden increase in pressure within the anterior chamber, which is transmitted to the lens–iris diaphragm, propelling it backward. Dynamics of this injury are similar to that of lens dislocation; however, in angle recession, lens position is usually normal. In few cases, force of injury is sufficient to produce associated injury to the trabecular meshwork, which may eventually lead to glaucoma.

**Signs and symptoms:** In cases wherein glaucoma develops, the onset is almost invariably delayed from the time of the injury and progresses slowly. As with other forms of chronic glaucoma, visual loss is insidious, beginning with the peripheral areas of the visual field. Angle recession glaucoma should always be suspected in cases of unilateral chronic glaucoma.

**Examination:** Usual methods for evaluating chronic glaucoma, including evaluation of IOP and visual field examination, with special attention to classic field defects commonly seen in chronic glaucoma

**BOX 47.2 MOST COMMON CAUSES OF RED EYE**

- Conjunctivitis
- Episcleritis and scleritis
- Keratitis and corneal ulcer
- Iritis and intraocular infections (endophthalmitis)
- Glaucoma (acute and chronic)
- Dry eye
- Subconjunctival hematoma (hyposphagma)
- Corneal and conjunctival foreign body
- Corneal abrasion
- Corneal flash burn
- Chemical burns
- Blunt or penetrating trauma to the eye
- Allergic reaction
- Blepharitis

**Treatment:** Angle recession glaucoma is frequently not responsive to medical therapy and may require surgical treatment.

**Prognosis:** Varies with extent of pressure elevation, length of time it has been present, and responsiveness to treatment

### Red Eye

**Description:** The aim of management should be differentiation of the symptom of red eye and assessment of the underlying disease. In general, redness of the eye can be caused by hyperemia with dilation of the conjunctival, episcleral, or scleral vessels (caused by trauma, chemical burns, or immunologic reactions); inflammatory reactions from infections (bacterial, viral, or fungal); or chronic reactions of the external eye from systemic causes (Box 47.2).

**Signs and symptoms:** Important aspects of history:

- Association with pain
- Pain, itching, and visual decrease or loss
- Mucopurulent discharge, watering, blepharospasm (lagophthalmus), or systemic (fever, nausea) findings
- Foreign body sensation
- Decreased visual acuity

**Examination:** Should include the anatomic location of redness (eyelids, conjunctiva, cornea, sclera and episclera, or intraocular); measurement of IOP (to rule out acute closed angle glaucoma) and visual acuity (evaluation if decreased, needs urgent referral to ophthalmologist). Measure pupil size and response to light. Fluorescein testing may be necessary.

**Treatment and prognosis:** Depends on the cause of symptoms; athletes should not return to play until the cause of symptoms is ascertained

## INJURY PREVENTION

### Risk Factors to Be Considered

- Physical development and skill level
  - Beginners may have increased risk because of lack of necessary refinement of skill of sport.
  - Advanced players, particularly in certain high-risk sports, may play more aggressively and be at a higher risk of eye injury.
- **Existing visual impairment** increases the risk of injury.
- **Preexisting eye disease** may present an increased risk to athletes in all risk groups.
  - Conditions that may lead to serious eye disorders or get worse after even minor trauma to the eye include retinal detachment, retinal degeneration, severe myopia, thin sclera, and prior ocular surgery.
  - Systemic disease with eye involvement and previous serious ocular injuries may be risk factors as well.

### Types of Eye Protectors

**Total head protector:** A combination of helmet and face shield designed to protect eyes, teeth, jaw, and larynx and transfer forces to skull; designed for use in high-risk sports that require total head protection: football, hockey, and lacrosse

**Full face protector:** Designed for use in conjunction with eye protectors for high-risk sports that do not require protection for brain: fencing and for certain positions in baseball and softball

**Helmet with separate eye protectors:** For use in sports with low risk of injuries to lower face and neck: cycling, snowmobiling, skiing, automobile racing, and bobsled racing

**Helmet only:** Helmets are designed to protect the brain only. They afford little protection to the face or eyes and are used in boxing and cycling.

**Sports eye protectors:** Used only to protect eyes and are recommended in all high-risk sports for which additional head and face protection is impractical: all racquet sports, baseball, soccer, basketball, and softball (see Table 47.4)

**"Sports" sunglasses:** Most are inadequate for both impact resistance and ultraviolet radiation blockage. Adequate eyewear of this type should:

- Contain a manufacturer's statement recommending intended sport
- Block light from sides and below
- Protect from glare (transmit 30% of light)
- Be lightweight, cosmetically acceptable, and aerodynamically designed to prevent drying in wind

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# MAXILLOFACIAL INJURIES

# 48

Scott A. Escher • Michael K. Case • Thomas S. MacKenzie

## GENERAL PRINCIPLES

### Epidemiology

- 3%–29% of facial injuries are a result of sporting activities
- 60%–90% of facial injuries in sports occur in males aged 10–29 years
- Approximately 75% of facial fractures involve the zygoma, mandible, or nose.
- The most commonly injured teeth are the maxillary central incisors, followed by the lateral incisors and the mandibular incisors.

### Initiation of Care of the Head-Injured Athlete

#### Airway Injury or Compromise

- Follow the ABCs (airway, breathing, and circulation) of basic life support.
- May need to secure airway before making any other assessment
- If the neck is injured, use the jaw thrust maneuver.
- May be difficult to maintain airway with unstable mandibular fracture and certain soft tissue injuries
- Can use oral airway or endotracheal tube as indicated in unconscious patients. A nasal trumpet works well in an awake patient without a midface fracture.
- Cricothyrotomy may be the only option in emergency.

#### Cervical Spine Injury and Concussion (See Chapter 46, Neck Injuries and Chapter 45, Head Injuries)

- The spine must be stabilized if there is any doubt regarding injury or if the athlete shows altered mental status.
- With facial injuries, the physician must check for associated cervical spine injuries or concussion.

#### History

- Check for history of abnormalities such as crooked nose, missing teeth, or anisocoria.

#### Physical Examination

INSPECTION

- Observe for facial asymmetry, widening of the midface, ocular asymmetry, and malocclusion from multiple angles.
- Early examination is optimal before the swelling causes any asymmetry.
- Observe for malocclusion.
- Bleeding or bruising sites may be indicative of other possible injuries.

PALPATION

- Systematically palpate bony structures, including the maxilla and mandible, bimanually with gloved fingers in the oral cavity.
- Conduct sensory examination for possible nerve injuries. Three branches of the trigeminal nerve supply the face: the ophthalmic nerve innervates the forehead, maxillary nerve innervates the cheek and midface, and mandibular nerve innervates the jaw (Fig. 48.1).

RANGE OF MOTION

- Assess mandibular motion. Pain with opening or closing can indicate fracture.
- Ocular motions can be altered in certain facial fractures, indicating ocular muscle entrapment or nerve injury, which may require more immediate treatment.

#### Imaging Studies

CONVENTIONAL RADIOGRAPHY

- Conventional radiography is rarely used for facial bone evaluation because imaging adds little to the clinical examination. However, if needed, most common radiographs:
  - For nose: Right and left lateral, superoinferior axial occlusal, and Waters' views
  - For facial bones: Submentovertex, Waters', right and left lateral obliques, Towne's, and posteroanterior views
- Panoramic radiographs often used for dental root injuries

CROSS-SECTIONAL IMAGING

- Computed tomography (CT) and magnetic resonance imaging (MRI) are much superior to conventional radiographs in identifying both normal and abnormal anatomy, particularly in the pediatric population. These provide more information regarding severity and displacement in case of an injury, which aids surgical planning.
- Facial bone CT offers better anatomic details, particularly bony anatomy, and is the study of choice over plain radiographs in trauma. Three-dimensional CT reconstructions can be a valuable tool in diagnosis.
- MRI is superior in soft tissue imaging, does not utilize ionizing radiation, and displays vascular anatomy without using contrast, but it is rarely used in trauma.

## COMMON INJURIES

### Soft Tissue Injuries

#### Lacerations

**Examination:** Assess for both sensory and motor nerve injuries as well as underlying bone damage.

**Treatment:** Cleanse with sterile water or irrigating solution. Administer local anesthesia with lidocaine and epinephrine to help with hemostasis. Certain experts advocate not using epinephrine on ear or nose; may need layered closure of deep wounds with a dissolving suture to re-approximate subcuticular tissues. Skin closure can be with a 5-0 or 6-0 suture or tissue adhesive if skin edges closely aligned; appropriate alignment of vermilion border of lip important for cosmetic purposes. If the laceration is minor and bleeding can be controlled and can be covered with occlusive dressing, the athlete can return to competition.

### Ear Injuries

#### Auricular Hematoma ("Cauliflower Ear")

**Mechanism of injury:** Caused by shear forces: e.g., in wrestlers without head protection; this results in blood and/or serum

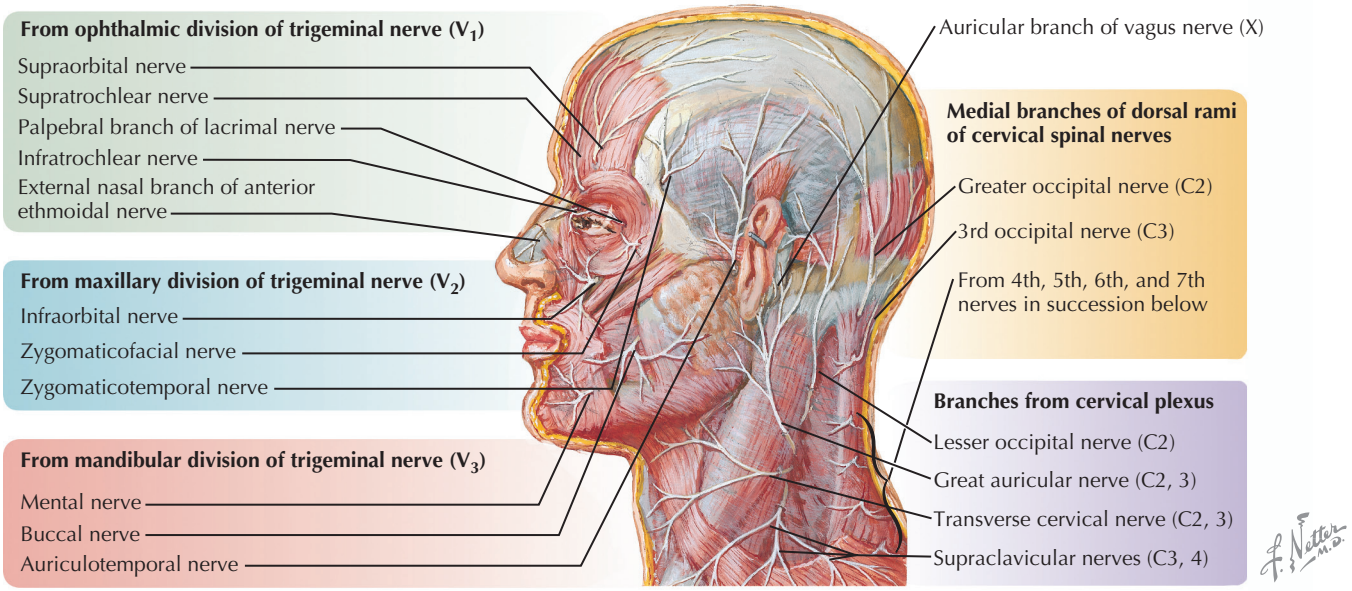



**Figure 48.1.** Cutaneous nerves of head and neck.

accumulation between the ear cartilage and the perichondrium or between sheared layers of perichondrium.

**Examination:** Swelling or fluctuant areas in the cartilaginous area, usually in outer or lateral regions (Fig. 48.2)

**Treatment:** May usually continue participation in the event wherein the injury occurs; initially treated by aspiration of hematoma or incision and drainage after anesthetizing:

- Permanent disfigurement can occur if not appropriately treated.
- Field-block anesthesia by infiltrating the posterior sulcus as well as the skin anterior to the helix and tragus with 1% or 2% lidocaine without epinephrine; care should be taken to avoid injecting near the facial nerve.
- Hematoma will reoccur if not bolstered to hold the packing onto the area with slight pressure. Bolster can be a dental roll on both sides of the pinna held in place by a 3-0 or 4-0 monofilament nonabsorbable suture. Alternatively, a button or silicone splint can be placed on either side of the ear. To prevent further trauma, the wrestler must use headgear and leave the bolster in place for 7 days (see Fig. 48.2).
- Always use antibiotics with *Staphylococcus* coverage with a bolster in place.
- If infection occurs, bolster must be removed and the area must be incised, drained, and treated with an antibiotic that covers *Staphylococcus* and *Pseudomonas*.

**Prevention:** Appropriately fitted headgear

### Ear Laceration (Including Aforementioned Lacerations)

- Sew the ear in layers with cartilage, perichondrium, and finally skin. On the lateral portion of ear, all three layers may have to be sutured together.
- Attempt to minimize sutures in cartilage.
- Use an undyed absorbable 6-0 suture for cartilage and perichondrium.

### Otitis Externa ("Swimmer's Ear")

**Mechanism of injury:** Disruption of the thin ear canal skin. Water-sport athletes are at a particularly high risk; usually caused by *Pseudomonas aeruginosa* or *Staphylococcus aureus*

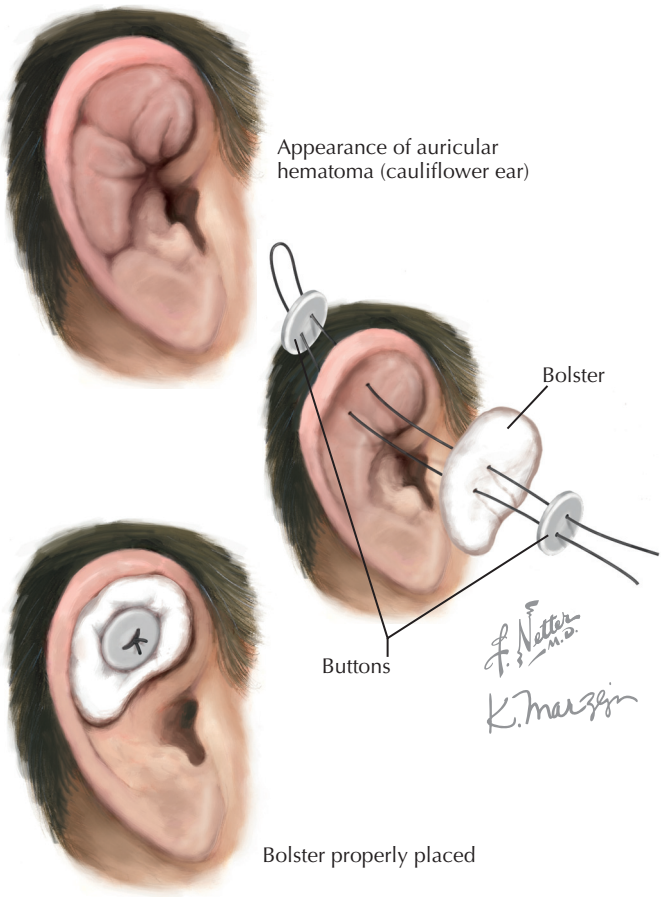



**Figure 48.2.** Cauliflower ear.

**Examination:**
- Pain and discomfort with motion of pinna
- Swollen, inflamed, and erythematous external auditory canal
- Purulent discharge

**Treatment:**
- Suction ear to remove debris.
- Antibiotics: corticosteroid-combination drops; quinolones are the best choice but can be costly
- May need a wick to allow antibiotics to penetrate to innermost portion of the ear canal if marked swelling present; leave in place for 3–5 days
- Consider treating significant infections with an oral antibiotic that has *Pseudomonas* coverage.
- Stay out of water until asymptomatic.

**Prevention:** Dry ears after swimming; use of a hair dryer to ear can help reduce moisture. A combination of alcohol and vinegar or commercially available alcohol-based products such as Swim Ear® can help lessen moisture and may reduce the incidence of otitis externa.

### Tympanic Membrane Perforation

**Mechanism of injury:** Can occur after skydiving or scuba diving with pressure changes; a blow to the ear or a fall onto the ear while water skiing

**Examination:**
- Hearing loss and serous or bloody drainage from ear
- Visible perforation in tympanic membrane
- Associated vertigo can signify ossicular disruption

**Treatment:** Most (85%–90%) will heal without treatment; if it does not heal within 2–3 weeks, refer to an otolaryngologist. In water sports, use earplugs to keep water out of ear canals until perforation heals. Avoid sports with large changes in pressure, such as platform diving, until perforation heals. If injury occurred in river or lake, consider antibiotic eardrops. For suspected ossicular disruption, the athlete must be immediately referred to an otolaryngologist.

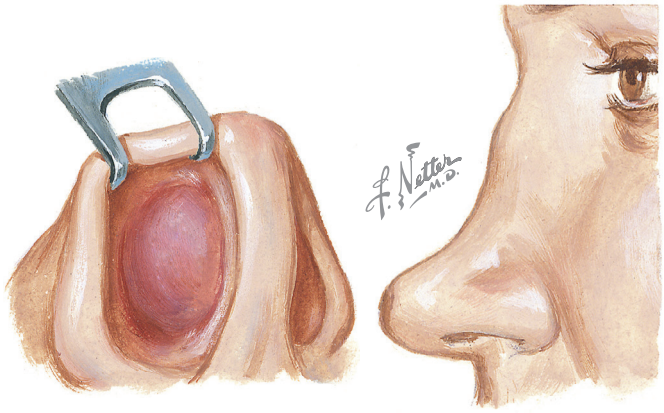
## Dental Injuries

**History:**
- Spontaneous pain may indicate pulp exposure.
- Tender teeth with chewing may indicate injury to the periodontal ligament.
- Teeth sensitivity to extremes of temperature may indicate pulp exposure or inflammation.
- Changes in bite patterns (e.g., malocclusion) suggest facial fracture or dental subluxation.

**Examination:** Examine teeth for fractures and laxity and soft tissues for associated lacerations and bruising. Panoramic radiographs or CT should be performed with dental fractures or luxation (loosening). Assess for root or bony fractures or in case of younger athletes, for permanent tooth bud displacement.

**Treatment:**
- ***Primary Teeth:*** Primary goal is to prevent injury to permanent teeth.
  - Injuries needing urgent dental evaluation:
    - Displaced or significantly loose primary teeth
    - Do not replace avulsed or extruded (completely dislocated) teeth because this may injure the underlying permanent teeth.
    - Crown fractures involving the pulp need prompt referral to prevent infection.
  - Injuries needing dental evaluation within a few days:
    - Mildly subluxed teeth, which appear in normal position but have pain with chewing, should be treated with a soft diet
    - Crown fractures not involving the pulp
    - Root fractures
- ***Permanent Teeth:*** Subluxed permanent teeth that are crooked should be examined by a dentist as soon as possible, but no on-field reduction is needed. Fractures involving the pulp can be painful. Fractures of teeth can be treated hours after the injury.
- Avulsed or displaced teeth: Survival of the delicate periodontal ligament cells on the root of a tooth is necessary for tooth survival. Little chance for dental survival after 1 hour out of the socket. Needs immediate dental referral for emergent reimplantation. Field care:
  - Handle only by crown
  - May rinse with saline or tap water, but do not rub or clean root
  - Athlete can keep tooth in place with finger pressure or by biting on a gauze pad
  - Alternatively transport by storing in Hank balanced salt solution, (e.g., Save-A-Tooth®). This will maintain the viability of the periodontal ligament cells longer, increasing the likelihood of successful reimplantation. Cold milk is a good alternative to this. Saline solution is another alternative. Water is not helpful in extending the viability of the periodontal ligament cells.
  - Tetanus prophylaxis should be given if tooth is contaminated with dirt and last tetanus shot was more than 5 years previous.

**Prevention:** Mouth guards can prevent dental injury. Basketball players are seven times more likely to have an orofacial injury when not wearing a mouth guard.




Examination for septal hematoma

Cartilaginous deformity secondary to untreated septal hematoma

**Figure 48.3.** Nasal fracture.

## Nasal Fracture

**Mechanism of injury:** Trauma to nose. Nasal fractures constitute 50% of facial fractures in adults. Injury to bone–cartilage interface can be similar to nasal bone fracture, and diagnosis is made clinically.

**Examination:** Signs and symptoms include epistaxis, nasal asymmetry, crepitus on palpation, swelling, and nasal airway obstruction. Assess for septal hematoma (Fig. 48.3), submucosal swelling on one or both sides of septum. Septal hematoma causes injury to blood supply to nasal cartilage.

**Imaging:** Although radiographs may show fracture, they are not necessary. Radiographs may be normal if injury is at the bone cartilaginous interface. Certain experts advocate prereduction photograph for medicolegal purposes.

**Treatment:**
- Control hemorrhage with compression or nasal packing; most bleeding will stop with time. Topical vasoconstriction or clotting agents, such as Arista™ may be used.
- It is imperative to treat septal hematoma within 24–48 hours to prevent permanent damage to underlying nasal cartilage (i.e., saddle nose deformity) (see Fig. 48.3). Use antibiotic with *Staphylococcus* coverage if packing or hematoma is present.
- Reduce displaced fractures immediately or wait 5–7 days for swelling to diminish; waiting longer can make reduction more difficult.
- Control fracture with splinting; complex fractures may require operative reduction.

**Return to play:**
- Restrict aerobic activity for a few days
- Facial protection can be purchased off the shelf or individually fabricated. Athletes can return to contact/collision sports with the facemask in approximately 4 weeks. Many athletes return earlier than 4 weeks with the understanding that they may refracture and require further treatment.

## Epistaxis

**Mechanism of injury:** Usually occurs spontaneously at the Kiesselbach's plexus on the nasal septum in the area of thin nasal mucosa overlying blood vessels. Trauma is the second most common cause of epistaxis. Posterior bleeding is less common and can be difficult to control.

**Examination:** If bleeding site is difficult to find, a nasal telescope can be used.

**Treatment:** Most nosebleeds resolve spontaneously in a few minutes; apply pressure to the upper lip over the superior labial artery. Pinch the nostrils against the nasal septum, which may constrict blood flow from the anterior ethmoidal and sphenopalatine arteries (Fig. 48.4). If the bleeding site is at Kiesselbach's plexus, a folded dental roll temporarily inserted into the naris will apply pressure over the bleeding site and may allow the athlete to finish the contest. If time allows, consider vasoconstriction of the area with topical oxymetazoline, cocaine, and epinephrine 1:100,000, or a combination of lidocaine, epinephrine, and tetracaine. Subsequently, chemically cauterize the area with a silver nitrate stick or use electrocautery. Anterior nasal bleeding that does not stop with these maneuvers can be treated with nasal packing. Posterior bleeds and those anterior bleeds not controlled with nasal packing need an urgent visit with an otolaryngologist. Petrolatum jelly applied nightly to the nasal septum can prevent drying and cracking of the nasal mucosa and may help prevent epistaxis.

## Mandibular Fractures

**Mechanism of injury:** Usually direct trauma to jaw

**Examination:** Signs and symptoms include pain with palpation, malocclusion; loose teeth, loose segments of jaw; laceration of overlying oral mucosa; sublingual ecchymosis; inability to open or close jaw; may have numbness if injury involves mandibular nerve (Fig. 48.5).

**Imaging:** Although a CT scan shows bone anatomy better, a panoramic radiograph shows dental root injury better.

**Treatment:** If the athlete can breathe through the nose, initially immobilize with an ace wrap until followed up with imaging. Reduction followed by bony immobilization with maxillomandibular fixation (wiring of teeth) or bone plating. With wiring, the jaw is immobilized for 4–6 weeks; with bone plating, can move jaw immediately but chewing is limited for approximately 4 weeks. With bone plating, airway issues, oral hygiene, and diet are less problematic. Infection is relatively common and antibiotics should be used prophylactically. Pediatric patients, due to growth potential, can be treated with resorbable bone plates.

**Rehabilitation:** Therapy after jaw wiring may improve jaw opening.

**Return to play:** Return to noncontact sports in 4 weeks; contact sports in 2–3 months.

## Maxillary Fractures

**Mechanism of injury:** Usually secondary to direct trauma to midface; classified using Le Fort scheme (Fig. 48.6). Injuries

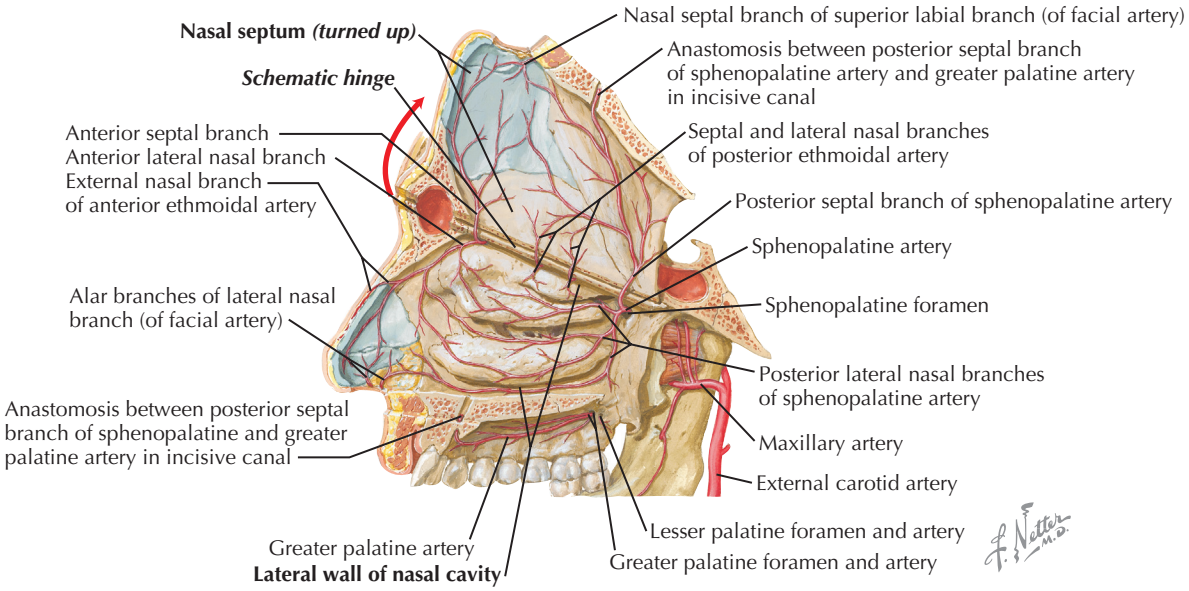



**Figure 48.4.** Arteries of nasal cavity: nasal septum turned up.

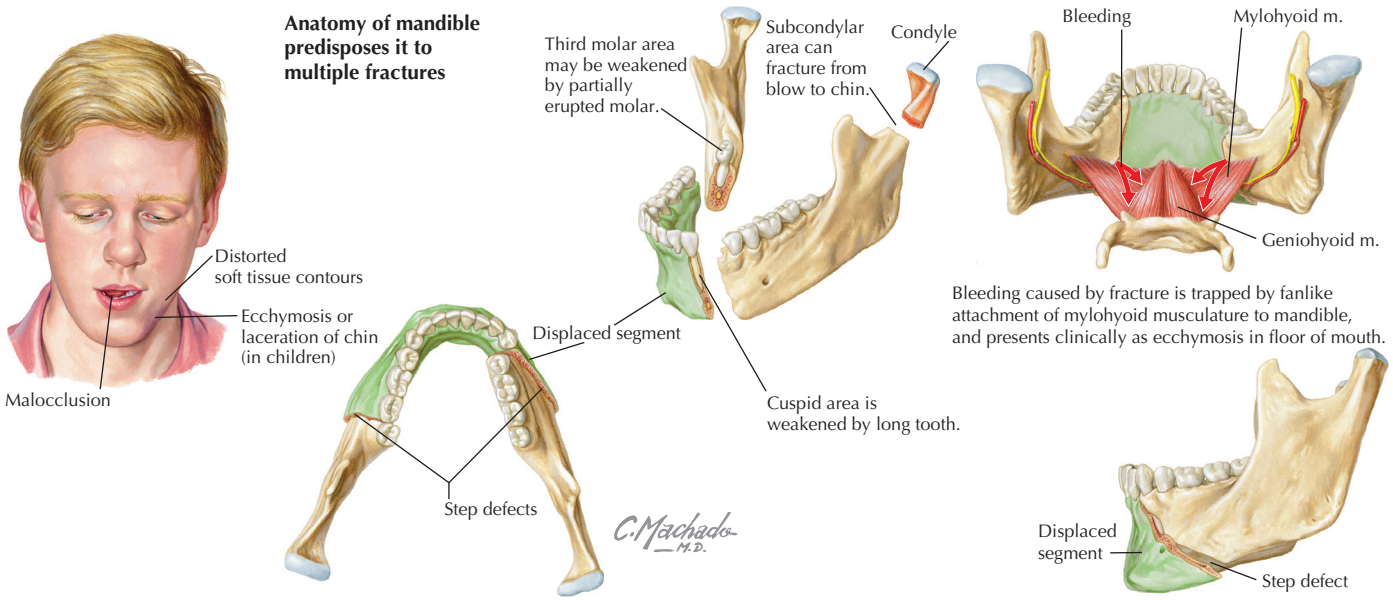



**Figure 48.5.** Mandibular fractures.

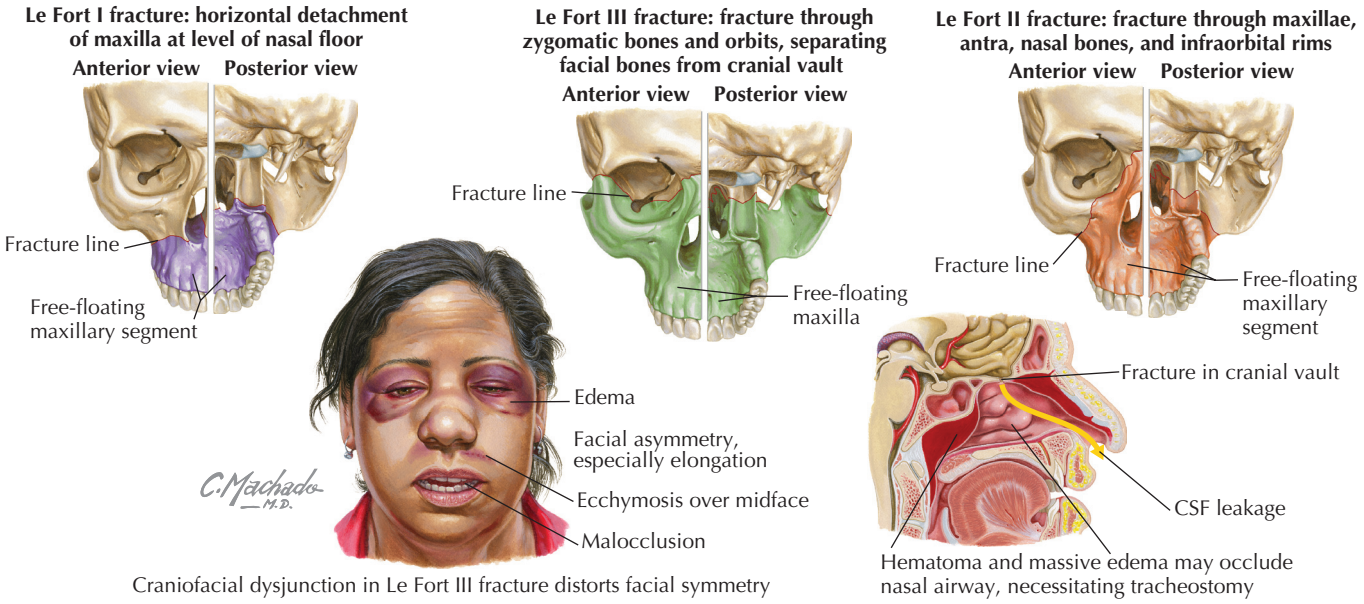



**Figure 48.6.** Midface fractures: Le Fort.

may include a combination of classic Le Fort fracture patterns; may be associated with brain, cervical, or ocular injuries

**Examination:** Signs and symptoms include asymmetry or altered contour of face, flattening of the midface; midface is mobile when applying a force directed anteriorly to the anterior maxillary alveolus; epistaxis; malocclusion and loose dentition; and diplopia or globe malposition. Documentation of the function of all cranial nerves should be done. Injury to the infraorbital nerve is common in midface fractures, resulting in altered sensation of cheek and upper lip.

**Radiology:** Facial bone CT scan for evaluation for maxillary fractures. Waters' view plain film can be helpful if no CT scan available.

**Treatment:** Secure airway, if athlete is able, a forward sitting position will allow blood to drain outside the body and may facilitate mouth breathing. Nasotracheal airway contraindicated without fiber-optic placement in these injuries. Most surgeons advocate open reduction with internal fixation with titanium miniplates and microplates as soon as medically prudent based on other injuries. Le Fort I fractures can be treated with intermaxillary fixation (wiring). Pediatric fractures can develop

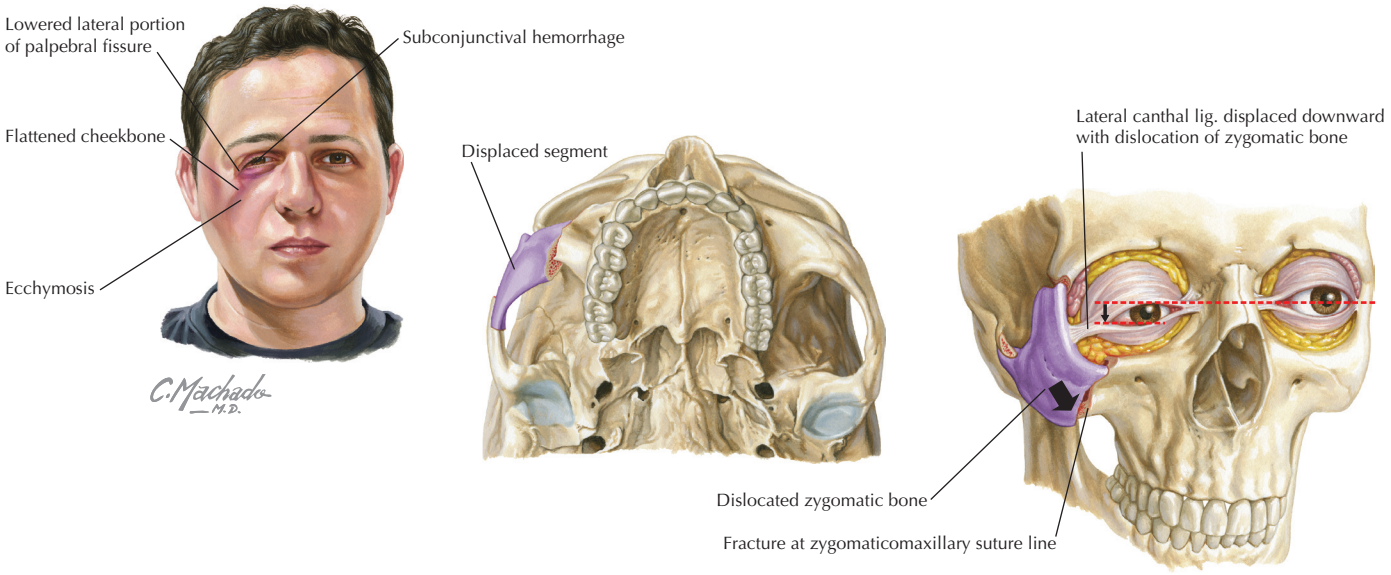



**Figure 48.7.** Zygomatic fractures.

further deformity with growth, and parents should be made aware of this possibility. Resorbable plates can be used in certain cases in children.

## Zygoma Fractures

**Mechanism of injury:** Usually from direct force to cheek. A zygomatic arch fracture can occur in isolation or in association with a zygomatic complex fracture (also called a *tripod fracture*) that includes the lateral orbital wall, the orbital rim and possibly the orbital floor, the zygoma and the zygomatic buttress of the maxilla.

**Examination:** Signs and symptoms include:

- Facial asymmetry
  - Flattening of malar eminence of the involved side due to the masseter muscle pulling the malar eminence inferiorly. This may not be apparent until swelling resolves.
  - Enophthalmos (sinking of the eyeball into the orbital cavity) if orbital floor is sufficiently damaged.
- Diplopia if inferior rectus or inferior oblique muscles are entrapped indicating orbital floor fracture.
- Pain with mastication due to zygomatic arch depression and the action of the temporalis muscle
- Numbness of affected cheek
- See Fig. 48.7 for hallmarks of zygomatic fractures.

**Imaging:** Radiographs—Caldwell, Waters', and submentovertex views will identify most fractures except orbital fractures. CT scan is preferred with axial and coronal imaging usually sufficient for surgeon to plan treatment.

**Treatment:** Nonsurgically if <2 mm of displacement; however, cosmetic deformity can become apparent when facial swelling diminishes. Surgical fixation after reduction is often performed 3–7 days after injury. Athletes with nonsurgically treated fractures should use a soft diet for 1–2 weeks to minimize tension on fracture from masseter muscle; those with surgically treated fractures can use soft diet for 1 week.

**Return to play:** Return to noncontact sports in 3–4 weeks and contact sports in 6–8 weeks

## Frontal Sinus Fracture

**Mechanism of injury:** Direct trauma to sinus; uncommon fracture; more common in adults because pediatric population may not have developed sinuses; anterior wall of sinus is strongest portion of sinus

**Examination:** Signs and symptoms include frontal headache, epistaxis, forehead numbness from supraorbital nerve injury, anosmia (alteration in sense of smell) if associated fracture of anterior fossa floor, cerebrospinal fluid leakage if posterior wall of frontal sinus fractured, and depression of frontal area of skull can be seen and palpated (swelling may make this difficult).

**Imaging:** CT imaging of face and head

**Treatment:** Referral to a maxillofacial surgeon is indicated. Nondisplaced fractures limited to the anterior wall of the frontal sinus may be nonsurgically treated. Certain frontal sinus fractures must be surgically explored to prevent complications such as deformity, damage to the frontonasal duct, and subsequent mucocele formation. Posterior wall fractures may result in damage to the dura and possible infection of the meninges and brain.

## Orbital and Ophthalmic Injuries

See Chapter 47, Eye Injuries.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# SHOULDER INJURIES

# 49

Charles T. Crellin • Kevin M. Honig • Eric C. McCarty • Jonathan T. Bravman

## HISTORY

- A careful history will help establish the diagnosis and formulate a treatment plan.
- Important factors include the chief complaint, mechanism of injury, hand dominance, what sport the athlete plays, and prior treatments.
- Common complaints are "pain with overhead activities," "loss of range of motion," "pain at night when I lie on that side," and "a feeling of the shoulder coming out of the joint."

## PHYSICAL EXAMINATION

Evaluation of shoulder pathology should include an examination of the cervical spine to rule out referred pain.

### Inspection

- It is important to visualize the entire shoulder during an examination and compare it with the unaffected side (including the scapula).
- Check for muscle atrophy, which can indicate neurologic dysfunction or chronic injury.
- Examine for scapular dyskinesia or winging.
- Note bony prominence that could represent acromioclavicular (AC) separation, clavicle fracture, sternoclavicular (SC) subluxation, or degenerative disease.

### Palpation

- Palpate bony landmarks for tenderness or crepitus—AC joint, clavicle, SC joint, greater tuberosity, and coracoid.
- Extension and internal rotation of the arm deliver a greater tuberosity from under the acromion.

### Motion

- It is important to compare active and passive range of motion to the unaffected side. Forward flexion, abduction, and external rotation are measured in degrees from neutral rotation. Internal rotation is measured in relation to the spinal level that can be reached posteriorly (Fig. 49.1A).
- Does motion cause pain or produce a feeling of instability?
- The normal ratio of glenohumeral (GH) to scapulothoracic motion is 2 : 1 (Fig. 49.1B).

### Manual Muscle Testing

**Supraspinatus:** (Jobe test) Resistance is applied with the patient's arm abducted 90 degrees, forward flexed 30 degrees, and internally rotated (thumb pointing down).

**Infraspinatus/teres minor:** Resistance to external rotation with the arm adducted and the elbow preferentially flexed 90 degrees assesses the infraspinatus. Resistance to external rotation with the arm abducted and the elbow flexed 90 degrees assesses both the infraspinatus and teres minor (Fig. 49.1).

**Subscapularis:** Resistance to internal rotation with the arm adducted and the elbow flexed 90 degrees (Fig. 49.1F)

## SPECIFIC TESTS

### Impingement

Positive tests produce pain at the anterior or lateral aspect of the shoulder.

**Hawkins' test:** The arm is passively forward flexed to 90 degrees and then forcibly internally rotated (Fig. 49.2A).

**Neer's sign:** The patient's arm, with the forearm pronated, is passively forward flexed while the scapula is stabilized (Fig. 49.2B).

**Painful arc:** The patient actively elevates the arm in the scapular plane and then lowers it in the same plane. Pain during range of motion between 60 and 120 degrees is positive.

**Coracoid impingement sign:** The patient's arm is passively placed in a position of forward flexion, adduction, and internal rotation with pain produced directly over the coracoid.

### Rotator Cuff Tear

**Jobe test:** Isolates the **supraspinatus** (see earlier discussion); pain can also be indicative of subacromial impingement

**Champagne toast:** The arm is abducted against resistance at 30 degrees of abduction, 30 degrees of forward flexion, and mild external rotation; this test isolates the **supraspinatus.**

**Drop arm sign:** The patient fully elevates the arm in the plane of the scapula and then tries to lower it slowly. Sudden dropping of the arm or pain while doing so suggests a supraspinatus tear.

**External rotation lag sign:** With the arm adducted and the elbow flexed 90 degrees, the arm is passively brought to maximal external rotation. Inability of the patient to actively maintain the arm in the externally rotated position indicates a **massive tear** involving the **infraspinatus** (Fig. 49.2C).

**Lift-off test:** The dorsum of the patient's hand is placed against the lumbar spine (Fig. 49.2D). The patient then lifts the hand away from the back, maintaining the elbow in the coronal plane. Inability to do so indicates a lower **subscapularis** tear.

**Belly-press test:** The patient places both hands on the abdomen, internally rotates to bring the elbows forward beyond the coronal plane, and then presses the hands into the abdomen. Inability to move the elbow beyond the coronal plane indicates an upper **subscapularis** tear (Fig. 49.2D).

**Hornblower's sign:** The patient is asked to hold the arm in a 90-degree abduction and a 90-degree external rotation. A positive sign, wherein the arm falls into internal rotation, represents a **teres minor** pathology.

### Biceps Tendon Pathology

**Speed's test:** With the forearm supinated and the elbow extended, the patient forward flexes the arm against resistance. A positive test produces anterior shoulder pain.

**Yergason's test:** With the elbow in 90 degrees of flexion, the patient supinates the forearm against a resistive force. A positive test produces pain in the biceps region.

**Upper cut test:** With the elbow in 90 degrees of flexion and the forearm supinated, an upward and cross body motion is performed against resistance. A positive test will induce pain.

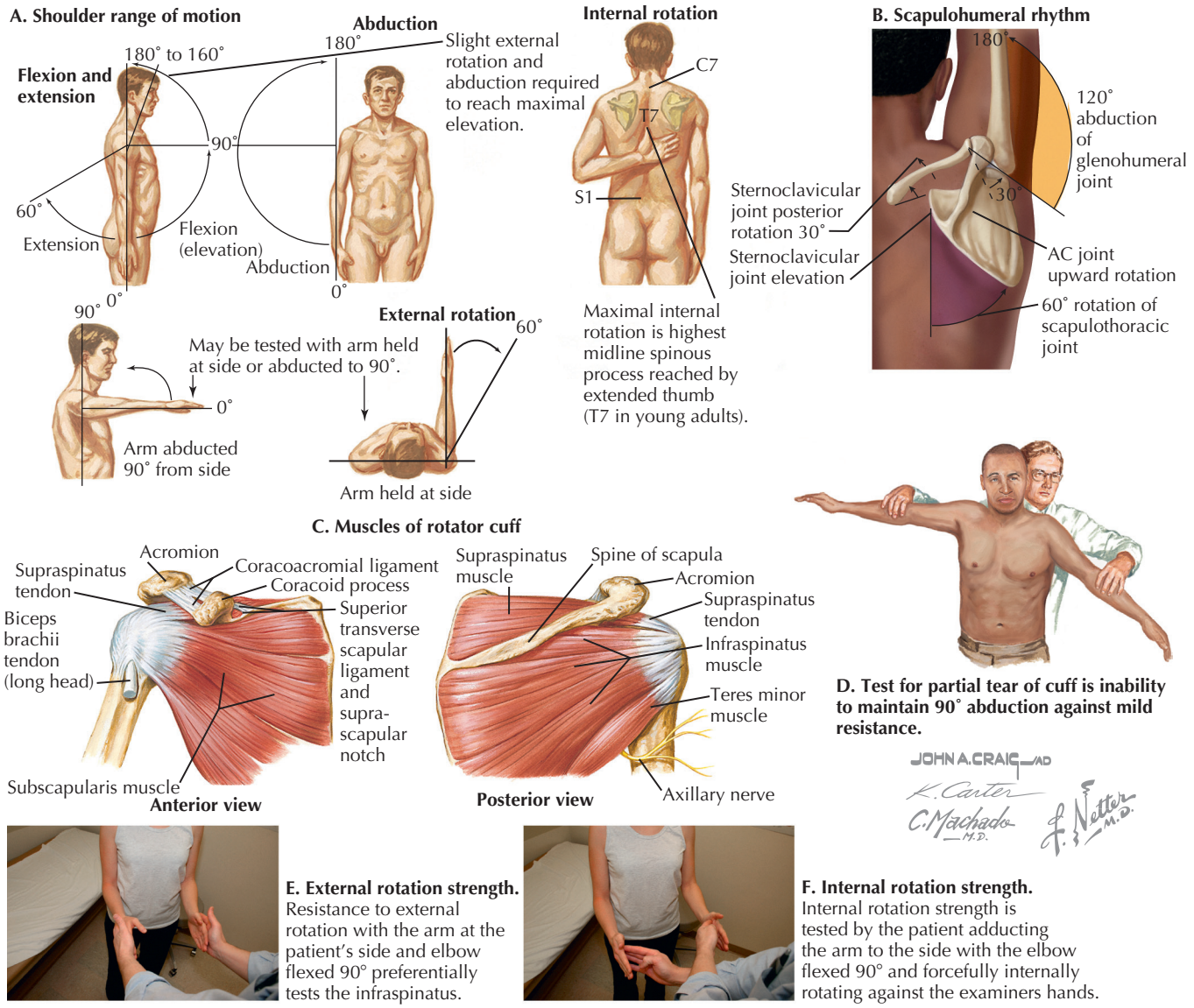



**Figure 49.1.** Physical examination of shoulder.

## Instability

It is important to perform these tests on the contralateral extremity for comparison and assessment of the patient's normal laxity.

### Anterior

**Apprehension test:** Best performed supine (table stabilizes the scapula); the arm is passively abducted to 90 degrees and then progressively externally rotated while the patient's response is noted. A positive test produces a patient response of "apprehension" by reproducing the patient's symptoms of anterior instability.

**Relocation test:** A posteriorly directed force is applied to the proximal humerus while performing the **apprehension test.** The test is positive when the patient's "apprehension" is relieved and greater external rotation can be achieved. **Note:** If the **apprehension test** produces pain (as opposed to a feeling of instability) that is relieved by the **relocation test**, it is suggestive of **internal impingement (Jobe relocation test)**.

**Load and shift test:** The humeral head is loaded to center it within the glenoid. It is then translated anteriorly and posteriorly. The amount of translation is graded as follows: grade 0, minimal; grade I, up to the rim of the glenoid; grade II, over the glenoid rim but spontaneously reduces; or grade III, over the glenoid rim and does not spontaneously reduce.

### Posterior

**Load and shift test:** See previous discussion.

**Posterior stress test:** Performed supine; the arm is flexed to 90 degrees and internally rotated. A posteriorly directed force is then applied to the humerus. A positive test causes subluxation.

**Jerk test:** Performed upright; the arm and the elbow are flexed 90 degrees. The arm is internally rotated and the humerus is loaded posteriorly. A positive test can cause posterior subluxation of the humeral head that is then reduced with a "jerk" when extending the arm.

### Multidirectional

**Sulcus sign:** Traction is applied to the arm in an inferior direction while observing the area lateral to the acromion for a "sulcus." Presence of a sulcus >1 cm indicates **inferior** laxity.

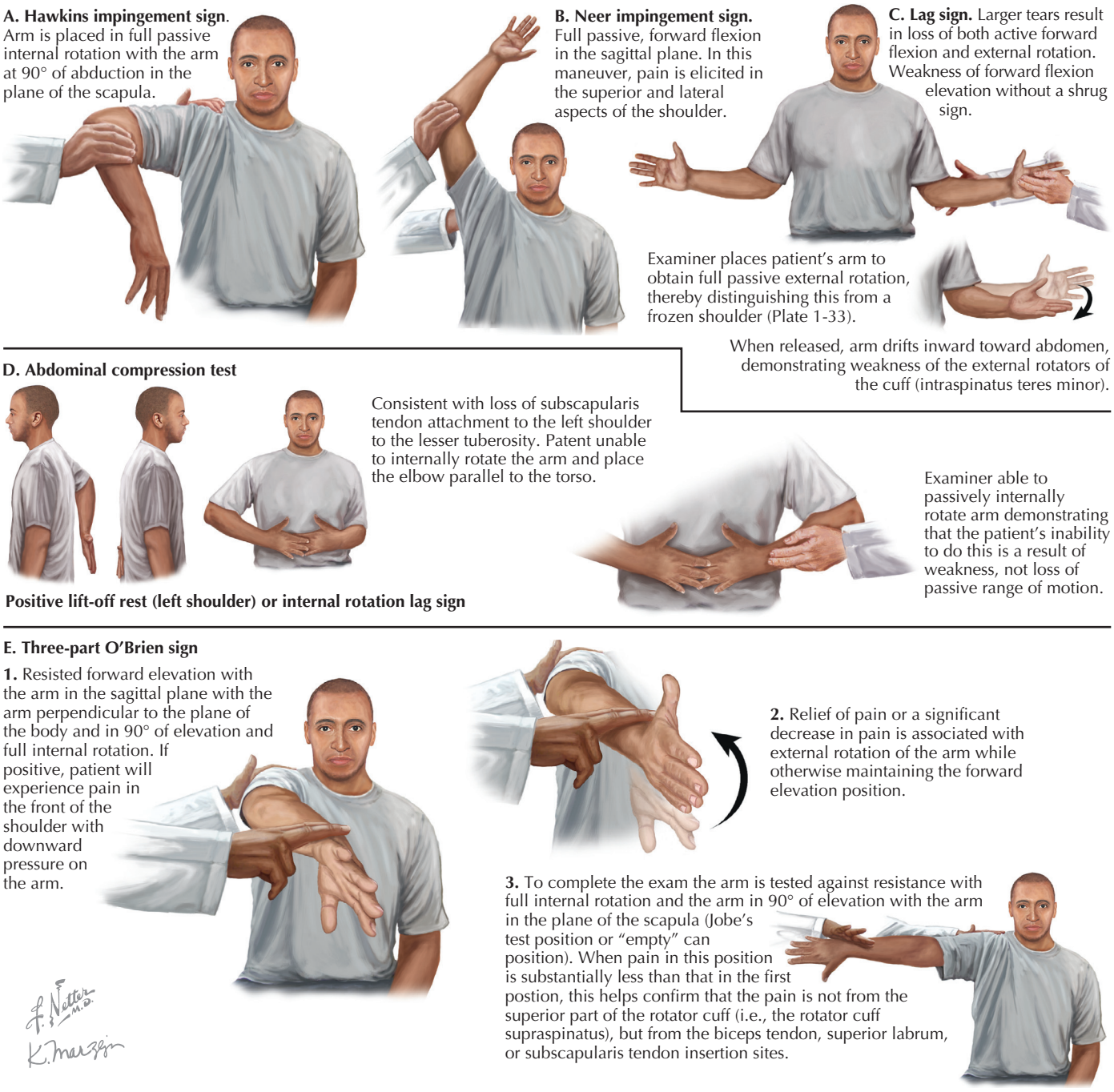



**Figure 49.2.** Special tests.

## Labrum

**Clunk test:** Performed supine; the arm is fully abducted, and the examiner's hand is placed on the posterior aspect of the humeral head. An anterior force is applied to the humerus, while the other hand rotates the humerus. Positive findings include a "clunk," pain, and grinding.

**O'Brien's test (active compression test):** The arm is positioned in 90 degrees of flexion and 10–15 degrees of adduction. A downward force is applied by the examiner as the patient resists, first with the arm internally rotated (thumb down) and then with the arm externally rotated (thumb up). A positive test causes pain felt deep within the joint that is reduced or relieved with the arm externally rotated, which indicates superior/posterior labral pathology (Fig. 49.2D).

**Kim test:** Performed upright; with the arm in a 90-degree abduction, a strong axial load is applied. While the arm is elevated to 45 degrees upward and diagonally, a downward and backward force is applied. A positive test is sudden onset of posterior shoulder pain, which indicates a posteroinferior labral lesion.

**Biceps load test:** Performed supine; the arm is abducted 120 degrees, maximally externally rotated, the forearm supinated, and the elbow flexed 90 degrees. Active elbow flexion is then performed against resistance. A positive test produces pain suggestive of a superior labrum anterior and posterior (SLAP) tear.

**Anterior slide test:** The patient's hands are placed on the hips with the thumbs pointing posteriorly. An axial load is applied at the elbow toward the GH joint against patient resistance. A positive test produces pain suggestive of a SLAP tear.

**Modified dynamic labral shear:** The arm is flexed 90 degrees at the elbow, abducted in the scapular plane to above 120 degrees and externally rotated to tightness. The arm is then lowered from a 120- to a 60-degree abduction, keeping the arm maximally externally rotated. A positive test produces pain or a painful click and indicates superior labral pathology.

### Acromioclavicular Joint

Direct palpation over the AC joint causes pain.

**Cross-arm adduction test:** The arm is flexed 90 degrees and then adducted across the chest. A positive test causes pain at the AC joint.

**O'Brien's test:** Can cause pain localized to the AC joint and should be distinguished from pain deep within the shoulder

## IMAGING OF THE SHOULDER

### Radiographs

**Anteroposterior (AP):** Taken in the plane of the thorax; provides an oblique view of the GH joint because of the anteverted position of the scapula on the posterolateral aspect of the thoracic cage (Fig. 49.3A).

**True AP (Grashey view):** Taken in the plane of the scapula; provides a true AP view of the GH joint by angling the beam approximately 45 degrees in the medial-to-lateral direction or by rotating the patient and placing the scapula flat on a radiograph cassette.

**Axillary view:** Important for evaluating dislocations; useful for evaluating fractures of the coracoid and the anterior or posterior glenoid rim; will additionally reveal an os acromiale

**West Point axillary lateral:** Provides a tangential view to the anteriorinferior glenoid rim useful for evaluating instability cases

**Scapular Y view:** Lateral radiographs recorded in the scapular plane; helpful in evaluating the relationship of the humerus to the glenoid fossa; tilting the beam caudally by 10 degrees produces a supraspinatus **outlet view,** which allows assessment of acromion morphology

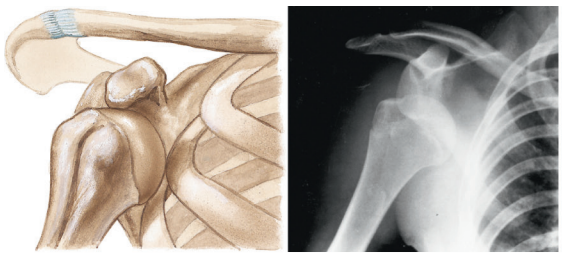
**Stryker notch view:** The patient is placed supine with the hand on the superior aspect of the head with the finger directed posteriorly. The beam is aimed 10 degrees cephalad at the coracoid; evaluates compression fractures or defects in the posterolateral aspect of the humeral head (Hill–Sachs lesion) (Fig. 49.3B)

**Serendipity view:** A 40-degree cephalic tilt view for visualization of the SC joint

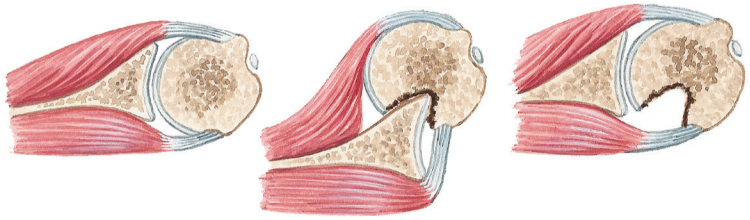
**Zanca view:** Provides clear view of the AC joint by directing the x-ray beam 10–15 degrees cephalad; allows assessment of AC separations, distal clavicular osteolysis, and distal clavicle fractures

### Magnetic Resonance Imaging (MRI)

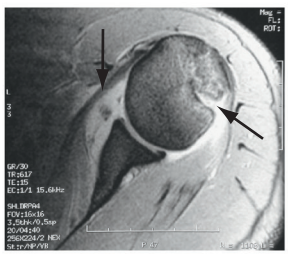
Magnetic resonance imaging (MRI) is the gold standard for evaluating soft tissue structures and cartilage; magnetic resonance arthrogram (MRA) preferred for labral pathology



**A.** Anterior shoulder dislocation

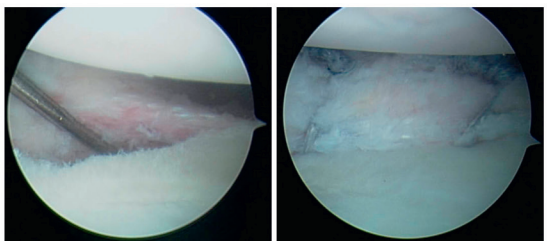


**B.** Anterior humeral dislocation with resulting Hill-Sachs deformity



**C.** MRI Bankart and Hill-Sachs

**D.** Reduction maneuvers for anterior shoulder dislocation



**E.** Arthroscopic images of Bankart

**Figure 49.3.** Anterior glenohumeral instability.

## SPECIFIC SHOULDER INJURIES

### Instability

**Description:** Defined as symptomatic, abnormal translation of the humeral head on the glenoid; instability can be classified in several ways—*direction of instability* (**anterior, posterior, multidirectional**), *traumatic versus atraumatic*, and *degree of instability*

### Anterior Glenohumeral Instability

**Description:** Most common direction of instability

**Mechanism of injury:** Direct or indirect trauma leading to tearing and attenuation of the anterior capsulolabral complex; direct trauma involves a blow to the posterior shoulder; indirect trauma involves injury to the arm in a position of abduction, extension, and external rotation position

**Presentation:** Can present with pain, feeling of weakness, instability, or recurrent dislocations; dislocations in younger patients are associated with *Bankart lesions*, whereas patients aged over 40 years typically have associated rotator cuff tears. A Bankart lesion is an avulsion of the anteroinferior glenoid labrum.

**Physical examination:** In acute dislocations, the lateral shoulder will lose its normal contour with fullness present anteriorly. The arm is held in slight abduction and in external rotation. A careful neurovascular examination is important. The nerve most commonly injured is the axillary nerve. The following examination maneuvers will be positive in patients with recurrent anterior instability: **apprehension test, relocation test,** and **load and shift test**.

**Differential diagnosis:** Multidirectional instability (MDI), rotator cuff tear, SLAP lesion, and proximal humerus fracture

**Diagnostics:**

- **Radiographs:** A standard shoulder series (AP, axillary, and scapular-Y) to ensure the humeral head is reduced. The **Stryker Notch** view can detect a Hill–Sachs lesion. The **West Point axillary view** will provide better evaluation of the anterior glenoid rim for possible *bony Bankart lesions*.
- **3D CT:** Gold standard to evaluate bony pathology (particularly in recurrent instability to evaluate critical glenoid bone loss)
- **MRI or MRA:** Will demonstrate *Bankart lesions*, anterior capsule pathology and presence of Hill–Sachs lesion (with associated T2 edema in acute injuries) (Fig. 49.3C)

**Treatment:**

- Acute anterior dislocations require urgent reduction. Several reduction methods have been described. The Stimson technique is a relatively atraumatic technique. The patient is placed prone, and weights are placed on the affected wrist (see Fig. 49.3D). A variation of this technique can be performed on the field by placing the athlete in the supine position and applying traction on the wrist in forward flexion and counter traction on the chest (see Fig. 49.3D). Another technique is the Milch technique; this is performed with the physician placing a hand on the superior aspect of the dislocated shoulder while using a thumb to stabilize the humeral head in a fixed position. The arm is then abducted with application of a gentle longitudinal traction while the humeral head is manipulated with the thumb over the glenoid rim. Slight external rotation can help facilitate the manipulation of the humeral head over the glenoid rim. Intra-articular injection of a local anesthetic has been shown to be effective in aiding reduction, although relaxation with an IV sedative may be necessary.
- The value of postreduction treatment with sling immobilization (particularly in external rotation) is controversial. There is no consensus in the literature on the utility of sling use, period of immobilization, or the degree of rotation. It is currently recommended for 4–6 weeks of immobilization in neutral rotation.
- Initial treatment is physical therapy focusing on strengthening dynamic stabilizers of the GH joint (rotator cuff muscles, deltoid and scapula stabilizers) and maintaining GH motion.
- Recurrent instability should be surgically treated with anterior stabilization. Current arthroscopic techniques have results equivalent to those with open techniques (Fig. 49.3E). Bony deficiencies of the anterior glenoid or large, engaging Hill–Sachs lesions may have to be addressed for successful outcomes.

**Prognosis and return to play:** The re-dislocation rate is approximately 90% in patients aged <20 years and decreases with increasing age. The decision to treat initially with surgical stabilization versus nonoperative therapy must take into account several factors such as patient age, activity level, and the specific sport. Return to play after nonoperative therapy requires near-normal range of motion, strength, and functional ability. Return to play after surgical stabilization is typically after 4–6 months.

### Posterior Glenohumeral Instability

**Description:** Tear or stretching of posterior capsulolabral structures leading to dislocation or subluxation

**Mechanism of injury:** Posterior subluxation or dislocation can result from a traumatic event with the arm in a position of flexion, adduction, and internal rotation causing a *reverse Bankart lesion* (Fig. 49.3E); more commonly, it can result from repetitive microtrauma causing a labral tear or capsular attenuation. This mechanism is commonly associated with an offensive lineman in football jamming his opponents while blocking.

**Presentation:** Posterior dislocation is rare compared to an anterior dislocation, and are easily missed upon initial evaluation. Patients presenting after a seizure or electrical shock should raise suspicion of a posterior dislocation. More commonly, posterior instability presents as pain or a feeling of instability posteriorly with a load to the arm in a position of forward flexion, adduction, and internal rotation.

**Physical examination:** In acute posterior dislocations, the arm is held in adduction and internal rotation. A prominent coracoid anteriorly and posterior fullness are present. The following maneuvers will be positive in patients with recurrent posterior instability: **load and shift test, posterior stress test,** and **jerk test**.

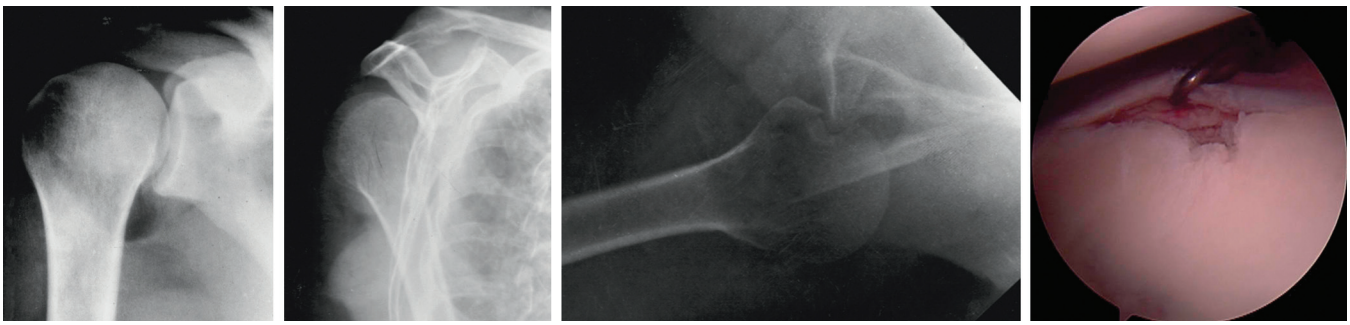
**Differential diagnosis:** MDI, rotator cuff tear, SLAP tear, and proximal humerus fracture

**Diagnostics:**

- **Radiographs:** A standard shoulder series (AP, scapular-Y, and axillary view) to ensure the humeral head is reduced. AP view may reveal "lightbulb sign." Plain radiograph films will also reveal reverse Hill–Sachs lesions indicative of a posterior dislocation and allow for evaluation of bony contributions to posterior instability, such as glenoid fractures, hypoplasia, or excessive retroversion (Fig. 49.4 A, B, and C).
- **CT:** Improved assessment of glenoid deformities contributing to instability (i.e., retroversion)
- **MRI or MRA:** Will demonstrate *reverse Bankart lesions* (posterior labral tear) or a redundant, attenuated posterior capsule (Fig. 49.4D)

**Treatment:**

- Acute posterior dislocations require reduction. This is often more difficult than reduction of an anterior dislocation. With the patient supine, traction is applied in line with the deformity while the humeral head is guided into the joint. Avoid external rotation to prevent proximal humerus fracture.
- For recurrent posterior instability, a majority will improve with physical therapy focusing on dynamic stabilizers of the



**A.** Anteroposterior radiograph. Difficult to determine if humeral head within, anterior, or posterior to glenoid cavity.

**B.** Lateral radiograph (parallel to plane of body of scapula). Humeral head clearly seen to be posterior to glenoid cavity.

**C.** True axillary view. Also shows humeral head posterior to glenoid cavity.

**D.** Posterior labral tear.

**Figure 49.4.** Posterior glenohumeral instability.

shoulder, particularly the posterior deltoid and external rotators. Surgery is indicated for patients who fail nonoperative treatment. Surgery is directed at the pathology with capsular plication for attenuated capsule and repair of *reverse Bankart lesions*. Bony abnormalities of the glenoid must be identified and addressed if present.

**Prognosis and return to play:** Criteria are similar to those for anterior instability.

## Multidirectional Instability (MDI)

**Description:** Symptomatic instability in more than one direction—inferior plus anterior or posterior

**Mechanism of injury:** Often atraumatic in the setting of generalized laxity or from repetitive microtrauma, most common in overhead athletes such as swimmers and volleyball players; the primary pathology is an attenuated inferior capsule with associated globally increased capsular volume

**Presentation:** A majority are young adults and often bilateral. Patients present with pain, instability, and occasionally transient neurologic symptoms. Symptoms when carrying heavy objects at one's side are indicative of inferior instability.

**Physical examination:** Examine for signs of generalized laxity, such as hyperextension of the elbows or ability to bring the thumb to the forearm (Beighton score). A positive **sulcus** and/or **Gagey sign** indicates inferior instability. Apply the tests described earlier to evaluate for anterior and posterior instability.

**Differential diagnosis:** Unidirectional instability, rotator cuff tear, and SLAP lesions

**Diagnostics:**

- **Radiographs:** Usually normal but may reveal a Hill–Sachs or bony Bankart lesion
- **MRA:** Can demonstrate excessive capsular volume and if have superimposed unidirectional instability, will demonstrate a Bankart lesion

**Treatment:** A majority of patients respond to physical therapy to strengthen the dynamic stabilizers of the shoulder. Those who fail nonoperative treatment are candidates for surgical stabilization via capsular shift/plication.

**Prognosis and return to play:** Even with surgical stabilization, a majority of patients return to a competitive level. The time frame for return to play after surgery is similar to unidirectional instability.

## Biceps Tendon Pathology

### Tendonitis

**Description:** *Primary* tendonitis is an isolated inflammatory condition of the long head of the biceps (LHB) brachii tendon in the intertubercular (bicipital) groove. More commonly, it occurs as a *secondary* process in conjunction with pathologic changes to surrounding structures in the shoulder such as rotator cuff pathology, impingement syndrome, bursitis, and AC joint disorders. This results in fraying or degeneration of the proximal LHB tendon.

**Mechanism of injury:** Overuse injury causing repetitive trauma to the LHB tendon. Chronic inflammation can lead to the tendon sheath becoming thickened and the tendon developing degenerative changes. These changes lead to scar tissue formation and the LHB becomes fixed within the bicipital groove.

**Presentation:** Pain in the anterior aspect of the shoulder that may radiate down the biceps; usually, a history of overuse and no history of trauma

**Physical examination:**

- Tenderness anteriorly over the bicipital groove
- Speed's, Yergason's, and upper cut tests are positive
- Because of its association with impingement, Hawkins' and Neer's tests are often positive.

**Differential diagnosis:** Rotator cuff pathology and labral tear

**Diagnostics:**

- Radiographs are normal in primary bicipital tendonitis; they may show an acromial spur suggestive of impingement associated with secondary bicipital tendonitis.
- Ultrasound will show thickened tendon within the bicipital groove, but this test is heavily reliant on operator technique.
- MRI is the gold standard and may show edema in or around the tendon or a thickened or split tendon. MRI is additionally helpful at showing associated pathology.

**Treatment:** Begins with nonoperative therapy consisting of rest and nonsteroidal anti-inflammatory drugs (NSAIDs), followed by range-of-motion exercises. Corticosteroid injections have utility in the bicipital sheath (via ultrasound guidance) or in the intra-articular space or subacromial space (particularly for secondary tendonitis). Surgery is indicated for those who fail conservative therapy. Surgical options include tenotomy or tenodesis with concomitant management of associated pathology.

**Prognosis and return to play:** Once pain has resolved enough to allow near-normal range of motion and strength. For isolated biceps tenodesis, a sling is typically used for 3–4 weeks with light work allowed at 4 weeks and completely unrestricted activity at 3–4 months.

### Instability/Subluxation

**Description:** The LHB tendon subluxates out of the bicipital groove.

**Mechanism of injury:** Invariably associated with complete or partial tear of the subscapularis and structures of the rotator interval that comprise the bicep pulley system

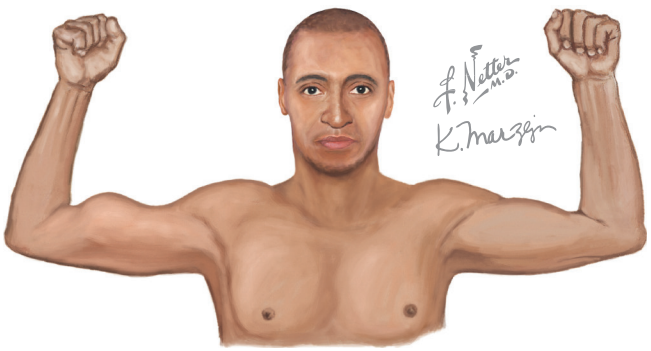
**Presentation:** Similar to bicipital tendonitis; patients may also report popping during shoulder motion
**Physical examination:** Similar to bicipital tendinitis
**Differential diagnosis:** Bicipital tendonitis and rotator cuff pathology
**Diagnostics:** Radiographs will be normal. MRI will reveal injury to the subscapularis and dislocation of the proximal biceps tendon from the bicipital groove, if present. Ultrasound is both sensitive and specific for diagnosing subluxation.
**Treatment:** Conservative therapy is similar to that for bicipital tendonitis. Surgical intervention (tenotomy or tenodesis) is more appropriate as primary treatment in young active patients or in those who fail conservative therapy. It is important to address associated cuff pathology as well.
**Prognosis and return to play:** Those undergoing tenotomy can return to play when near-normal strength and range of motion has returned. Tenodesis procedures will require 4–6 months to allow adequate healing and rehabilitation.

### Rupture

**Description:** Disruption of the LHB tendon
**Mechanism of injury:** Most commonly caused by forceful elbow flexion against resistance
**Presentation:** In acute traumatic ruptures, patients present with pain and ecchymosis anteriorly. Ruptures in older patients are often the result of attritional degeneration or chronic tendonitis. Such patients are often unaware that they have sustained a rupture.
**Physical examination:** Acutely, patients will experience tenderness over the anterior shoulder and the upper arm. Swelling and ecchymosis will be present. The classic "Popeye" deformity will be present because the biceps will be more prominent in the middle of the arm (Fig. 49.5). Patients may experience a slight decrease in the strength of elbow flexion and forearm supination.
**Differential diagnosis:** Proximal humerus fracture, bicipital tendonitis/instability/subluxation, rotator cuff tear, and pectoralis tear
**Diagnostics:** Radiographs are unremarkable. MRI will confirm the diagnosis.
**Treatment:** Pain control and therapy to maintain motion in the elderly. Surgical tenodesis may be more appropriate for young active patients or those concerned with the cosmesis of the "Popeye" deformity.
**Prognosis and return to play:** Same as discussed earlier for tenotomy and tenodesis

### SLAP Lesion

**Description:** The SLAP lesion describes any injury to the superior glenoid labrum.

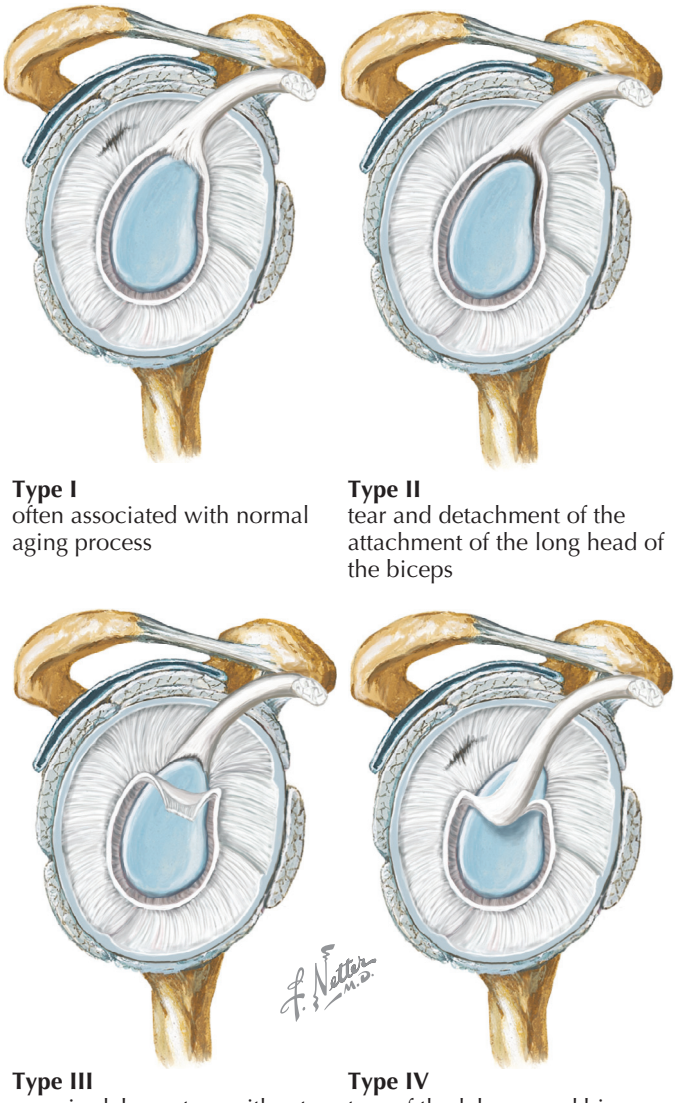


Rupture of tendon of long head of right biceps brachii muscle indicated by active flexion of elbow

**Figure 49.5.** Rupture of long head biceps brachii muscle.

**Mechanism of injury:** Several are atraumatic with an insidious onset of pain. Traumatic mechanisms include traction, compression, and direct blow injuries. Traction injuries occur in overhead-throwing athletes or in a sudden pull in an inferior or superior direction, such as catching oneself from falling. Compression injury occurs when falling onto an outstretched slightly abducted arm.
**Classification:** The original classification described four types of lesions, as described below (Fig. 49.6). Several additional types have since been added.

- **Type I:** Fraying of the superior labrum but the biceps anchor and labrum are still attached
- **Type II:** Most common clinically significant lesion; the superior labrum and biceps anchor are detached from the superior glenoid; further classified as anterior, posterior, or combined; posterior and combined type II SLAPs are often associated with throwing athletes because of the *"peel-back" phenomenon* as the arm is abducted while in an externally rotated position.



**Type I**
often associated with normal aging process

**Type II**
tear and detachment of the attachment of the long head of the biceps

**Type III**
superior labrum tear without involvement of the long head of the biceps

**Type IV**
tear of the labrum and biceps tendon

**Figure 49.6.** Superior labrum anterior and posterior (SLAP) injury.

- **Type III:** A bucket handle tear of the superior labrum, but the biceps anchor is still attached to the glenoid
- **Type IV:** The bucket handle tear of the superior labrum extends into the biceps tendon

**Presentation:** Patients frequently complain of pain, particularly during overhead activities, and decreased function. Patients also report mechanical symptoms of popping, clicking, or catching with motion. Symptoms are usually difficult to distinguish from those associated with impingement or rotator cuff tears.

**Physical examination:** Several examination maneuvers have been described, but no one test has proven to be consistently successful in diagnosing a SLAP tear. SLAP lesions are often associated with rotator cuff tears or instability, complicating the diagnosis. The following examination maneuvers are considered positive for SLAP lesions if the patient describes pain as deep within the joint: **O'Brien's test, biceps load test, anterior slide test modified dynamic labral shear, positive apprehension test,** and **positive relocation test**.

**Differential diagnosis:** Instability, impingement, rotator cuff tear, and GH arthritis

**Diagnostics:** MRI and MRA are the best imaging studies to evaluate for a SLAP lesion. Great variability in the normal appearance of the superior labrum exists, making interpretation difficult, but presence of a superior paralabral cyst has a high correlation with a SLAP lesion.

**Treatment:** Nonoperative treatment begins with rest, NSAIDs, and physical therapy to focus on strengthening and stretching because several overhead athletes will have an associated tight posterior capsule and loss of internal rotation. Patients who fail conservative therapy are indicated for surgery. Surgical treatment is dictated by the type of SLAP lesion. In general, types I and III lesions require debridement, type II lesions require repair, and type IV lesions require repair or tenodesis depending on the extent of involvement of the biceps tendon.

**Prognosis and return to play:** Throwing athletes require 6–7 months to fully rehabilitate; nonthrowing athletes typically return to sport at 4–6 months.

## Rotator Cuff Pathology

### Impingement Syndrome

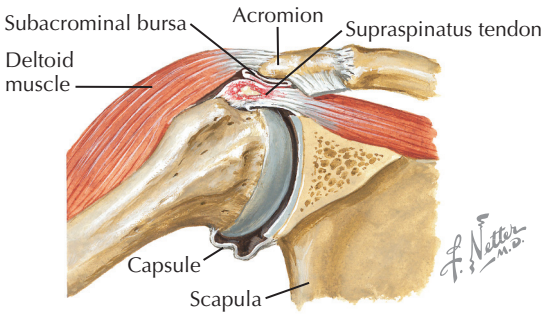
**Description:** Impingement syndrome encompasses a spectrum of pathologies, including subacromial bursitis, rotator cuff tendinopathy, and partial-thickness rotator cuff tears. Three types of impingement entities have been described: **subacromial impingement**, **internal impingement,** and **subcoracoid impingement**.

**Mechanism of injury:**

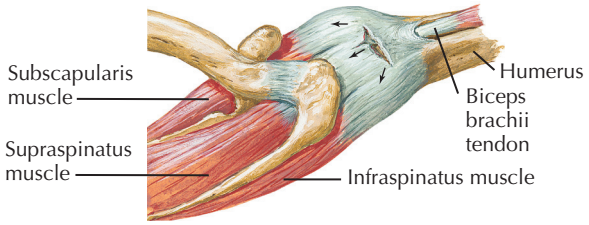
- **Subacromial impingement:** Impingement of the rotator cuff on the undersurface of the acromion and coracoclavicular ligament (Fig. 49.7); this can be the result of acromion morphology, rotator cuff muscle fatigue, degenerative tendinopathy, or AC joint spurring/hypertrophy.
- **Internal impingement:** Particularly in throwers, the articular surface of the rotator cuff comes into contact with the superior glenoid labrum. Although considered anatomically normal, overuse combined with instability or loss of internal rotation leads to combined articular-sided rotator cuff and SLAP tears.
- **Subcoracoid impingement:** Contact between the rotator cuff and a prominent coracoid; the prominence can be idiopathic or iatrogenic (after osteotomy)

**Presentation:**

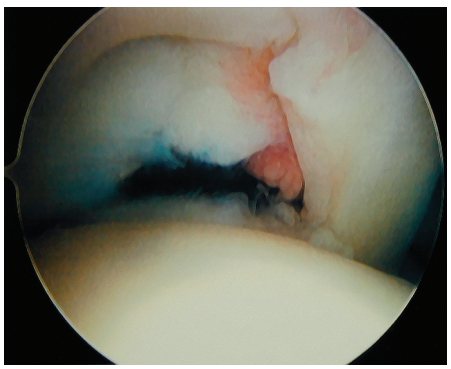
- **Subacromial impingement:** Presents with symptoms typical for rotator cuff pathology; this includes anterolateral shoulder pain that radiates to the lateral arm. Pain is



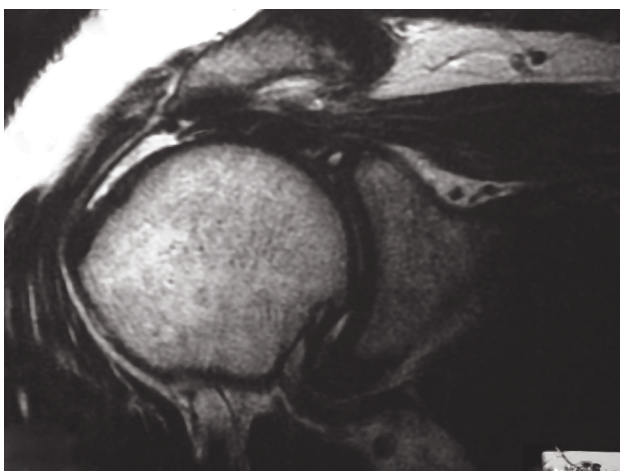

**Rotator cuff disease**




**Acute rupture (superior view).** Often associated with splitting tear parallel to tendon fibers. Further retraction results in crescentic defect as shown at right.



Arthroscopic image of small rotator cuff tear as viewed from the joint



MRI large cuff tear

**Figure 49.7.** Rotator cuff injury.

exacerbated by overhead activities. Pain at night and when lying on the affected shoulder is extremely common.
- **Internal impingement:** Posterior–superior shoulder pain or pain described as deep in the joint; exacerbated by activities that place the arm in an abducted, externally rotated position
- **Subcoracoid impingement:** Anterior shoulder pain exacerbated by activities that involve forward flexion and internal rotation

**Physical examination (see the earlier "special test" section for description of tests):** Neer's impingement sign, Hawkins' test, painful arc, coracoid impingement sign, and Jobe relocation test

**Differential diagnosis:** Instability, GH arthritis, adhesive capsulitis, cervical radiculopathy, calcific tendonitis, AC joint arthritis, and thoracic outlet syndrome

**Diagnostics:**
- **Neer's test:** A subacromial lidocaine injection is given. Resolution of pain when performing Neer's impingement sign maneuver is a positive test.
- **Coracoid impingement test:** Lidocaine injection just lateral to the coracoid; resolution of symptoms when performing the coracoid impingement sign maneuver indicates a positive test.
- **Radiographs:**
  - AP of the shoulder to rule out GH arthritis
  - Outlet view allows evaluation of acromion morphology (type I—flat, type II—curved, type III—hooked). Type III acromions are associated with a greater incidence of rotator cuff pathology.
  - Axillary view allows evaluation of the coracoid.
- **MRI:** Will demonstrate rotator cuff tendinopathy, partial- or full-thickness rotator cuff tears, and subacromial bursitis
- **Coracohumeral index:** Distance between the coracoid and humerus can be measured on a CT or MRI. Normal is 8.6 mm with the arm in maximal internal rotation; symptomatic patients display a mean distance of 5.5 mm.

**Treatment:**
- NSAIDs and physical therapy to strengthen the rotator cuff and scapular stabilizers; subacromial corticosteroid injections are used in subacromial impingement.
- A majority of patients improve with conservative therapy, but those who fail may be indicated for surgical decompression. *Subcoracoid decompression* involves a partial resection of the coracoid; it involves bursectomy and acromioplasty. Surgical treatment for *internal impingement* is directed at the SLAP lesion and rotator cuff tear as necessary.

**Prognosis and return to play:** Athletes involved in overhead activities can return to sports once pain has resolved enough to allow for normal range of motion and near-normal strength.

## Rotator Cuff Tear

**Description:** Disruption of the tendon or tendons of the rotator cuff muscles; the supraspinatus tendon is most commonly involved (Fig. 49.7)

**Mechanism of injury:** Tears can occur acutely from direct or indirect trauma. Alternatively, chronic tears can be the result of long-standing tendinopathy that eventually progresses to a tear.

**Presentation:** Similar to subacromial impingement, patients present with anterolateral shoulder pain exacerbated by overhead activities. Night pain and pain while sleeping on the affected side are common. Weakness may be present depending on the acuity or size of the tear.

**Physical examination:** Inspection for atrophy of the supra or infraspinatus, which if present, indicates a chronic condition. Evaluate active and passive range of motion. Positive findings during the following examination maneuvers are suggestive of a rotator cuff tear:
- Supraspinatus testing
  - Strength testing
  - Champagne toast
  - Jobe test
  - Drop-arm sign
  - Impingement tests
- Infraspinatus
  - External rotation strength at 0 and 90; also tests teres minor
  - External rotation lag sign
- Teres minor
  - Hornblower's (indicative of massive tear)
- Subscapularis
  - Internal rotation strength
  - Lift-off test
  - Belly-press test

**Differential diagnosis:** Impingement, AC joint arthritis, biceps pathology, GH instability, adhesive capsulitis, and cervical radiculopathy

**Diagnostics:**
- **Radiographs:** AP view to identify proximal migration of the humeral head, which would indicate a massive/chronic and typically irreparable tear (acromiohumeral distance <7 mm)
- **MRI:** Extremely sensitive and specific for rotator cuff disease; can distinguish partial- and full-thickness tears. Other notable findings that can influence treatment plan and prognosis are fatty infiltration and atrophy of the rotator cuff muscles and degree of tear retraction (Fig. 49.7).
- **Ultrasound:** Extremely sensitive and specific for rotator cuff tears; cheaper than MRI, but is highly operator dependent

**Treatment:** Decision making should be individualized and take into account the patient's age and activity level. Nonoperative treatment tends to be less successful in patients who present with a symptom duration of >1 year and significant weakness. Nonoperative treatment is similar to that for impingement. Patients who fail nonoperative treatment are indicated for surgical repair. Acute traumatic tears are best treated with prompt surgical repair.

**Prognosis and return to play:** Overhead athletes undergoing surgical repair may require 6 months to 1 year before being able to fully return to sports. Only 50% of professional athletes return to same level of play as prior to injury, but most recreational athletes are able to return to prior level of play.

## Acromioclavicular Joint Injuries

### Sprains/Separations

**Description:** Involves sequential injury to the AC and coracoclavicular (CC) ligaments; includes involvement of the deltoid and trapezius muscle and fascia in higher-degree injuries

**Classification:**
- **Type I:** Sprain of the AC ligament; AC and CC ligaments are intact; normal radiographs (Fig. 49.8)
- **Type II:** Rupture of the AC ligament; CC ligaments are intact; radiographs will show slight elevation of the clavicle. The AC joint is unstable to examination, but a stress radiograph will *not* produce 100% separation.
- **Type III:** Complete rupture of the AC and CC ligaments with 100% superior displacement of the clavicle
- **Type IV:** Complete separation of the AC joint with the clavicle displaced posteriorly through the trapezial fascia
- **Type V:** More severe Type III with complete rupture of the AC and CC ligaments; also involves disruption of the trapezial and deltoid fascia off of the acromion and clavicle. The clavicle is superiorly displaced by 100%–300%.

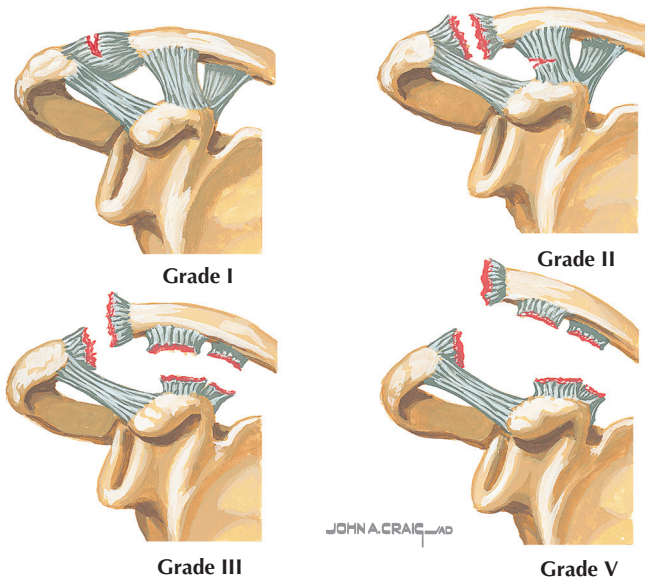



Figure 49.8. Acromioclavicular dislocation.

- **Type VI:** A rare inferior dislocation of the clavicle into a subcoracoid (lodged behind the conjoined tendon) or subacromial position

**Mechanism of injury:** A vast majority of such injuries are result from direct trauma by a fall or blow onto the shoulder with the arm adducted. An indirect mechanism involves falling onto an outstretched hand, which drives the humerus proximally into the acromion.

**Presentation:** The patient will usually report the aforementioned mechanism and complain of pain at the anterior/superior aspect of the shoulder. Common examples include hockey players being checked into the boards, football players taking a blow to the shoulder pads, or a cyclist falling off of a bike.

**Physical examination:** Tenderness will be present over the AC joint. Prominence of the distal clavicle will be evident in types II, III, and V. Stability of the distal clavicle can be assessed although it may be difficult in the acute setting. The **cross-arm adduction test** will be positive. The **O'Brien test** has been shown to have a high specificity for AC sprains if the pain localizes to that region.

**Differential diagnosis:** Clavicle fracture, distal clavicular osteolysis, and shoulder contusion

**Diagnostics:** In standard radiographs of the shoulder, the AC joint is overpenetrated and poorly visualized.

- **Bilateral AP**: Used to compare displacement of AC and CC to uninjured side
- **Zanca view:** See earlier discussion under Imaging of the Shoulder
- **Axillary view:** Allows evaluation for anterior or posterior displacement of the clavicle
- **MRI**: Can directly visualize the CC ligaments, allowing for more definitive classification of injury

**Treatment:**

- Types I and II are nonoperatively treated with a brief period of immobilization in a sling, ice, and analgesics followed by physical therapy. A local anesthetic injection can be used for in-game situations to allow the athlete to resume play. Chronic type II injuries that are symptomatic can be surgically treated with distal clavicle excision and anatomic reconstruction.
- Type III: Treatment is controversial, but most lean toward nonoperative treatment. If the patient has persistent pain or is unable to return to the desired level of activity, distal clavicle excision and anatomic reconstruction can be performed. Certain experts favor early operative management for high-level throwing athletes.
- Typically, types IV, V, and VI are surgically treated.

**Prognosis and return to play:** Those nonoperatively treated will be able to return to sports in 1–6 weeks depending on the severity of the injury and sport played. Those surgically treated will require 4–6 months before returning to sports.

### Distal Clavicle Osteolysis

**Description:** A painful condition of the AC joint caused by lysis and resorption of the distal clavicle

**Mechanism of injury:** Overuse injury producing repetitive microtrauma to the distal clavicle that leads to bone resorption

**Presentation:** Most common in young males and often bilateral; frequently associated with weightlifting. Patients present with pain over the anterior/superior aspect of the shoulder. Pain is exacerbated with more demanding activities. Weightlifters are more symptomatic with specific exercises such as bench pressing, dips, and push-ups.

**Physical examination:** Tenderness over the AC joint; there may be a prominence of the distal clavicle and crepitus with motion. The **cross-arm adduction test** will elicit pain. Stability of the distal clavicle should be assessed because this may affect surgical planning.

**Differential diagnosis:** Rotator cuff disease, infection, hyperparathyroidism, clavicle fracture, and AC separation

**Diagnostics:**

- Zanca view reveals osteopenia and expansion of the distal clavicle. Joint space widening and cysts may be present as well.
- MRI will reveal T2 edema in the distal clavicle and periarticularly throughout the AC joint.
- Bone scan is sensitive and is useful in cases wherein radiographic findings are not obvious.
- A diagnostic injection of local anesthetic into the AC joint is useful in cases wherein the diagnosis is unclear.

**Treatment:** Initial treatment should be nonoperative and comprises modification of activities and weightlifting technique, NSAIDs, and corticosteroid injections. Distal clavicle resection is indicated in patients who fail nonoperative treatment or cannot tolerate the extended course usually required for complete resolution of symptoms.

**Prognosis and return to play:** Those nonoperatively treated with a local injection or symptomatic treatment can return to sports as tolerated; those surgically treated will require at least 4–6 weeks before returning to sports.

## Sternoclavicular Joint Injuries

### Sprains, Subluxations, and Dislocations

**Description:** Involves injury (ranging from stretching to partial tearing to complete rupture) of the ligaments of the SC joint; these ligaments (intra-articular disc ligament, extra-articular costoclavicular ligament, capsular ligament, and interclavicular ligament) provide most of the stability to this extremely incongruent joint. Acute sprains are classified as mild (stable joint), moderate (joint subluxation), and severe (joint dislocation). Dislocations are further classified according to the direction of dislocation: anterior (more common) or posterior (Fig. 49.9). In the skeletally immature, the injury may be through the medial clavicular physis, which is the last of the long bones in the body to close. Can also present as spontaneous, atraumatic anterior subluxation.

**Mechanism of injury:** These injuries are the result of direct or indirect forces.

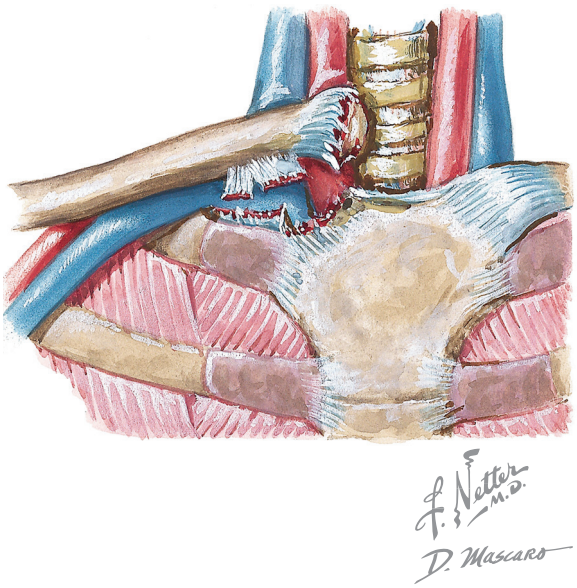



**Figure 49.9.** Dislocation of acromioclavicular or sternoclavicular joint.

- Indirect forces are more common and are applied to the SC joint from the anterolateral or posterolateral aspects of the shoulder. Compression and rolling forward of the shoulder cause a posterior dislocation, whereas compression and rolling backward of the shoulder cause an anterior dislocation. Such mechanisms can be seen in football pile-ons.
- Direct force applied over the anteromedial aspect of the clavicle will result in a posterior dislocation.

**Presentation:**

- Acute traumatic dislocations or physeal fractures present with significant pain and will often be supporting the arm with the uninjured side. Pain is exacerbated by shoulder motion. Dislocations will give an appearance of shortening of the shoulder. Patient will often tilt head toward injured side to minimize traction of sternocleidomastoid.
- A majority of spontaneous, atraumatic subluxations are not painful and occur with overhead elevation of the arm. The subluxation reduces when the arm is brought back down. These patients are usually in their teens to 20s, and numerous patients have generalized ligamentous laxity.

**Physical examination:**

- Anterior dislocations: Tenderness and swelling exists over a prominent medial end of the clavicle.
- Posterior dislocations: The medial end of the clavicle may not be palpable because it sits posterior to the sternum. Significant swelling anteriorly over the SC joint has been known to mask a posterior dislocation. Patients may have difficulty breathing or swallowing. Venous congestion can be present in the neck or extremity. Compression on the trachea or great vessels can make these injuries a medical emergency.

**Differential diagnosis:** Sternum fracture, rib fracture, and contusion

**Diagnostics:**

- **Radiographs:** Because of its location and the surrounding anatomy, the SC joint is difficult to image by using plain radiography. Several special views have been developed over time to maximize visualization.
- Serendipity view (see earlier description under Imaging of the Shoulder): Anterior dislocations will appear displaced superiorly to a horizontal line off of the normal clavicle. Posterior dislocations will appear displaced inferiorly.
- Heinig view: A lateral view centered at the manubrium and shot tangential to the SC joint and parallel to the opposite clavicle
- **CT scan:** The best imaging modality to evaluate the SC joint

**Treatment:**

- Mild sprain: Ice in the initial 12–24 hours; sling immobilization for comfort for initial 3–4 days, followed by a progressive range of motion and return to activities
- Moderate sprain/subluxation: Ice in the initial 12–24 hours; figure-of-eight strap to hold shoulders back and reduce SC subluxation; sling to support upper extremity
- Dislocation:
  - Anterior: Gentle reduction followed by figure-of-eight strap for 6 weeks and a sling; anterior dislocations are often unstable after reduction and may re-dislocate after figure-of-eight strap is discontinued. Conservative versus surgical treatment is then based on patient's symptoms and whether or not activities are limited.
  - Posterior: Closed reduction is the treatment of choice for acute injuries. It usually requires general anesthesia and results in a stable SC joint. Appropriate consultants should be available given the associated vascular injuries that have been known to occur. Again, postreduction care includes a figure-of-eight strap and a sling.
- Atraumatic spontaneous subluxation: Symptoms resolve spontaneously. Surgery is not indicated.
- Physeal fracture: Reduction and immobilization similar to dislocations; however, period of immobilization is shorter (3–4 weeks) because of fracture healing.

**Prognosis and return to play:** Patients report good to excellent outcomes with both operative and nonoperative management. Patients with mild and moderate sprains can return to sports in 2–4 weeks once the pain has resolved and motion has returned. Those with anterior dislocations should be withheld from sports for 6–8 weeks. Those with posterior dislocations should be withheld for a longer duration to allow for complete ligament healing, given the potential complications associated with a recurrent posterior dislocation.

## Clavicle Fracture

**Description:** Accounts for about 2.5% of all fractures; 80% occur in the middle third of the clavicle

**Mechanism of injury:** Similar mechanism as AC joint separations or direct blows to the clavicle

**Presentation:** The patient is usually splinting the injured extremity and can give a clear history of the injury-causing event (Fig. 49.10). A visible deformity with marked swelling and ecchymosis may be present in displaced fractures.

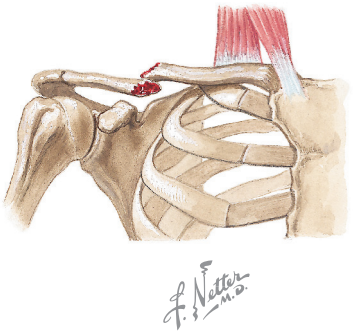
**Physical examination:**

- Important to check the neurovascular status of the involved extremity
- Evaluate the overlying skin at the fracture site to determine if it is at a risk of breakdown, converting a closed fracture into an open fracture.
- Rule out any associated musculoskeletal injuries to the cervical spine or ipsilateral upper extremity, and any visceral injuries such as a pneumothorax.

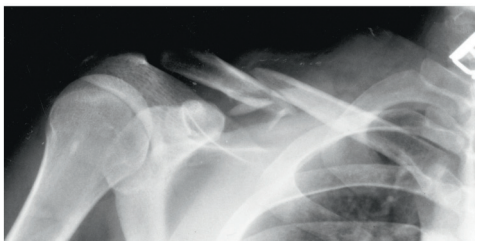
**Differential diagnosis:** AC separation, SC dislocation, scapula fracture, contusion, and congenital pseudoarthrosis

**Diagnostics:** Standard radiograph series of the shoulder to evaluate for associated shoulder girdle injuries (Fig. 49.10); a standard AP radiograph will not adequately demonstrate true displacement of the fracture. Therefore, an additional 45-degree cephalic tilt or axillary view with slight cephalic tilt is recommended.

**Treatment:** Historically, middle clavicle fractures have been nonoperatively treated with immobilization in a sling with good



Fracture of middle third of clavicle (most common). Medial fragment displaced upward by pull of sternocleidomastoid muscle; lateral fragment displaced downward by weight of shoulder. Fractures occur most often in children.



Anteroposterior radiograph. Fracture of middle third of clavicle.

**Figure 49.10.** Fracture of the clavicle.

results in terms of union and function despite the deformity associated with healing of displaced fractures. More recently, literature suggests higher nonunion rates and lesser outcomes associated with shortening of the clavicle (>2 cm). Surgical treatment has been shown to have fewer nonunions, faster time to union, and greater functional outcomes. Surgery also has implant-related complications resulting in multiple surgeries. With no proven advantage to surgical versus conservative management, the decision should be individualized based on the patient's goals and wishes. Surgical treatment is indicated in fractures that are open, tenting the skin, or associated with a neurovascular injury.

**Prognosis and return to play:** Noncontact athletes can return to sports once evidence of radiographic healing is present and full, painless, active range of motion with near-normal strength has returned. Contact athletes should be withheld for 2–3 months to allow for adequate healing.

## Proximal Humeral Epiphysitis (Little Leaguer's Shoulder)

**Description:** Growth plate injury (epiphysiolysis) of the proximal humerus

**Mechanism of injury:** Caused by overuse and repetitive microtrauma in overhead, skeletally immature athlete; poor throwing technique can contribute to the cause of injury

**Presentation:** Diffuse shoulder pain, worse with throwing or extremes of motion; usually gradual in onset and may present after a recent increase in throwing activity

**Physical examination:** Pain with palpation about the proximal humeral physis and possibly at extremes of shoulder range of motion; patients may present with external rotation contractures and decreased internal rotation

**Differential diagnosis:** Osteochondrosis of the proximal humerus, instability, and impingement

**Diagnostics:** Radiographs of the shoulder, including an AP with the arm externally rotated, allows evaluation of the physis; usually reveals physeal widening but may demonstrate metaphyseal fragmentation and periosteal reaction depending on severity. MRI will reveal the injury in occult cases not detected on radiographs.

**Treatment:** Most of these injuries are subtle with minimal displacement. Treatment for these cases begins with cessation of throwing for approximately 3 months. Then, a progressive throwing program should be initiated. Attention should be given to the athlete's throwing mechanics. In cases of significant displacement and/or angulation, treatment is based on the age and growth remaining of the patient.

**Prognosis and return to play:** Adherence to the treatment protocol of rest, followed by a throwing program with appropriate mechanics, will return a vast majority to play without return of symptoms.

## Glenohumeral Internal Rotation Deficit (GIRD)

**Description:** Loss of glenohumeral internal rotation of >25 degrees; overhead athletes will often gain external rotation adaptively to increase velocity. The gain in external rotation usually leads to a loss of internal rotation. If the loss of internal rotation exceeds the gain in external rotation, it is considered pathologic.

**Mechanism of injury:** Posterior–inferior capsular contracture; the posterior capsular contracture alters the kinematics of the shoulder, leading to SLAP lesions (most commonly type II) or impingement.

**Presentation:** Patients complain of shoulder pain and decreased performance. They may report difficulty reaching across their body or up their back.

**Physical examination:** External and internal rotation are documented with the patient supine and the scapula stabilized. Impingement tests and O'Brien's test will be positive if impingement or a SLAP tear has developed.

**Differential diagnosis:** Adhesive capsulitis and physiologic loss of internal rotation (internal rotation loss is *not* greater than external rotation gain)

**Diagnostics:** Diagnosis is clinical. MRI will demonstrate associated SLAP lesions or cuff pathology indicative of impingement. Bennet lesion (posterior capsular calcification) may be present.

**Treatment:** Physical therapy to focus on posterior capsular stretching. Patients who do not regain internal rotation with a therapy program are candidates for arthroscopic release of the posterior–inferior capsule. Patients with recalcitrant pain despite improvement in internal rotation may have a SLAP lesion that needs to be surgically addressed.

**Prognosis and return to play:** 90% of patients respond to a stretching program in 2 weeks. A prophylactic stretching program will protect against GIRD and the potential development of an associated SLAP lesion.

## Adhesive Capsulitis (Frozen Shoulder)

**Description:** Characterized by pain and gradual loss of both active and passive motion of the GH joint caused by soft tissue contracture; risk factors include female gender, middle age, smoking, immobilization, and endocrine disorders such as diabetes and thyroid disease.

**Mechanism of injury:** Capsulitis can be idiopathic, which is termed "primary adhesive capsulitis." Etiology is unknown. When the condition is caused by a known intrinsic or extrinsic cause, such as postsurgical or posttraumatic, it is termed "secondary adhesive capsulitis."

**Presentation:** Depends on stage of the disease, but the hallmarks are pain and limited active and passive motion; four stages have been described.

- Stage 1 ("painful stage"): Pain with active and passive range of motion; positive rest and night pain; symptoms present <3 months

- Stage 2 ("freezing stage"): Chronic pain and progressive loss of range of motion; positive rest and night pain; symptoms present 3–9 months
- Stage 3 ("frozen stage"): Significant shoulder stiffness; minimal rest and night pain; symptoms present 9–15 months
- Stage 4 ("thawing stage"): Minimal pain and progressive improvement in range of motion

**Physical examination:** Tenderness to palpation over deltoid insertion and on deep palpation over anterior and posterior capsule; careful examination of passive and active range of motion of the GH joint; make sure to stabilize the scapula so as not to be fooled by scapulothoracic motion, which can be increased to compensate for a lack of GH joint motion.

**Differential diagnosis:** GH arthritis, rotator cuff disease, and polymyalgia rheumatica

**Diagnostics:** Clinical diagnosis is adhesive capsulitis. Radiographs are unremarkable but are useful to rule out other pathologies. MRI findings include thickening of the coracohumeral ligament and joint capsule including the rotator interval.

**Treatment:**

- Options include benign neglect, physical therapy, NSAIDs, intra-articular corticosteroid injections, manipulation under anesthesia, hydrodilatation, and surgical capsular release.
- Treatment should be individualized depending on the stage of disease at presentation. NSAIDs and corticosteroid injections are beneficial for the inflammatory process associated with stages 1 and 2. Physical therapy during these stages focuses on gentle range-of-motion exercises and modalities for pain and inflammation. Range-of-motion exercises should be more aggressive during stages 3 and 4.
- Surgery is indicated for those who fail nonoperative treatment.
- Secondary adhesive capsulitis may be treated more aggressively.

**Prognosis and return to play:** A majority of patients will improve with conservative therapy but it is a protracted course. Up to 35% of patients can have minimal pain, minor residual deficits in motion, although functional limitations may exist in the long term.

## Pectoralis Major Tear

**Description:** Tear of the pectoralis major tendon; more commonly isolated to sternal head; usually a distal injury occurring as a tendon avulsion or rupture at the myotendinous junction; can occur as a proximal injury to the muscle belly

**Mechanism of injury:** Distal injuries are associated with strenuous activity such as football, wrestling, and weightlifting (during eccentric phase of bench press exercise). Proximal injuries to the muscle belly usually are the result of direct trauma.

**Presentation:** Patients may remember a specific incident and report feeling a tearing sensation and a "pop;" complaints of pain around the chest, axilla, and upper arm, associated with weakness and painful limited motion

**Physical examination:** Acutely, swelling and ecchymosis will be present over the anterior chest wall and the upper arm (if distal injury). Weakness will be evident with resisted adduction and internal rotation. Distal injuries will often have a palpable defect in the axilla and noticeable asymmetry compared to contralateral side in complete tears.

**Differential diagnosis:** Biceps tendon rupture, anterior dislocation that spontaneously reduced, rotator cuff tear, and contusion

**Diagnostics:** Although radiographs are usually normal, avulsion injuries and loss of the normal pectoralis major shadow may be detected. MRI is the modality of choice. It can distinguish between partial and complete tears and acute and chronic tears.

**Treatment:** Nonsurgical management applies only to proximal injuries or partial tears distally. Treatment consists of rest, ice, and physical therapy to maintain shoulder motion. Resisted strengthening should be incorporated after 6 weeks. Surgical management is indicated for complete distal tears.

**Prognosis and return to play:** Athletes undergoing surgical repair can expect a return to full or near-full strength.

## Proximal Humerus Fracture

**Description:** Commonly described by the Neer classification, which divides the proximal humerus into four parts: humeral head, greater tuberosity, lesser tuberosity, and humeral shaft; a fracture fragment is considered a part if it is displaced >1 cm (0.5 cm if the greater tuberosity) or angulated >45 degrees (Fig. 49.11).

**Mechanism of injury:** Sports-related fractures are usually caused by a high-energy impact or are avulsion fractures of the greater or lesser tuberosity associated with a dislocation. Fractures in the older population result from a fall onto the shoulder or an outstretched arm.

**Presentation:** Pain, swelling, ecchymosis, and inability to move the shoulder; patients often splint their arm against their body and support it with the uninjured arm

**Physical examination:** Tenderness and swelling about the shoulder will be present. Pain and crepitus with range of motion; a good neurovascular examination is important

**Differential diagnosis:** Dislocation, rotator cuff tear, contusion, clavicle fracture, and scapula fracture

**Diagnostics:** Plain radiographs are diagnostic.

- Standard trauma series of the shoulder (AP, axillary, and scapular-Y views.) The axillary view is critical to rule out a concomitant dislocation. If the patient is not able to tolerate

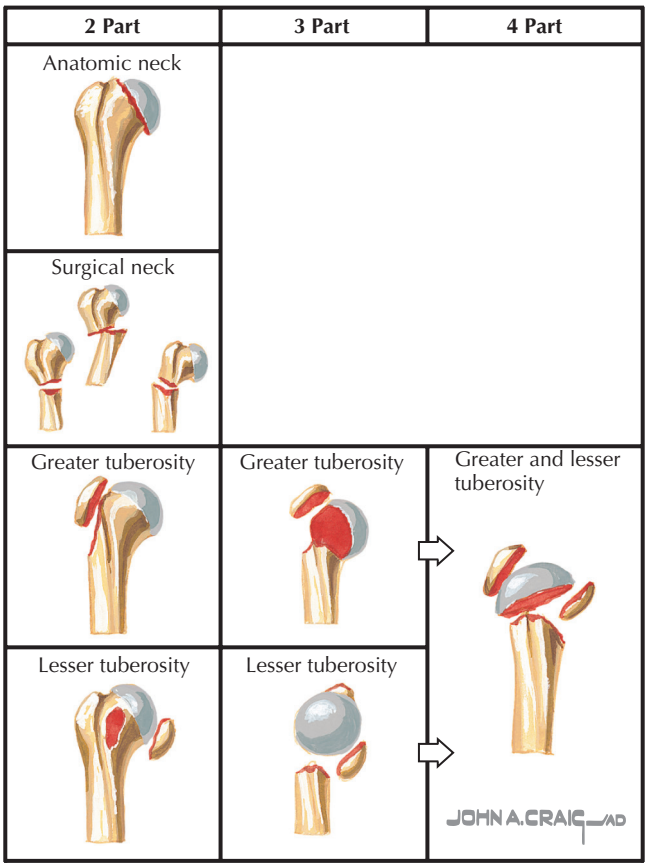



**Figure 49.11.** Neer classification of proximal humerus fractures.

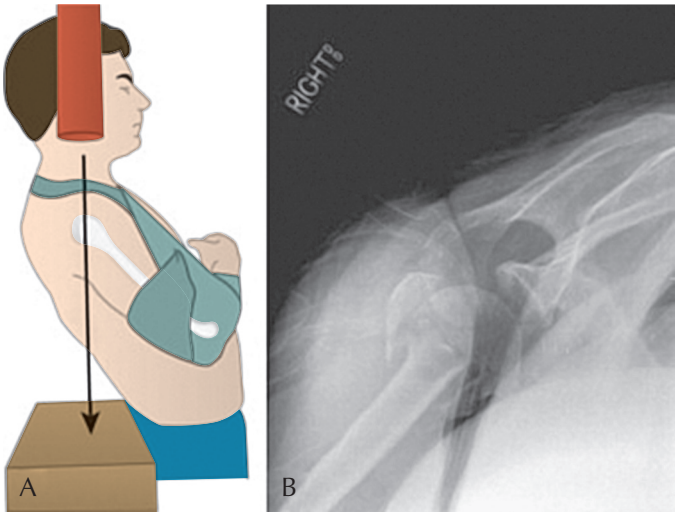



**Figure 49.12.** **A**. Velpeau axillary view may be taken; this allows the patient to keep the arm immobilized in the sling. The patient leans back and over the plate while the beam is directed from superior to inferior. **B**. Velpeau axillary radiograph showing proximal humerus fracture. Note that the head is located and the greater tuberosity is displaced posteriorly. (From Cuomo F, Zuckerman JD. *Proximal Humerus Fracture*. In: Browner BD, Techniques in Orthopaedics, vol 9. New York: Raven Press; 1994;9:143.)

a traditional axillary view, a *Velpeau axillary* can be taken without moving the arm (Fig. 49.12).

- CT is essential to evaluate the amount of displacement of the greater tuberosity and articular involvement.

**Treatment:** Most fractures are minimally displaced and can be treated in a sling with early passive range of motion. Displaced fractures (>1 cm) require open reduction and internal fixation. Recent data support arthroplasty solutions (reverse total shoulder) in the setting of displaced four-part fractures in elderly patients.

**Prognosis and return to play:** Noncontact, nonoverhead athletes can return in 2–3 months, once adequate fracture healing has occurred. Overhead athletes will require a longer recovery time to allow for return of full range of motion and strength.

## Neurological Syndromes Affecting Shoulder

### Parsonage–Turner Syndrome (or Brachial Neuritis)

**Description:** Inflammation of nerves of the brachial plexus

**Mechanism of injury:** Unknown etiology; often associated with preceding upper respiratory tract infection or unusually heavy exercise

**Presentation:** Initial complaint is spontaneous pain in the shoulder or the arm. As the pain resolves, weakness ensues.

**Physical examination:** Affected nerve distribution is variable but those most commonly affected are the axillary, musculocutaneous, suprascapular, and long thoracic nerves. Weakness in the corresponding muscle groups would be noted. Muscle atrophy may be apparent. Sensation is usually intact.

**Differential diagnosis:** Rotator cuff tear, adhesive capsulitis, suprascapular nerve entrapment, cervical spine pathology, and Pancoast tumor

**Diagnostics:** Imaging is not generally useful for diagnosis of brachial neuritis but is recommended to rule out intrinsic pathology. MRI is the imaging modality of choice. Electromyography (EMG) can be useful in diagnosing and in localizing the nerves involved.

**Treatment:** Pain control during the painful phase; physical therapy to maintain range of motion and progression to strengthening exercises as weakness resolves; certain studies have recommended early use of corticosteroids, but this is yet to be proven.

**Prognosis and return to play:** Self-limiting condition with overall good prognosis; time frame for complete recovery is variable: 36% recover by 1 year, 75% by 2 years, and 89% by 3 years.

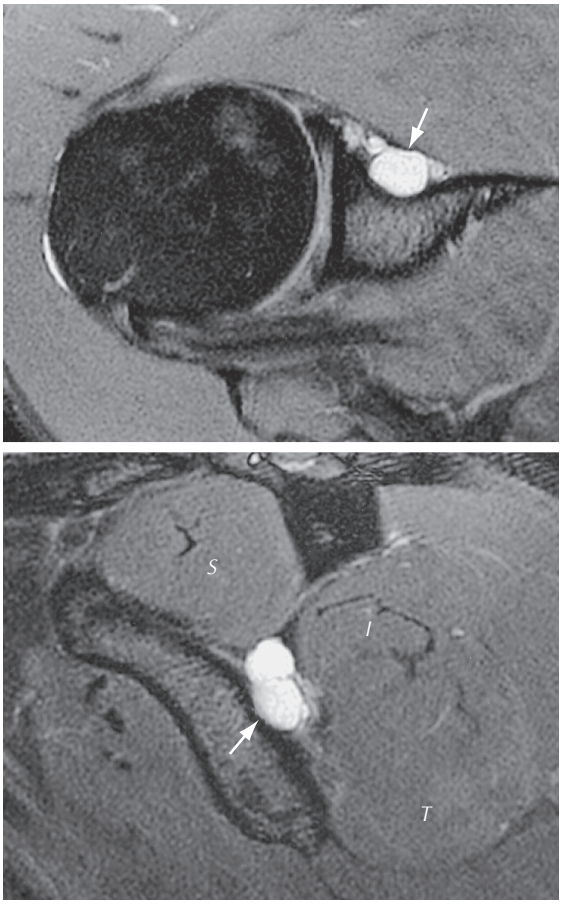



**Figure 49.13.** Spinoglenoid cyst. (From DeLee J, Drez D, Miller M. *DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice*, 2nd ed. Philadelphia: Saunders, Elsevier; 2002.)

### Suprascapular Neuropathy

**Description:** The suprascapular nerve provides motor function to the supra and infraspinatus muscle. Injury to the nerve can cause paralysis of these muscles.

**Mechanism of injury:** Can occur from a compressive lesion or from positional traction on the nerve caused by overuse (e.g., volleyball players or baseball pitchers); injury usually occurs at one of two locations: suprascapular notch or spinoglenoid notch. Compression at these locations can result from a ganglion or paralabral cyst (in the setting of labral tear), hypertrophied/calcified transverse scapular ligament, narrow suprascapular notch, or lipoma (Fig. 49.13).

**Presentation:** Vague deep pain in posterolateral shoulder with possible radiating down the arm or into the neck; difficulty elevating the arm past horizontal

**Physical examination:** Should focus on strength testing of the rotator cuff and shoulder girdle muscles; important to inspect for atrophy of the supra and/or infraspinatus muscles, which would be present in later stages. Distinguishing the involvement of both the supra and infraspinatus versus the infraspinatus alone indicates the location of injury. Injury at the suprascapular notch will involve both muscles, whereas injury at the spinoglenoid notch will be isolated to the infraspinatus.

**Differential diagnosis:** Cervical spine pathology, rotator cuff tear, and Parsonage–Turner syndrome

**Diagnostics:** Plain radiographs are usually normal. EMG can be helpful for diagnosis and to localize site of compression. MRI will demonstrate discrete compressive lesions and muscle atrophy.

**Treatment:** A majority of patients respond to conservative therapy consisting of activity modification, NSAIDs, analgesics, and physical therapy to strengthen the rotator cuff and scapular stabilizers. Surgical intervention is indicated in patients who fail conservative therapy, show signs of atrophy, or have structural compression. Ganglion cysts in the spinoglenoid notch are often the result of superior labral pathology. Treatment to address this intra-articular pathology also successfully decompresses the cyst.

**Prognosis and return to play:** A majority of nonoperative patients have good to excellent results at 6 months. Patients can return to play once near-normal strength and motion has returned.

## Scapular Dyskinesia

**Description:** Abnormal motion of the scapula in relation to the thoracic cage and a visible altered position of the scapula; dyskinesia of the scapula leads to altered kinematics of the GH and AC joints. SICK scapular syndrome is a severe form of scapular dyskinesia in that the scapula is in malposition at rest and motion. Findings can be remembered by the acronym **SICK** (**s**capular malposition, **i**nferior medial border prominence, **c**oracoid pain and malposition, and dys**k**inesia of the scapula).

**Mechanism of injury:** Most commonly the result of abnormal muscle activation and coordination; contracture of shoulder muscles, ligaments, and capsular structures (e.g., GIRD) can contribute to dyskinesia. Bony abnormalities that affect shoulder girdle motion, such as malunited clavicle fractures or AC joint injuries, can play a role.

**Classification:**

- **Type I:** Prominence of the inferior–medial scapular border
- **Type II:** Prominence of the medial scapular border
- **Type III:** Prominence of the superomedial scapular border; types I and II are associated with labral pathology whereas type III is associated with impingement and rotator cuff pathology

**Presentation:** Among throwing athletes, patients often complain of pain anteriorly and decreased performance level. Onset is usually insidious.

**Physical examination:**

- Examine for any abnormal static scapular position such as winging, elevation, depression, or rotation; also during shoulder motion to evaluate dynamic asymmetry
- Patients with SICK syndrome will have pain anteriorly over the coracoid caused by tightness of the pectoralis minor.
- Scapular pinch test: Isometric retraction of the scapulas will elicit a burning sensation in <20 seconds in those with scapular muscle weakness.
- Scapular assistance test: Determines if scapular dyskinesia is contributing to impingement signs; the scapula is stabilized by the examiner with forward flexion of the extremity; if impingement pain resolves or improves, the test is positive.
- Scapular retraction test: Scapula is stabilized in a retracted position. The test is positive if either the rotator cuff strength is improved or pain and impingement with the Jobe relocation test is improved.

**Differential diagnosis:** Serratus anterior injury, long thoracic nerve injury, trapezius injury, and spinal accessory nerve injury

**Diagnostics:** Radiographs will be normal. MRI will reveal associated pathology to the rotator cuff or labrum.

**Treatment:** The mainstay of treatment is physical therapy. This focuses on the periscapular muscles and their coordination in repositioning the scapula. Stretching of tight structures such as the posterior capsule and pectoralis minor is important. Therapy should include the trunk and lower extremities because of their involvement in the kinetic chain of throwing. Surgical treatment may be necessary to address associated rotator cuff or labral pathology.

**Prognosis and return to play:** Once the scapula is symmetric with the contralateral side, the throwing athlete can return to play, approximately 3 months.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 50 ELBOW INJURIES

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## GENERAL PRINCIPLES

### History and Physical Examination

#### History

- Hand dominance
- Location
  - Medial, lateral, anterior, and posterior
- Type of pain
  - Radiating symptoms
  - Numbness/tingling
  - Stiffness
  - Mechanical symptoms (locking and catching)
- Duration of symptoms
- Mechanism of injury
- Pain modifiers
- History of previous injuries
- Treatments rendered and results of treatments
- Recent changes in technique or training regimen

#### Physical Examination

- Inspection:
  - Compare to uninjured arm
  - Skin changes
  - Swelling
  - Ecchymosis
  - Muscle atrophy/hypertrophy
  - Carrying angle (cubitus valgus and cubitus varus)
- Neurovascular examination
  - Median, ulnar, radial, medial, and lateral antebrachial cutaneous sensation
  - Two-point discrimination at the fingertips (≤5 mm is normal)
  - Median, ulnar, and radial motor
- Range of motion (ROM) (Fig. 50.1)
  - Flexion and extension
  - Forearm pronation and supination
- Strength testing with isometric resistance
- Stability
  - Valgus–varus laxity
  - Posterolateral rotary laxity
  - Ulnar collateral ligament laxity
- Palpation: Identify areas of tenderness as anterior, posterior, medial, or lateral, and focus on key anatomic structures.
- Provocative maneuvers

### Ancillary Tests

- Radiographs: Anteroposterior (AP) and lateral
  - Special views:
    - 45-degree flexion view (capitellum)
    - Oblique (radial head view)
    - Axial projections (olecranon fossa or gun-sight view)
    - Gravity/manual stress view
- Computed tomography (CT) or magnetic resonance arthrogram: Articular incongruity, loose bodies, and ligament injuries
- CT scan: Fracture-dislocation, osteophytes/exostosis, and tendon calcification
- Magnetic resonance imaging (MRI): Soft tissue mass, ligament attenuation/rupture, chondral defects, and loose bodies
- Arthroscopy (intra-articular inspection): Loose bodies, chondral lesions, and synovitis
- Electromyogram–nerve conduction study (EMG–NCS): Nerve compression
- Ultrasound: Dynamic evaluation for ligament laxity or ulnar nerve instability

## ANTERIOR ELBOW INJURIES

### Distal Biceps Rupture

**Description:** Traumatic avulsion of the distal biceps tendon from the bicipital tuberosity of the proximal radius

**Mechanism of injury:** Eccentric muscle contraction against an extension load on a flexed elbow

**Presentation:** Atypical injury: 97% of biceps ruptures are proximal, only 3% of biceps ruptures occur at elbow; most often occur in males in the 4th–5th decades of life in the dominant extremity; often associated with a pop or tearing sensation in the proximal forearm or antecubital fossa

**Physical examination:** Tenderness to palpation in the antecubital fossa, acute swelling and ecchymosis are common, palpable tendon defect in complete tears (partial tears less common), tendon retraction if lacertus fibrosus torn ("Popeye sign"), and weakness with supination/elbow flexion

**Differential diagnosis:** Biceps tendonitis, bicipitoradial bursitis, and lateral antebrachial cutaneous nerve entrapment

**Diagnosis:** Primarily a clinical diagnosis

- Hook test: Flex the elbow to 90 degrees in full supination, and insert a finger from the lateral side into antecubital fossa to cord-like structure.
- Biceps squeeze test: Rest the elbow on a patient's lap in slight pronation and midflexion, firmly squeeze the belly of the biceps muscle with both hands, and observe for supination.
- Pain and/or weakness with resisted forearm supination

**Imaging:** Radiographs to rule out associated elbow injuries and evaluate irregularities around the radial tuberosity; ultrasound is cost effective but user dependent. MRI should be performed with the patient prone, shoulder abducted overhead, elbow flexed to 90 degrees, and forearm fully supinated.

**Treatment:** Acute anatomic repair superior to nonsurgical treatment. Allograft/autograft may be required in chronic cases if the tendon length cannot be restored. One- and two-incision repair techniques described; concern for heterotopic bone formation/radioulnar synostosis with the two-incision technique. Most common complication with either approach is LABC palsy (15%–40%), which is higher with the one-incision technique.

**Prognosis and return to sport:** A season-ending injury; patients surgically treated early can be expected to have near-full return of power and function with elbow flexion and supination.

### Pronator Syndrome (Median Nerve Entrapment)

**Description:** Compression of the median nerve at the level of the elbow with resultant nerve irritation (Fig. 50.2)

**Mechanism of injury:** No specific mechanism of injury has been associated with this disorder. Four possible sites of compression have been identified:

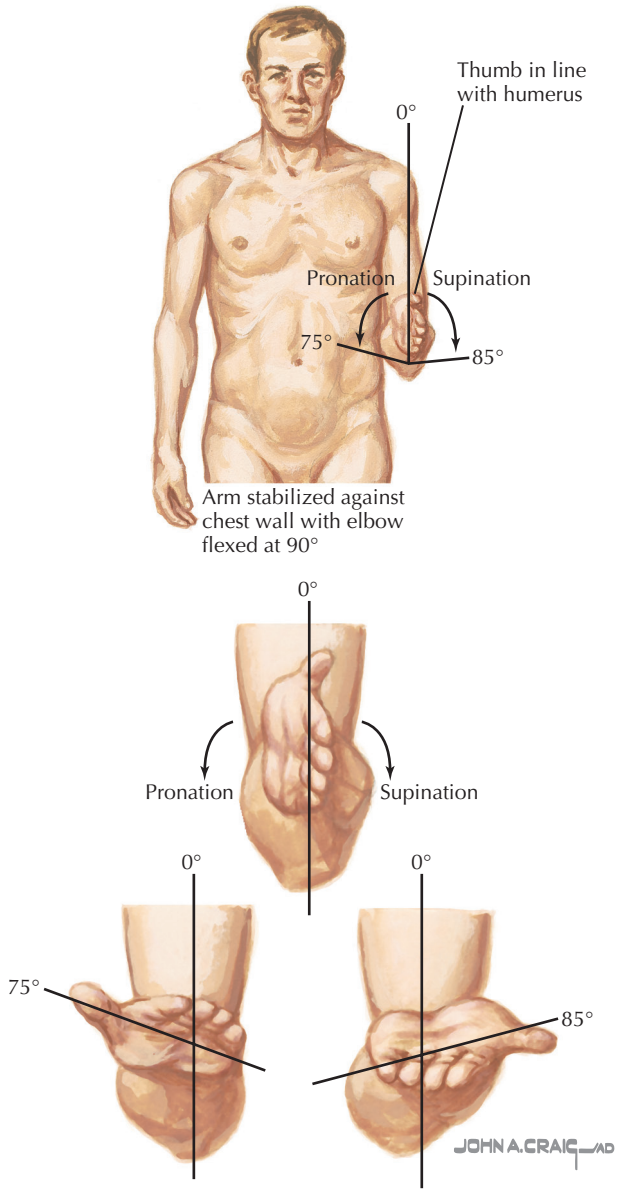



**Figure 50.1.** Measurement of pronation/supination.

- Tendinous arch of the flexor digitorum superficialis ("sublimis bridge")
- An accessory head of the FPL (Gantzer's muscle)
- Beneath the ligament of Struthers in patients with a supracondylar process
- At the lacertus fibrosus at the level of the elbow joint

**Presentation:** Presents similarly to carpal tunnel syndrome with numbness and paresthesias in the volar radial three and a half digits and volar forearm/wrist pain; distinguished from carpal tunnel syndrome by decreased sensation over the thenar eminence in the distribution of the palmar cutaneous branch of the median nerve. Symptom severity may increase with activity such as weightlifting, competitive driving, and underarm pitching. Nighttime symptoms are less common than those in carpal tunnel syndrome.

**Physical examination:**

- Pronator compression test: Apply pressure proximal/lateral to proximal edge of PT muscle belly, which reproduces pain/paresthesias within 30 seconds.
- Resisted pronation/supination can reproduce symptoms
- Resisted flexion of PIP of the middle finger can reproduce symptoms caused by compression of FDS heads.
- May have a positive Tinel sign over volar forearm

**Differential diagnosis:** Carpal tunnel syndrome, cervical spine or brachial plexus nerve compression, flexor–pronator tendonitis, and biceps tendonitis

**Diagnostics:** Plain radiographs are often normal but can reveal a supracondylar process (present in 1% of people, 5 cm proximal to the medial epicondyle); electromyogram (EMG) and nerve conduction study (NCS) help to rule out other sites of compression; however, EMG may not reveal nerve compression despite symptoms.

**Treatment:** Initial nonsurgical management with activity modification, forearm flexor stretching, and anti-inflammatory medications administered from weeks to months; if not responsive to nonsurgical management, then complete decompression of median nerve throughout its course in the proximal forearm should be performed by targeting release of the ligament of Struthers, the lacertus fibrosus, deep head of the pronator teres, and the FDS arch.

**Prognosis and return to sport:** Early active ROM, full return to activity by 6–8 weeks, and return to sport dependent upon restoration of strength and ROM

## POSTERIOR ELBOW INJURIES

### Triceps Rupture

**Description:** Traumatic avulsion of the triceps tendon from its insertion on the olecranon process of the ulna or avulsion of the olecranon process from the ulna with triceps tendon attached

**Mechanism of injury:** Most commonly occurs from forceful eccentric contraction of triceps such as fall onto outstretched hand or weightlifting

**Presentation:** Twice as common in males; associated with chronic anabolic steroids, systemic corticosteroids, and certain metabolic/systemic disorders. Patients report a recognizable event including a painful pop or a tearing sensation in the posterior elbow usually during an eccentric load with loss of elbow extension strength after the injury.

**Physical examination:** Tenderness to palpation along olecranon and distal triceps, acute ecchymosis, and edema; palpable defect of triceps tendon or step-off at olecranon; weak elbow extension/inability to hold elbow extended against gravity but not necessarily complete loss of extension strength; modified Thompson squeeze test: compressing the muscle bulk of the triceps fails to cause elbow extension

**Differential diagnosis:** Triceps tendonitis, partial triceps tear, olecranon bursitis, olecranon stress fracture, and posterior elbow impingement

**Diagnostics:** Largely a clinical diagnosis; "flake sign" (small bony avulsion fragment from olecranon process) observed in 80% of cases and pathognomonic for triceps rupture; MRI or ultrasound can help distinguish partial from complete tendon rupture and should be obtained to confirm severity of injury

**Treatment:** Nonsurgical treatment indicated only in elderly sedentary patients and is controversial in those with partial tears, comprises splint immobilization with the elbow in 30 degrees of flexion for approximately 3–4 weeks, followed by progressive flexion mobilization. Most complete ruptures or high-grade partial ruptures are managed with primary surgical repair within 2–3 weeks of injury.

**Prognosis and return to sport:** In general, a season-ending injury with at least 4–6 months of recovery/rehabilitation time expected; most athletes are able to return to sport at a level similar to that during the preinjury state with slight loss

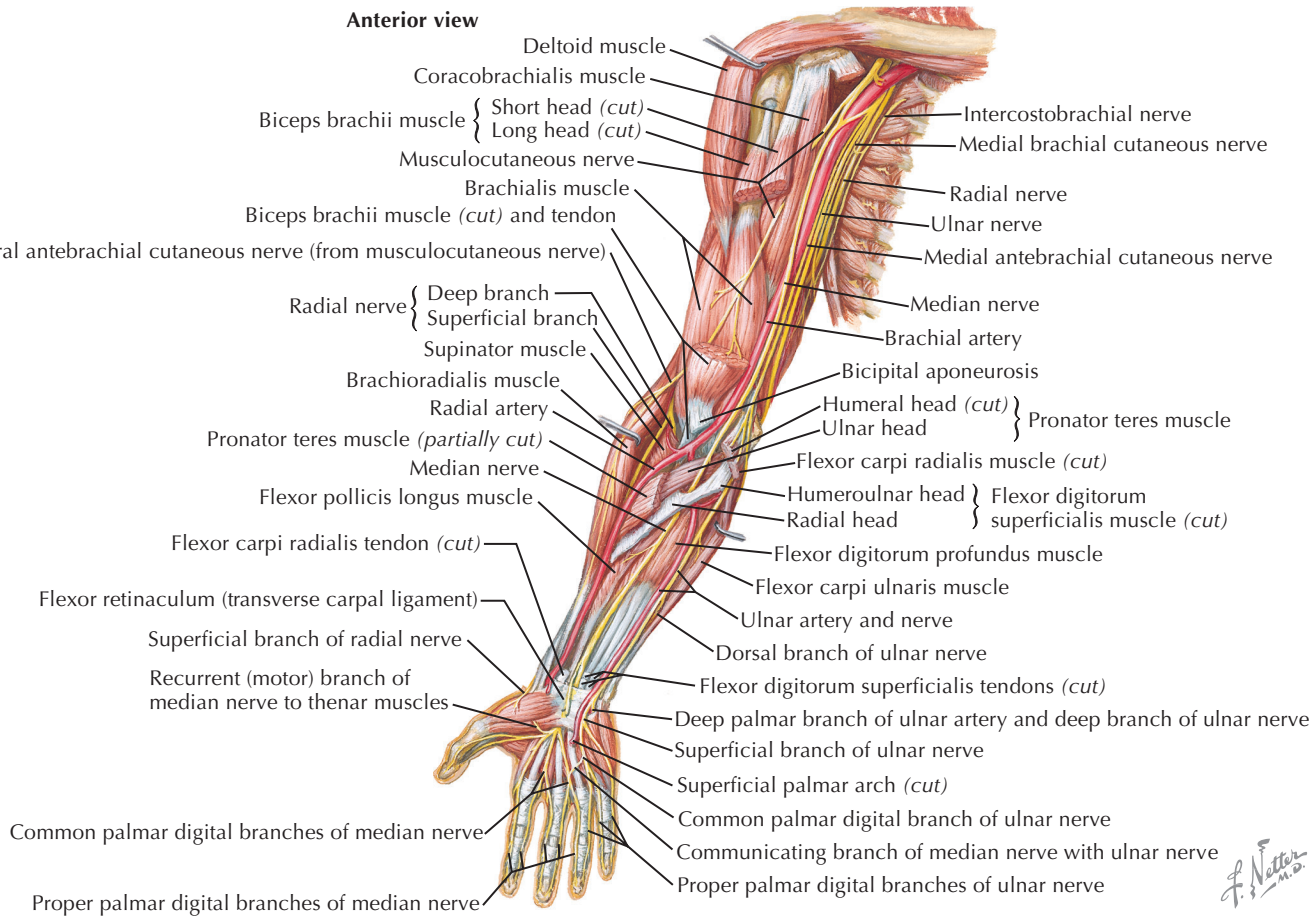



**Figure 50.2.** Nerves of the upper extremity.

of extension strength and ROM, with a re-rupture rate of up to 20%.

## Valgus Extension Overload

**Description:** Repetitive forceful shearing of olecranon within its fossa causing chondromalacia and olecranon osteophyte formation

**Mechanism of injury:** Overuse injury frequently seen in throwing athletes; bony constraints provide secondary stability to valgus stress to the elbow, which increases with extension; the olecranon traumatically abuts posteromedial olecranon fossa near full extension

**Presentation:** Pain localized to the medial aspect of olecranon in acceleration/deceleration throwing phases; limited extension because of impinging osteophytes; mechanical symptoms from loose bodies; exacerbated by medial ulnar collateral ligament (UCL) laxity; throwing athletes may complain of premature fatigue, loss of velocity, or loss of control

**Physical examination:** May demonstrate loss of terminal extension; posterior pain with pronation, valgus and extension; possible pain or laxity of the UCL with valgus stress; possible ulnar nerve irritation; can have palpable loose bodies/crepitus

**Differential diagnosis:** Olecranon bursitis, olecranon stress fracture, and triceps tendonitis

**Diagnostics:** Radiographs may posteromedial osteophytes, loose bodies, hypertrophic bone formation, calcification of the UCL, or medial epicondyle avulsion fractures. A 110-degree flexion oblique view can profile the posteromedial ulnar humeral joint. Radiographs may be normal; MRI can help to further assess the status of articular cartilage and better identify loose bodies

**Treatment:** Initial nonsurgical management of activity modification, NSAIDs, focused physical therapy with eccentric strengthening of wrist flexors, and when symptoms allow, a supervised throwing program with correction of flawed pitching mechanics. Patients failing nonsurgical treatment may be candidates for arthroscopic debridement to decompress the posterior compartment; avoid debriding >3 mm of bone because it increases the force in the medial UCL.

**Prognosis and return to sport:** A gentle throwing program should begin at approximately 6 weeks after surgery. Most throwing athletes return to previous level of competition within 3–4 months after surgery; patients with loose bodies or posterior impingement have better prognosis than those with degenerative changes within joint

## Olecranon Stress Fracture

**Description:** Stress injury across the proximal portion of the ulna

**Mechanism of injury:** Similar to valgus extension overload; repetitive abutment of olecranon into the olecranon fossa with repetitive traction from triceps contraction during deceleration phase of throwing and impaction of medial olecranon onto the medial olecranon fossa.

**Presentation:** Less common in adult throwers than adolescents and children; patients usually report insidious onset of pain in the posterior or posteromedial elbow.

**Physical examination:** Tenderness to palpation over the posterior/posteromedial olecranon; pain with forced passive elbow

extension and resisted extension; patients may demonstrate limited terminal extension

**Differential diagnosis:** Triceps tendonitis, olecranon bursitis, and valgus extension overload

**Diagnostics:** Plain radiograph: May show a transverse/oblique fracture line and/or sclerosis; MRI: May better delineate fracture patterns if not seen on radiographs; contralateral radiographs useful in skeletally immature patients because of variability in location of the olecranon physis

**Treatment:** Immediate cessation of throwing; may temporarily immobilize using a cast/splint; no return to sport until radiographic evidence of fracture healing and cessation of clinical symptoms (may take up to 6 months); patients failing to respond to conservative therapy may be considered for surgical treatment, consisting of compression screw fixation (oblique fractures) and tension band fixation (transverse fractures)

**Prognosis and return to sport:** Fractures typically heal with nonsurgical management or surgical fixation; athletes generally can return to sport within 3–6 months

### Olecranon Bursitis

**Description:** Inflammation of the bursa overlying the triceps tendon and olecranon of the ulna; can be acute or chronic and septic or aseptic (Fig. 50.3).

**Mechanism of injury:** Typically occurs as a result of direct trauma to the posterior elbow; may be secondary to a single direct blow or repetitive trauma; septic bursitis often occurs via direct traumatic inoculation; also associated with pre-existing systemic condition including immunosuppression, crystalline diseases, diabetes, and alcoholism

**Presentation:** Acute or gradual onset of bursal edema, erythema and tenderness in both septic and aseptic cases. While acute aseptic and septic cases may be painful, pain is more common throughout ROM in septic cases. Septic cases may demonstrate systemic symptoms.

**Physical examination:** Focal posterior elbow swelling; mobile, fluctuant mass that can wax and wane in size; may have associated lacerations/abrasions suggestive of septic bursitis; no restriction in ROM

**Differential diagnosis:** Gouty tophus, calcium pyrophosphate deposition, and cellulitis

**Diagnostics:** Radiographs should be obtained and may demonstrate bursal calcification or olecranon spurring; aspiration should be performed under sterile conditions if any concern for septic bursitis; fluid should be sent for cell count and differential, Gram stain/culture, glucose, and crystal analysis; *Staphylococcus aureus* most common cause of septic bursitis

**Figure 50.3.** Olecranon bursitis (student's elbow).

**Treatment:** Acute aseptic cases effectively treated with rest, short-term immobilization (3–5 days), compressive dressing, ice, and NSAIDs; chronic aseptic cases can be treated with aspiration and injection of corticosteroids, with a compressive dressing to be worn for 2–3 weeks; septic bursitis should be aspirated/excised with administration of culture-directed intravenous antibiotics (1–3 weeks), followed by 2 weeks of directed oral antibiotics; chronic aseptic cases can also be treated with excision of the bursal sac.

**Prognosis and return to sport:** Septic bursitis may be treated with needle aspiration and targeted antibiotics. In most cases, surgical intervention for septic bursitis does not provide long-term advantages in outcome; aseptic bursitis should be initially managed conservatively with NSAIDs, ice, and activity modification, which avoids elbow positions and directly compresses the bursa; patients nonsurgically treated or with aspiration are at a risk of recurrence; aseptic bursitis should not affect participation in sport (however, appropriate protective padding should be used upon return to sport); corticosteroid injections may be associated with an increased risk of septic bursitis and skin/fat atrophy; if conservative treatment fails, olecranon bursectomy (open or arthroscopic) may be considered, but it is associated with wound healing problems.

## MEDIAL ELBOW INJURIES

### Medial Epicondylitis

**Description:** Also known as "golfer's elbow"; flexor–pronator tendon degeneration with repetitive wrist extension and supination in activities requiring wrist flexion and pronation leading to angiofibrotic hyperplasia

**Mechanism of injury:** Repetitive eccentric loading of wrist flexors and forearm pronators; often combined with valgus overload at the elbow as in overhead throwing; common flexor tendon acts as a primary dynamic stabilizer of the elbow during overhead throwing and eccentrically contracts during ball release

**Presentation:** Often insidious onset of medial-sided elbow pain localized to medial epicondyle that may radiate to proximal forearm; exacerbated during late cocking/early acceleration phase of throwing/swinging; occurs less commonly than lateral epicondylitis; seen in overhead throwing athletes, golfers, bowlers, weightlifters, and football players; can be associated with ulnar neuritis/neuropathy

**Physical examination:** Usually full ROM; patients present with pain approximately 5–10 mm distal and volar to medial epicondyle with soft tissue swelling; symptoms exacerbated with resisted wrist flexion and forearm pronation in elbow extension; grip strength may be decreased compared to the contralateral side

**Differential diagnosis:** UCL sprain, flexor–pronator strain/tear, and ulnar neuritis

**Diagnostics:** Radiographs are typically normal but may demonstrate calcification of common flexor tendon; MRI is the best modality to identify pathologic changes in common flexor tendon; ultrasound is user dependent but may cost-effectively visualize tendinopathy

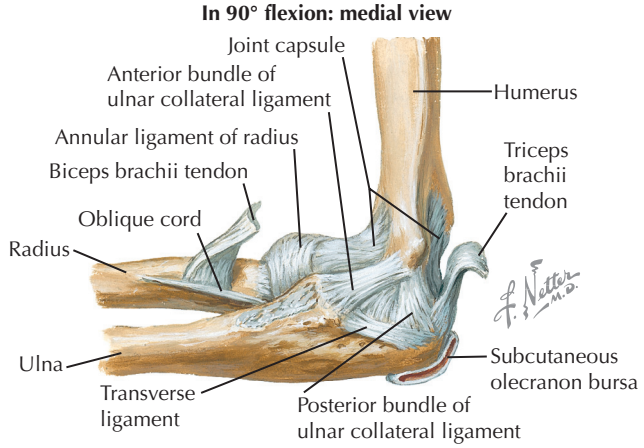
**Treatment:** Majority of patients will respond to conservative treatment consisting of activity modification, counterforce elbow bracing, NSAIDs, icing, and a physical therapy program focused on flexor–pronator stretching and strengthening after acute symptoms have resolved; corticosteroid and platelet-rich plasma injections may be considered in refractory cases; surgical intervention may be considered for those refractory to more conservative treatment and consists of either limited or extensive debridement of the common flexor tendon with either side-to-side tendon repair or reattachment to the medial epicondyle; concurrent procedures addressing the ulnar nerve or UCL should be performed as needed

**Prognosis and return to sport:** A majority of athletes who are nonsurgically managed return to sport when asymptomatic (typically a 6–12-week process); >90% exhibit good to excellent results with surgical intervention and return to sport within 3–6 months after surgery.

## Ulnar Collateral Ligament Injury

**Description:** Sprains, partial tears, or complete ruptures of the UCL (Fig. 50.4)

**Mechanism of injury:** Repetitive valgus stress (pitching, throwing, or racket sports) causes tensile loading of ulnar collateral ligament, resulting in ligament attenuation or tears



Milking maneuver valgus stress test

Moving valgus stress test

**Figure 50.4.** Ulnar collateral ligament sprain.

**Presentation:** Insidious medial elbow pain in late cocking and early acceleration phases of throwing; often asymptomatic at rest; throwing athletes often report decrease in velocity and accuracy. Pain returns when throwing exceeds approximately 75% of normal velocity. Patients with acute ruptures may describe acute pop that occurred while throwing; can have associated ulnar neuritis symptoms and valgus extension overload symptoms

**Physical examination:**

- Tenderness posterior to common flexor–tendon origin over UCL just distal to the tip of the medial epicondyle to the sublime tubercle
- Chronic tears may not demonstrate tenderness.
- Moving valgus stress test from 30 degrees of flexion (anterior band) to 90 degrees of flexion (entire UCL): abduct shoulder to 90 degrees in maximal external rotation with full flexion and valgus stress applied to the elbow, then quickly extending the elbow to 30 degrees
- Milking maneuver: abduct shoulder to 90 degrees, flex elbow to 90 degrees, and supinate forearm while pulling patients' thumb posteriorly to apply valgus load
- Valgus extension overload test: passive snapping of elbow into extension while maintaining valgus stress, indicates symptomatic posteromedial olecranon osteophyte (see Fig. 50.4)

**Differential diagnosis:** Ulnar neuritis, medial epicondylitis, flexor–pronator tendon rupture/muscle strain, and valgus extension overload

**Diagnostics:** Radiographs may demonstrate bony UCL avulsions and traction spurs or calcifications in UCL or posteromedial olecranon osteophytes. Bilateral valgus stress radiographs may demonstrate gapping indicative of ligament attenuation; dynamic stress ultrasound may better identify UCL integrity and joint laxity. MR arthrogram is the modality of choice to assess extent of UCL damage and to evaluate concurrent elbow injuries.

**Treatment:** Nonsurgical management may be first attempted consisting of throwing cessation and physical therapy focused on appropriate throwing mechanics, shoulder ROM, and core strengthening, followed by a graduated supervised throwing program. Complete ruptures in throwing athletes or partial ruptures in high-level athletes may be treated with UCL reconstruction, by using palmaris or gracilis autograft with concurrent treatment of additional elbow pathology

**Prognosis and return to sport:** Variable outcomes with nonsurgical management; typically, for a season-ending injury, return to competitive throwing usually 9–12 months after surgery with approximately 80% returning to the same or higher level of competition

## Medial Epicondyle Stress Lesions

**Description:** Also known as "little leaguer's elbow"; medial epicondyle apophyseal separation/fragmentation or avulsion of medial epicondyle apophysis and attached common flexor pronator tendon and UCL

**Mechanism of injury:** Generally seen in skeletally immature throwing athletes due to repetitive high tensile stress on the medial epicondyle apophysis from flexor–pronator mass and UCL; because physis is the weakest link in skeletally immature patients, it is unlikely to see UCL tears; correlated with excessive number of pitches thrown; acute trauma is less common cause

**Presentation:** Triad of symptoms: medial elbow pain with throwing, loss of throwing speed, and diminished throwing accuracy

**Physical examination:** Point tenderness at medial epicondyle; pain with valgus stress test without frank instability

**Differential diagnosis:** Ulnar collateral ligament injury, ulnar neuritis, medial epicondyle fracture, and flexor–pronator strain

**Diagnostics:** Bilateral radiographs should be obtained with the injured side demonstrating apophyseal widening or, less commonly, fragmentation; avulsion with displacement is rarely seen

**Treatment:** Physeal widening and minimally displaced fractures (<0.5–1 cm) are nonsurgically managed with brief immobilization (1–3 weeks), followed by ROM protecting against valgus force and resisted flexion/pronation until symptoms subside; avoid throwing for at least 3 months followed by progressive throwing program with focus on appropriate throwing mechanics; significantly displaced fractures (>1 cm) and fragments incarcerated in the joint must be surgically fixed.

**Prognosis and return to sport:** Full recovery is generally achieved with both nonsurgical and surgical management; recurrence is possible, particularly with poor throwing mechanics.

### Ulnar Nerve Compression Syndrome

**Description:** Also known as *cubital tunnel syndrome*; symptomatic dysfunction of the ulnar nerve at the level of the elbow due to compression, traction, and friction

**Mechanism of injury:** May be incited by trauma, cubitus valgus deformity, or subluxing ulnar nerve at the medial epicondyle; associated with medial epicondylitis; also seen in weightlifters concentrating on triceps strengthening

**Presentation:** Insidious onset of aching medial elbow/forearm pain and numbness/paresthesias of ring/small fingers with grip weakness; ROM not limited; often awakens patient at night as elbow is frequently flexed thereby decreasing space within cubital tunnel

**Physical examination:** Two-point discrimination diminished in small finger and ulnar aspect of ring finger: positive Tinel sign over cubital tunnel; positive ulnar nerve compression test; subluxation of ulnar nerve with elbow flexion; grip weakness; weak flexor digitorum profundus (FDP) to small finger; interossei, adductor pollicus, and ulnar lumbrical weakness (Wartenberg, Froment, and claw-hand deformities, respectively)

**Diagnostics:** Clinical diagnosis
EMG–NCS: slowing of conduction velocity across the elbow (may provide false negatives due to variable compression of fascicles); radiographs: usually normal but may rarely have osteophytes or cubitus valgus deformity

**Differential diagnosis:** Cervical radiculopathy, thoracic outlet syndrome, ulnar nerve compression at wrist (Guyon's canal-sensory disturbance on dorsal ulnar hand in distribution of dorsal cutaneous branch of ulnar nerve), ulnar collateral ligament injury, and medial epicondylitis (often comorbid)

**Treatment:** NSAIDs, modification of training (avoid triceps strengthening exercises), nighttime extension splinting, elbow pads, and avoidance of direct medial elbow pressure (nonsurgical measures are 58% effective); decompression with or without anterior transposition (subcutaneous, submuscular or intramuscular) and medial epicondylectomy; higher revision rate with in situ decompression but higher complication rate with transposition

**Prognosis and return to sport:** Dependent on severity and chronicity of neuropathy; if nonsurgically treated, may return to sport based on symptoms; if surgically treated, may return to full activity at approximately 4–6 weeks after surgery

## LATERAL ELBOW INJURIES

### Lateral Epicondylitis

**Description:** Also known as "tennis elbow"; chronic symptomatic degeneration of the forearm common extensor tendons near the lateral epicondyle; most commonly affecting the extensor carpi radialis brevis (ECRB) (Fig. 50.5)

**Mechanism of injury:** Excessive and repetitive contraction of wrist extensors leads to extensor tendon degeneration and angiofibrotic hyperplasia within tendon(s); late-stage disease may lead to partial/complete rupture and/or calcification within tendon

**Presentation:** Ten times more frequent than medial epicondylitis; pain with any movement that puts force on extended wrist; increased risk with frequent racket sports, poor technique, dominant arm, and age 35–55 years; initially pain subsides with rest in early stages of the disease process

**Physical examination:** Tenderness to palpation over the lateral epicondyle and/or ECRB; pain with resisted wrist and long-finger extension with the forearm in pronation; pain with resisted supination; diminished grip strength; "chair testv": pain with lifting chair with forearm pronated (see Fig. 50.5)

**Diagnostics:** Clinical diagnosis; radiographs are usually normal (late stages may demonstrate calcific changes within tendon) but may be helpful to rule out radiocapitellar arthrosis; MRI may show inflammation of the ECRB; ultrasound may identify structural changes in affected tendons; MRI and ultrasound do not necessarily correlate with symptoms

**Differential diagnosis:** Posterior interosseous branch of the radial nerve (PIN) entrapment (radial tunnel syndrome), radiocapitellar arthrosis, osteochondritis dissecans, and cervical radiculopathy

**Treatment:** Nonsurgical management with activity modification, NSAIDs, physical therapy with focus on eccentric stretching/strengthening, and counterforce bracing; corticosteroid or platelet-rich plasma injection if severely painful or if symptoms persist despite other conservative measures; surgical treatment rare and only in refractory cases: open or arthroscopic release of common extensor origin with lengthening or repair per surgeon preference; racket sport athletes should analyze stroke mechanics and grip size

**Prognosis and return to sport:** Nonsurgical treatment is successful in approximately 90% of cases (a 6–12-week course). Among cases that are surgically treated using various approaches, 85%–90% patients return to full activity by 6 months.

### Osteochondritis Dissecans Capitellum

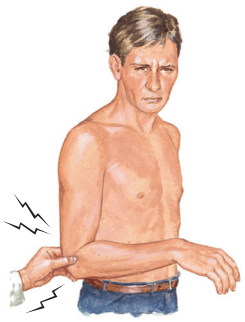
**Description:** Localized lesion involving a segment of articular cartilage and subchondral bone of the capitellum (Fig. 50.6)

**Mechanism of injury:** Repetitive microtrauma to capitellum from high valgus stresses leads to osteochondral injury; if overlying cartilage stable, underlying subchondral defect may fill in; unstable overlying cartilage likely leads to separation, fragmentation, and loose body formation without restoration of subchondral bone

**Presentation:** Insidious onset of activity-related lateral elbow pain in adolescents/young adults (age 11–21 years); consider Panner's disease in patients aged ≤10 years; more common in throwing sports and gymnastics, particularly in the dominant arm with throwers; may be bilateral in gymnasts. Pain resolves with rest early during the disease process. Pain with activities of daily living, mechanical symptoms, and stiffness occur in later stages.

**Physical examination:** Swelling and tenderness over radiocapitellar joint; occasional loss of terminal extension; positive radiocapitellar compression test with pronation/supination and axial load with elbow in extension; crepitus, clicking, and popping with ROM (suspect loose body)

**Diagnostics:** Radiographs: radiolucency and rarefaction of the capitellum, with flattening of the articular surface, possible sclerotic rim, possible fragmentation or loose bodies, obtain contralateral comparison views, 45-degree flexion, and oblique views; MRI: best for assessing extent of chondral damage; early low-signal changes on T1 images; T2 images helpful in visualizing intervening fluid in lesion consistent with fragment separation



**Epicondylitis (tennis elbow)** Exquisite tenderness over lateral or medial epicondyle of humerus

Resisted finger and wrist extension

Tenderness to palpation lateral epicondyle

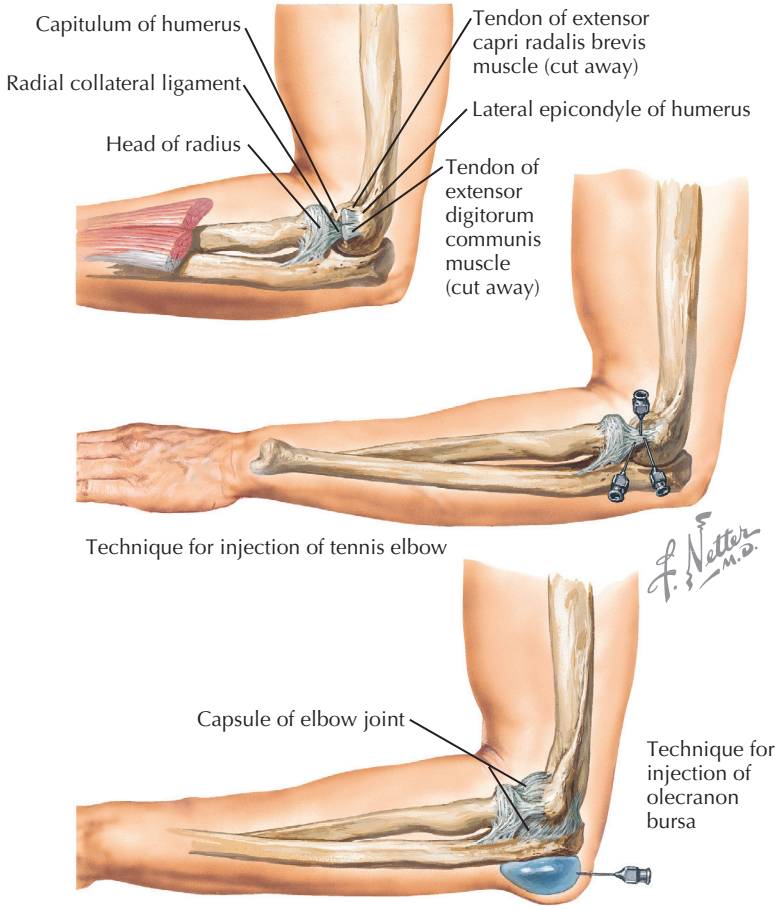



**Figure 50.5.** Tennis elbow.

**Differential diagnosis:** Panner's osteochondrosis, radiocapitellar chondrosis, and lateral epicondylitis

**Management:** For intact articular cartilage, period of rest with activity restriction along with physical therapy and gradual return to activities within 3–6 months; follow-up with serial radiographs. If fragment is displaced, recommended treatment is diagnostic arthroscopy, fixation of osteochondral fragment if intact and sufficiently large, excision of unstable articular fragment, and marrow stimulation of capitellar defect if fixation not possible; arthroscopy also indicated for loose bodies or continued pain despite conservative management; open procedure with possible osteochondral autograft for large lesions (>1 $cm^2$) or lesions violating the lateral column of the capitellum

**Prognosis and return to sport:** Early diagnosis and treatment are essential; for early lesions, athletes will miss remainder of the season, but long-term results are consistently good to excellent; for high-grade, unstable lesions, return to preinjury competition level is less likely, but long-term functional results are generally good, with occasional loss of terminal extension and mild pain with activities of daily living

## Posterior Interosseous Nerve (PIN) Compression Syndrome

**Description:** Also known as *radial tunnel syndrome*; compressive neuropathy of PIN causing pain only without motor or sensory dysfunction

**Mechanism of injury:** Compression of the PIN within one or multiple of the following locations: fibrous bands volar to radiocapitellar joint, radial recurrent vessels, medial edge of ECRB, arcade of Frohse (most common), or distal aspect of the supinator

**Presentation:** Aching lateral elbow pain from lateral epicondyle radiating into dorsoradial forearm; aggravated by pronation–supination activities and lifting objects; extensor weakness of wrist and fingers secondary to pain

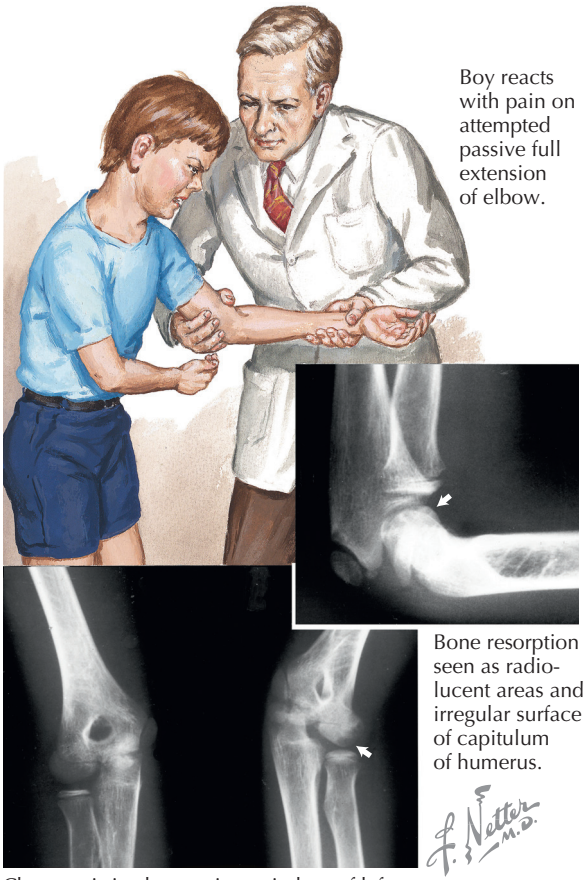
**Physical examination:** Tenderness to palpation over supinator arch (approximately 4 cm distal to lateral epicondyle); may have positive Tinel along course of PIN; pain with resisted long finger extension, pain with resisted supination in elbow/wrist extension, and pain with passive pronation in wrist flexion (passive stretch of supinator)

**Diagnostics:** Primarily a clinical diagnosis; radiographs: osteophyte at radiocapitellar joint (rare); MRI: may demonstrate late denervation/atrophy of supinator/extensors, may help identify sites of compression or rule out other pathology; diagnostic injection of local anesthetic into radial tunnel

**Differential diagnosis:** Lateral epicondylitis, C6/7 radiculopathy, extensor tendon rupture, distal PIN syndrome

**Treatment:** Rest, activity modification avoiding prolonged elbow extension with forearm pronation and wrist flexion, temporary dorsiflexion wrist splint, physical therapy for stretching, strengthening and nerve glides; corticosteroid injection in radial tunnel; surgical decompression of radial tunnel in recalcitrant cases after at least one year of nonsurgical treatment

**Prognosis and return to sport:** Nonsurgical management usually successful (80%); full return to sports usually by 4–8 weeks; surgical release also highly effective (80%); return to sport by 6–8 weeks



Boy reacts with pain on attempted passive full extension of elbow.

Bone resorption seen as radiolucent areas and irregular surface of capitulum of humerus.

Characteristic changes in capitulum of left humerus *(arrow)* compared with normal right elbow.

**Figure 50.6.** Osteochondritis dissecans capitellum.

## Dislocations and Fracture-Dislocations

**Description:** Fracture of the radius, ulna, or humerus, with or without dislocation or subluxation of the elbow joint (Fig. 50.7)

**Mechanism:** Usually a fall onto outstretched arm or onto the olecranon; may be associated with a high-energy traumatic event

**Presentation:** History of acute trauma; severe pain, exacerbated by subtle movement; and instability

**Physical examination:** Tenderness, deformity, swelling, and ecchymosis; limited and severely painful motion and crepitus; possible neurologic or vascular compromise; and varus/valgus instability

**Diagnostics:** Radiographs: fractures of radial head/neck, olecranon/ coronoid, and distal humerus; dislocation with or without fracture; proximal ulnar fracture with associated radial head dislocation (Monteggia fracture) and traction-view radiograph of elbow to delineate fracture pattern; CT can be useful to assess the fracture pattern and degree of comminution; MRI to assess ligamentous/chondral damage following acute fracture care

**Differential diagnosis:** Associated ligamentous injury

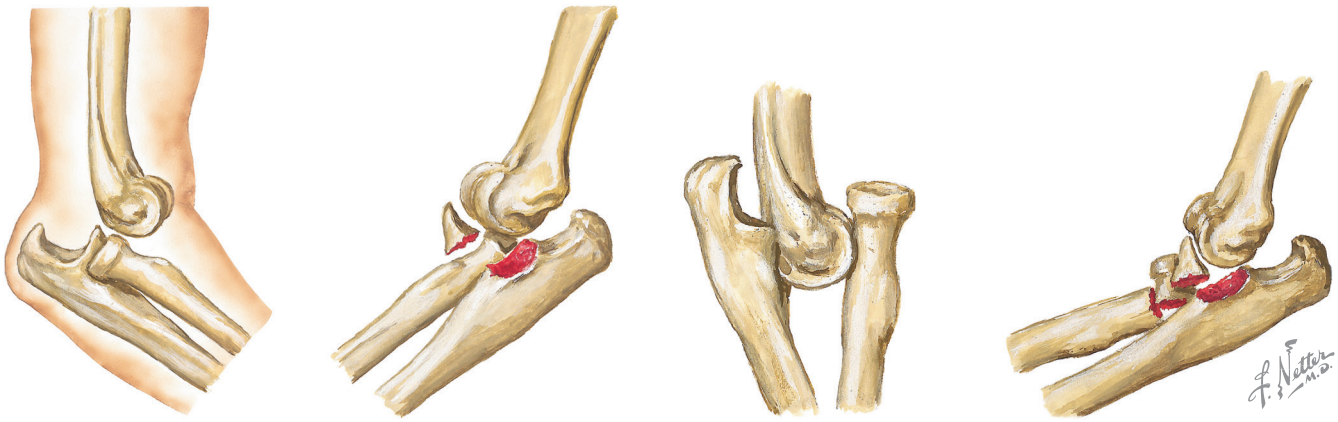
**Treatment:** Document neurovascular examination, if compromised, attempt reduction immediately; immobilize with splint; refer urgently to emergency department for evaluation and further treatment as indicated (closed reduction, open reduction and internal fixation, and staged ligamentous repair)

**Prognosis and return to sport:** Usually a season-ending injury: extended course of treatment and rehab; simple dislocations usually have good outcomes with early ROM; return to previous level of sports unlikely after complex fracture/dislocations

## Posterolateral Rotatory Instability (PLRI)

**Description:** Disruption of the lateral ligamentous complex leading to instability of the radiocapitellar and ulnar humeral joint

**Mechanism:** Initial injury often from a fall onto outstretched arm with axial load and forearm supination resulting in posterolateral dislocation or subluxation; this causes injury to the lateral ligament complex; may also be iatrogenic from surgery to the lateral elbow



Posterior dislocation. Note prominence of olecranon posteriorly and distal humerus anteriorly.

Fracture of coronoid process of ulna with posterior dislocation of elbow. Coronoid fracture may occur occasionally without dislocation.

Divergent dislocation, anterior-posterior type (rare). Medial-lateral type may also occur (extremely rare).

Posterior dislocation with fracture of both coronoid process and radial head. Rare but serious; poor outcome even with good treatment. May require total elbow replacement.

**Figure 50.7.** Dislocations of the elbow.

**Presentation:** Lateral elbow pain common; feeling of instability with axial load and forearm supination (pushing out of a chair); instability may manifest as a mechanical catch or pop with elbow extension

**Physical examination:** Tenderness, deformity, swelling, and ecchymosis in the acute setting (less common chronic injuries); may have varus laxity; apprehension or visible shifting of the elbow with valgus stress while moving the elbow from flexion to extension (apprehension test/lateral pivot shift test); apprehension with a chair-rise test: pain or instability reproduced as patient tries to push themselves out of a chair with their hands

**Diagnostics:** Radiographs to look for obvious fractures of radial head/neck, olecranon/ coronoid, and distal humerus; lateral radiograph is critical to evaluate for posterior radial head subluxation with the elbow extended and the forearm in supination; AP radiograph to evaluate for lateral ulnohumeral gapping; MRI following acute injury if the elbow is grossly unstable or if there is concern for an osteochondral injury

**Differential diagnosis:** Loose body causing mechanical symptoms

**Treatment:** For an acute injury, use radiographs to evaluate stability of the elbow in extension with forearm pronation and supination. If stable in extension with forearm pronation, splint in pronation for 2 weeks at 90 degrees; start early motion with the forearm in pronation. Allow supination with elbow flexion above 90 degrees. After 6 weeks, allow supination in extension if the radiocapitellar articulation remains concentric. Early repair is indicated for an acute elbow injury that is not stable with elbow flexion and pronation. Lateral UCL reconstruction is indicated for chronic posterolateral rotatory instability (PLRI) or if the elbow does not remain reduced in extension after an attempt at nonsurgical treatment.

**Prognosis and return to sport:** Usually a season-ending injury; extended course of treatment and rehabilitation; 4–6 months for return to sport depending on the demands of the sport

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# HAND AND WRIST INJURIES

# 51

Jeffry T. Watson • Douglas R. Weikert • Nathan van Zeeland

## GENERAL PRINCIPLES

### Overview

- Fortunately, most sports-related hand and wrist injuries, when addressed in a timely manner, do not represent a significant threat to limb viability, long-term function, or eventual return to sports.
- Perhaps the greatest morbidity from these injuries results from delayed presentations or missed injuries.
- Hand function is closely linked to full flexion of the ulnar three digits, prehension grip in the radial three digits, and a stable, mobile wrist.

### Physical Examination

- Dictated by the context of the injury. No single, comprehensive evaluation applies to all maladies.
- Attention is directed toward the individual part and system (bone, joint, tendon, nerve, etc.) in question.

### Observation/Inspection

- Focal swelling, digital perfusion, digital malrotation, digital cascade, and any penetrating injury must be noted.
- Any difference in posture of one digit relative to the others should not be dismissed or minimized because this often signifies a displaced fracture, tendon avulsion, or joint subluxation.
- In the absence of a penetrating injury, isolated pallor of a digit usually represents spasm of the digital vessels. Although this often resolves with digital warming or reduction of associated displaced fractures or dislocations, digital viability remains in question until perfusion is actually observed.
- Even in closed fractures, the digital vessels can tear or thrombose, representing a surgical emergency.
- Dorsal swelling of the hand is a nonspecific finding and may not represent a significant injury.

### Palpation

- Careful palpation of specific bones or ligaments in question is fundamental to assessment. There are not many "referred-pain" injuries in the hand or wrist—tenderness to palpation does not lie.
- Focused palpation will usually localize the injured structure within an area of generalized edema demonstrating diffuse swelling; for example, a swollen wrist following distal radius fracture or perilunate injury
- Tenderness over a scapholunate ligament, even in the setting of normal radiographs, will suggest an underlying ligament tear that would have poor prognosis if not recognized and treated.

## SPECIFIC INJURIES AND CONDITIONS

### Nail Bed Injury

**Description:** Any tear or disruption of the sterile or germinal matrix of the nail bed; may or may not be associated with an underlying distal phalanx fracture or actual disruption of the nail plate (Fig. 51.1)

**Mechanism of injury:** Usually caused by dorsal crush of the fingertip (such as when the fingertip is crushed by another player's cleated shoe); however, may also occur with axial load to the fingertip that results in flexion fracture of the distal phalangeal shaft and tear of the overlying nail bed

**Presentation:** If the nail bed is disrupted, subungual hematoma results. The tear may extend peripherally beyond the borders of the nail fold into the surrounding skin (see Fig. 51.1).

**Physical examination:** Gross instability of the fingertip with nail bed injury is suggestive of a concomitant distal phalangeal shaft fracture.

**Differential diagnosis:** If the base of the nail plate is flipped out dorsally over the nail fold, consider an open fracture of the proximal portion of the phalanx. In skeletally immature patients, the presence of the physis at this location may result in failure to recognize what is, in fact, an open Salter–Harris I phalangeal fracture.

**Diagnostics:** Physical examination is usually sufficient. Anteroposterior (AP) and lateral radiographs reveal an underlying distal phalanx fracture.

**Treatment:** For small subungual hematoma encompassing a portion of the nail plate, no intervention is necessary. Decompression of the hematoma through needle fenestration of the nail plate can offer pain relief; however, this may increase the likelihood of wound sepsis if performed on a playing field (see Fig. 51.1). For **larger hematomas** (50% of the nail plate) with tearing beyond the nail fold borders, formal repair is recommended. Under a digital block anesthetic, the nail plate should be removed, the wound irrigated, and the nail matrix repaired with either a topical skin adhesive or a 7-0 resorbable suture. The adjacent skin rip is repaired with a 5-0 nylon suture. An underlying phalangeal tuft fracture is nonsurgically managed. However, the associated unstable distal phalangeal shaft requires washout and perhaps pin stabilization.

**Prognosis and return to play:** If no nail bed repair is required, immediate return to play is alright. Following nail bed repair, the fingertip (including distal interphalangeal joint [DIP]) should be dressed and splinted to protect from impact. If there is an associated unstable phalangeal shaft fracture requiring pin fixation, return to play should be delayed until pin removal. Prognosis for nail plate growth is directly related to anatomical restoration of the nail bed. If there is a wide scar in the matrix, a ridge or split in the nail plate will occur. Open fractures through the nailbed require urgent surgical debridement in the operating room. Displaced distal phalangeal shaft fractures are often unstable and require pinning.

### Mallet Finger

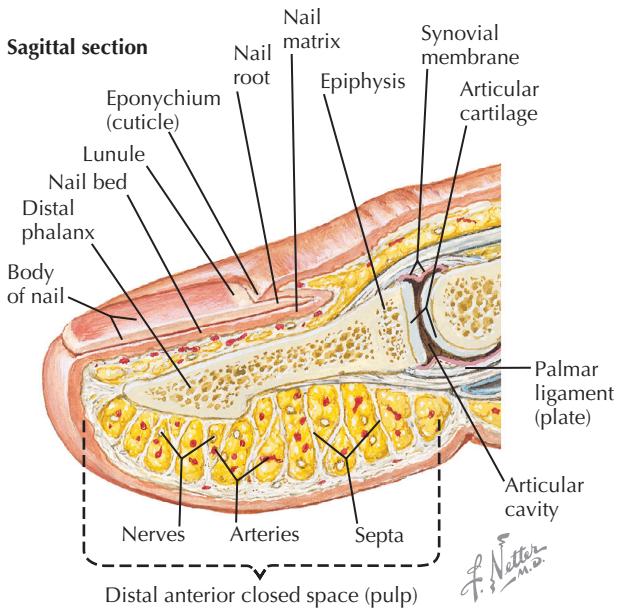
**Description:** Loss of terminal extensor mechanism attachment to the distal phalanx with resultant flexion deformity of the DIP joint

**Mechanism of injury:** Sudden forced flexion of the DIP joint during active extension through the terminal tendon, often as a result of a ball jamming the fingertip (Fig. 51.2)

**Presentation:** The DIP joint is maintained in flexion with an inability to actively bring the joint into full extension. Varying degrees of pain, often with minimal or no pain; swelling or ecchymosis may be noted over the dorsal aspect of the joint.

**Differential diagnosis:** Distal phalanx fracture or DIP dislocation

**Diagnostics:** Posteroanterior (PA) and lateral radiographs of a digit to assess if injury is limited to the soft tissue (tendon only) or has associated bony avulsion (see Fig. 51.2). Lateral



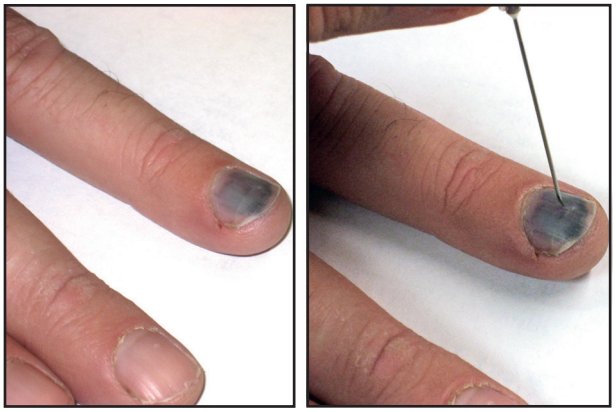
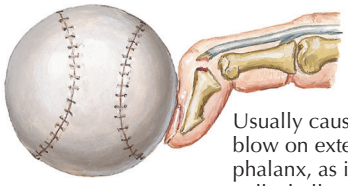
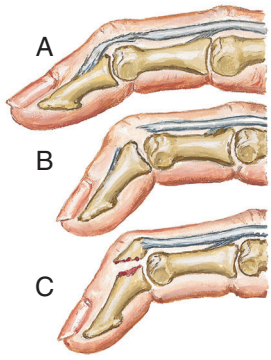




**Figure 51.1.** Nail bed injury.

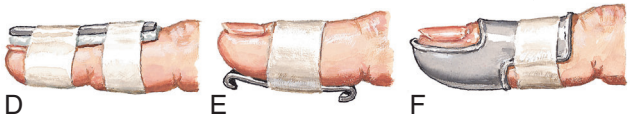
**Mallet finger of bone origin.** Avulsion of bone fragment and volar subluxation of distal phalanx



Usually caused by direct blow on extended distal phalanx, as in baseball, volleyball




**Degrees of mallet finger injury.** **A**. Extensor tendon stretched but not completely severed; mild finger drop and weak extensor ability retained. **B.** Tendon torn from its insertion. **C.** Bone fragment avulsed with tendon. In B and C there is 40° to 45° flexion deformity and loss of active extension.




**Treatment for mallet finger of tendon origin. D.** Padded dorsal splint. **E.** Unpadded volar splint. **F.** Stack splint. Proximal interphalangeal joint left free for active exercise.

**Figure 51.2.** Mallet finger.

radiographs determine stability based on the size of the bony component and whether palmar subluxation is present.

**Treatment:** Acute mallet injuries with no subluxation on the lateral radiographs require *full-time* splinting of the DIP joint in extension for 6–8 weeks (see Fig. 51.2). Displaced bony components with joint involvement of >30% and/or palmar subluxation often require surgery to restore joint congruity. The DIP joint will usually require transarticular pinning in full extension with surgery. Primary surgical repair of acute, closed, soft-tissue mallet injuries has not proven to be superior and may have more significant complications.

**Prognosis and return to play:** Noncompliance with splint wear will negatively impact the outcome and usually results in mild-to-moderate degrees of extension lag. The functional effect of this is variable. Most athletes nonsurgically treated will return to play within a week (while splinted). With surgical treatment, return to play will depend on the athlete's ability to protect a transarticular pin. Any activity requiring grasp is likely to result in bending or breakage of the pin.

## Jersey Finger

**Description:** Traumatic avulsion of flexor digitorum profundus (FDP) from distal phalanx (Fig. 51.3); tendon may detach alone or avulse a palmar fragment of the distal phalanx; ring finger most commonly affected

**Mechanism of injury:** Forced passive extension of DIP joint during active flexion of DIP joint

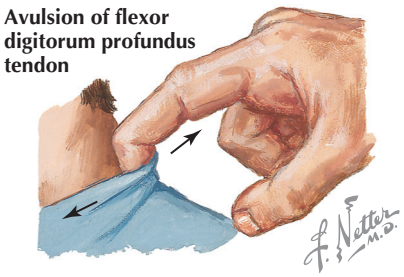
**Presentation:** Typically seen in football and rugby players attempting to grab a jersey; variable degree of pain, although the player may complain of pain proximally in the finger or palm at the level of the retracted tendon. Usual concern is inability to flex the involved DIP joint. Unfortunately, many of these cases are delayed presentations.

**Physical examination:** Ecchymosis may be present at DIP joint, depending on timing of presentation. The flexor tendon stump may be tender or palpable in the palm or along the digit, depending on the level of proximal retraction. Bony avulsions tend to become incarcerated along the flexor sheath (often at the A4 pulley over the middle phalanx). Loss of active DIP joint flexion is the most specific finding.

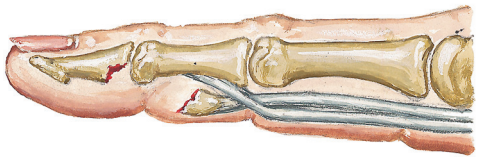
**Differential diagnosis:** Distal phalanx fracture and DIP joint dislocation

**Diagnostics:** PA and lateral radiographs of injured digit to check for bone avulsion fragment; ultrasound may identify a retracted tendon rupture.

**Treatment:** Surgical reattachment of the flexor tendon within 7–10 days if the tendon has retracted into the palm; if tendon has retracted only to the proximal interphalangeal (PIP) level, reattachment may be successful with a delay of up to a few

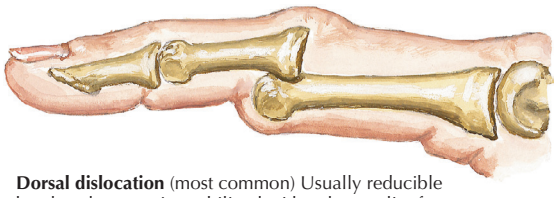



Caused by violent traction on flexed distal phalanx, as in catching on jersey of running football player

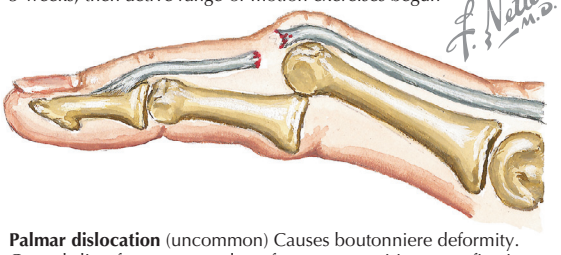


Flexor digitorum profundus tendon may be torn directly from distal phalanx or may avulse small or large bone fragment. Tendon usually retracts to about level of proximal interphalangeal joint, where it is stopped at its passage through flexor digitorum superficialis tendon; occasionally, it retracts into palm. Early open repair of tendon and its torn fibrous sheath indicated.

**Figure 51.3.** Jersey finger.



**Dorsal dislocation** (most common) Usually reducible by closed means, immobilized with palmar splint for 3 weeks, then active range-of-motion exercises begun




**Palmar dislocation** (uncommon) Causes boutonniere deformity. Central slip of extensor tendon often torn, requiring open fixation, followed by dorsal splinting to allow passive and active exercises of distal interphalangeal joint

Lateral radiograph of persistent PIP subluxation due to volvar fracture fragment

**Figure 51.4.** Proximal interphalangeal (PIP) joint dislocation.

weeks. For flexor tendon avulsions with bony component, internal fixation is necessary to restore continuity of the flexor tendon.

**Prognosis and return to play:** Soft tissue FDP avulsions require 12 weeks of protected activity before return to full gripping and grasping activities. Bony avulsions amenable to open reduction and internal fixation (ORIF) require 4–6 weeks of protected activity. Both types of FDP avulsions also require extensive hand therapy after surgery.

## Proximal Interphalangeal (PIP) Joint Dislocation and Fracture-Dislocation

**Description:** Usually, the middle phalanx displaces dorsal to the proximal phalanx. However, rotatory (with the proximal phalanx condyle protruding between the lateral band and central slip), volar, and lateral dislocations, though less frequent, do occur. With dorsal dislocation, fracture often occurs at middle phalangeal base (Fig. 51.4). Direct axial load, however, may result in a comminuted pilon fracture of the entire articular surface and metaphysis.

**Mechanism of injury:** Usually, hyperextension of PIP joint with varying degrees of axial loading; often occurs from a ball, another participant, or ground jamming into the finger.

**Presentation:** Usually with pain and swelling around PIP joint with or without angular deformity; patient will be apprehensive to active or passive motion

**Physical examination:** Pain localized to PIP joint with swelling; collateral ligaments will be tender because they are disrupted; occasionally will have skin laceration or a palmar skin tear.

**Differential diagnosis:** Volar plate injury without dislocation; phalangeal, articular, or periarticular fracture

**Diagnostics:** PA and lateral radiographs of the injured digit *must* be performed to verify congruent reduction and rule out fracture or subluxation but can be delayed for a few days if "on the field" reduction is clinically stable. If the initial reduction attempts seem unsuccessful, radiographs should be obtained before repeated efforts. Reduction may be impeded from a fracture component or different orientation of dislocation (see Fig. 51.4).

**Treatment:** Digital blocks with 1% lidocaine without epinephrine may be helpful. Closed reduction employing longitudinal traction, slight extension, and dorsal pressure over middle phalanx for dorsal dislocations. After reduction, range of motion (ROM) and joint stability must be evaluated. **Radiographic or fluoroscopic confirmation of reduction is required within a few days.** Without a significant periarticular fracture, instability requiring surgery is unlikely. For **rotatory dislocation,** manipulation with the metacarpophalangeal (MCP) and PIP in flexed position facilitates reduction. **Volar dislocation** is reduced with slight PIP flexion and dorsal translation of the middle phalangeal base. Following reduction of volar dislocation, it is *crucial* to protect the central extensor slip insertion (which generally is always disrupted in volar dislocation) with

immobilization of PIP in full extension in order to avoid inevitable progression to boutonniere deformity. For **fracture-dislocations**, closed reduction with longitudinal traction and subsequent dorsal blocking splint to hold the flexed PIP joint may be adequate, depending on size of fractured palmar joint margin. In general, if fracture involves <40% of the articular surface, this technique is useful. The joint is gradually moved into greater degrees of extension with weekly radiographic verification of maintained reduction over the ensuing 4–6 weeks. Fractures with persistent dorsal subluxation after closed reduction require surgical stabilization.

**Prognosis and return to play:** Return to play is usually minutes after closed reduction of dorsal dislocations. Buddy taping to adjacent digits or aluminum extension block splinting should suffice; **early follow-up radiographs are mandatory**. Athletes with stable reduction of dorsal dislocation without associated fracture can continue playing most sports, avoiding forced passive hyperextension for initial 3 weeks. Volar dislocations need to be splinted in full extension for approximately 6 weeks to protect the central slip. During this time, active DIP motion is employed to promote gliding of lateral bands. Swelling and stiffness may persist for several months. Open surgical treatment of fracture-dislocations requires no forceful grip or impact for 4–6 weeks and usually results in some loss of PIP motion.

## Metacarpal Fracture

**Description:** Fracture of metacarpal neck, shaft, or base

**Mechanism of injury:** Axial load or clenched fist impact are common mechanisms for distal or proximal metaphyseal fractures. Direct dorsal impact (such as baseball striking batter's hand or another participant stepping on hand) often results in shaft fractures (Fig. 51.5).

**Presentation:** Localized swelling, with or without angular deformity of digits

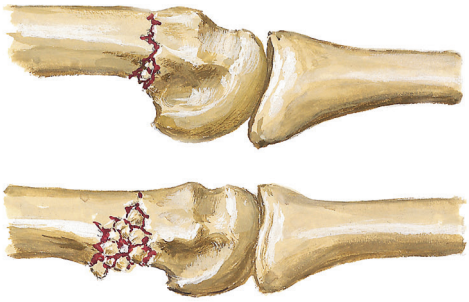
**Physical examination:** Point tender over metacarpal fracture with swelling; angular and sometimes rotational deformity (scissoring) of digits (see Fig. 51.5). Scissoring is more easily detected if patient is able to offer a certain degree of digital flexion.

**Differential diagnosis:** Contusion or MCP joint dislocation

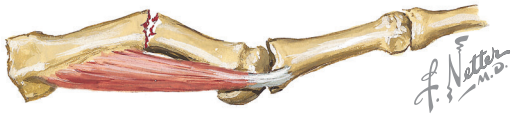
**Diagnostics:** PA, lateral, and oblique radiographs of the hand

**Treatment:** Surgical treatment for fractures with angulation of >50 degrees of ring and small fingers and angulation of >20 degrees of index and middle fingers. Shortening of >5 mm and shaft fractures of the border digits (small and index) may also need ORIF. Significant rotational deformity/scissoring also is an indication for ORIF. Most cases are nonsurgically treated with splint spanning wrist and hand for 3–4 weeks. Reduction of metacarpal neck fractures is practically difficult to maintain using a splint or cast. Pinning is usually required, often leading to joint stiffness; hence, most metacarpal neck fractures are allowed to heal with some flexion deformity as long as there is no rotational component or associated loss of PIP flexion.

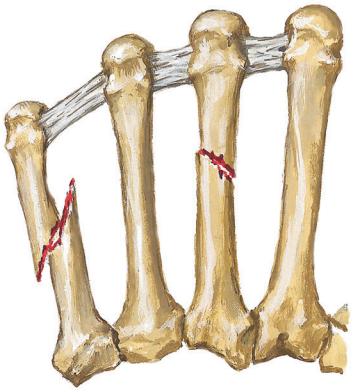
**Prognosis and return to play:** Stable fractures not requiring surgery usually require 3–4 weeks of splinting while swelling and soreness subsides. Transition to hand-based splints may allow skilled position players to return within 1–2 weeks depending on level of discomfort. Surgically treated fractures require 2–4 weeks before ROM and pain allow return to sports.



In fractures of metacarpal neck, volar cortex often comminuted, resulting in marked instability after reduction, which often necessitates pinning




Transverse fractures of metacarpal shaft usually angulated dorsally by pull of interosseous muscles.



Oblique fractures tend to shorten and rotate metacarpal, particularly in index and little fingers because metacarpals of middle and ring fingers are stabilized by deep transverse metacarpal ligaments.

Clinical scissoring due to malrotation of 4th and 5th metacarpal fractures

**Figure 51.5.** Metacarpal fracture.

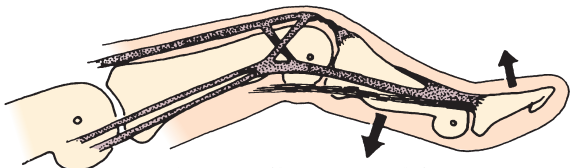
## Injuries to the PIP Joint Central Extensor Slip Insertion/Boutonniere Deformity

**Description:** Central slip of the extensor tendon inserts on the dorsal base of the middle phalanx. Disruption of this insertion results in loss of full active PIP joint extension. Over time, adjacent lateral band tendons migrate and become fixed palmar to the axis of rotation of the PIP joint, resulting in a boutonniere deformity characterized by PIP flexion and DIP hyperextension (Fig. 51.6).

**Mechanism of injury:** May occur with forced passive PIP flexion against active extension through the central slip tendon, resulting in avulsion; volar PIP dislocations often result in avulsion of the central slip insertion; dorsal PIP laceration through the central slip (hockey skate) will result in boutonniere deformity if left untreated.

**Presentation:** Often has a subtle presentation and a high index of suspicion is required. Nonspecific presence of swelling is usually present about the PIP joint, and the joint may be maintained in slight flexion (see Fig. 51.6). However, in the immediate phase, the patient may be able to maintain PIP extension through the lateral bands, which have not yet palmarly migrated.

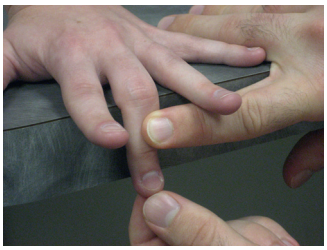
**Physical examination:** Palpate for tenderness directly over the central slip insertion on the dorsal middle phalangeal base. Collateral ligament tenderness may also be present, but tenderness at the central slip insertion should raise concern. A sensitive method to assess disruption of the central slip is the Elson test (see Fig. 51.6). A digit is placed on a table with the PIP joint flexed over the edge. While the proximal phalanx is held firmly flat on the table by the examiner, the patient attempts active



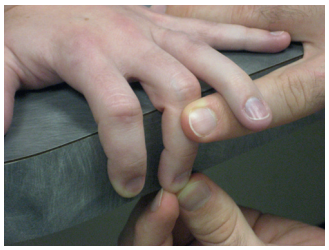
Schematic of boutonniere deformity following central slip disruption

Swelling following closed central slip disruption

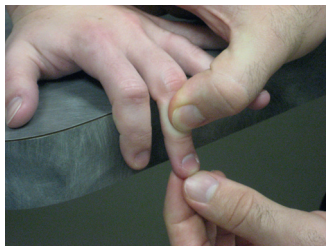
**Elson test**



PIP flexed over table edge, patient instructed to actively extend PIP.



While examiner resists active extension, DIP can be passively flexed without resistance.



If central slip is avulsed, the DIP cannot be passively flexed and may even extend slightly with resisted active PIP extension (positive Elson test).

**Figure 51.6.** Injuries to the PIP joint central extensor slip insertion/boutonniere deformity. (From Green D, Hotchkiss R, Pederson W, eds. *Green's Operative Hand Surgery*, 5th ed. Philadelphia: Churchill Livingstone, Elsevier; 2005.)

extension of the PIP joint. Any pressure felt by the examiner on the dorsum of the middle phalanx suggests some continuity of the central slip insertion. If the central slip has torn and retracted proximally, there will be loss of active PIP extension as well as reduced passive DIP flexion (normally floppy and supple) during the attempt.

**Differential diagnosis:** Nonspecific swelling around PIP joint could represent anything from mild collateral ligament injuries to periarticular fractures.

**Diagnostics:** PA and lateral plain radiographs are needed to rule out periarticular fractures or avulsion of dorsal margin of middle phalangeal base.

**Treatment:** For closed injuries noted early (within 2–3 weeks), the lateral bands may not have yet become fixed in a position palmar to the axis of rotation. Closed treatment with *full-time* PIP splinting in full extension and active DIP flexion/extension exercises often results in healing of the central slip to its insertion bed while preserving lateral band mobility; should be continued for 6–8 weeks. Open lacerations require primary surgical repair of the tendon followed by protection of the repair with full PIP extension splinting and active DIP motion as discussed earlier. Delayed presentations with fixed boutonniere postures are exceedingly difficult to treat. Salvage procedures in the form of terminal extensor tendon releases and even PIP fusion may be required depending on the degree and rigidity of the contracture.

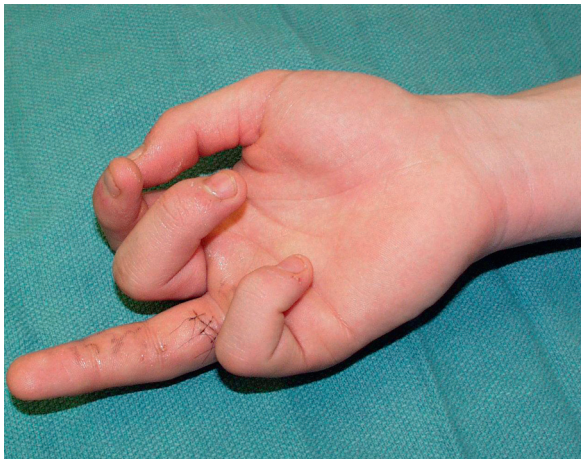
**Prognosis and return to play:** Primary prognostic factors are prompt diagnosis and initiation of closed treatment. The digit must be protected from PIP flexion for at least 6 weeks. Fixed boutonniere deformities have a poor prognosis in terms of regaining full active motion.

## Flexor Tendon Laceration

**Description:** Transection of flexor tendon at wrist, hand, or finger

**Mechanism of injury:** Laceration to palmar aspect of wrist, hand, or digit, often from cleat, spikes, or blade of skate

**Presentation:** Skin laceration with inability to flex digit(s) distally (Fig. 51.7); resting cascade of digit in question will demonstrate less resting flexion of the DIP or PIP joint than surrounding digits



Typical presentation of lacerated FDS and FDP tendons in finger

**Figure 51.7.** Flexor tendon laceration.

**Physical examination:** Laceration with no active flexion of digit at DIP and possibly PIP joint; neurovascular status may also be abnormal (digit pale, diminished capillary refill) if digital neurovascular bundles are also lacerated. Two-point sensory discrimination will be altered with transaction of the digital nerve.

**Differential diagnosis:** Open fracture or simple laceration

**Diagnostics:** PA and lateral radiographs of injured area

**Treatment:** Irrigation and dressing of wound with skin closure; tendons require surgical repair within 10 days; no role for nonsurgical treatment

**Prognosis and return to play:** Flexor tendon rehabilitation requires 6–8 weeks of extensive ROM therapy and up to 12 weeks for strengthening before returning to sports that require gripping and grasping.

## Phalangeal Fractures

**Description:** May occur as simple transverse patterns with minimal displacement or as more complicated, comminuted patterns

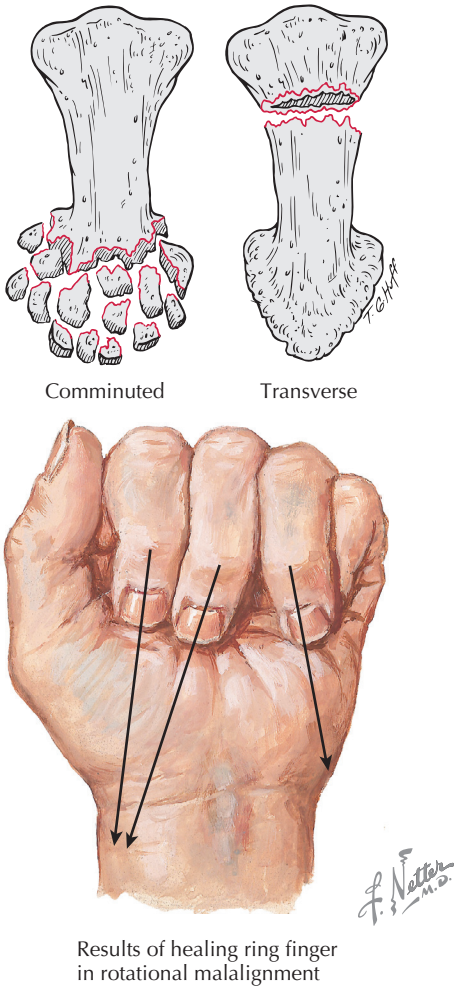



**Figure 51.8.** Phalangeal fractures. (From Browner B, Jupiter J, Levine A, Trafton P. Skeletal trauma. *Basic Science, Management, and Reconstruction*, 3rd ed. Philadelphia: Saunders, Elsevier; 2002.)

with marked displacement and associated soft tissue injury; in the distal phalanx, tuft fractures occur in the very tip of the bone, usually resulting from some form of crush to the fingertip, and often have a stellate pattern; distal phalangeal shaft and base fractures may result from a crush or bending force, and can be more unstable than tuft fractures (Fig. 51.8).

**Mechanism of injury:** Excessive axial, torsional, or bending forces; rate and direction of loading will determine the fracture pattern; sudden axial load from a ball to crush from another competitor's foot can result in various types of fractures, such as periarticular fractures or fracture-dislocations

**Presentation:** Nondisplaced fracture—pain, swelling, and associated apprehension to movement; fractures from crush injuries will often have a concomitant soft tissue injury component. Tuft fractures or displaced shaft fractures usually present with a subungual hematoma. Fractures with more comminution or displacement will likely result in some angular deformity of the digit. Be careful of any open wound around a displaced phalangeal shaft fracture because this may communicate with the fracture site.

**Physical examination:** Inspect soft tissues, checking for open wounds or subungual hematoma. If the base of the nail plate has flipped out of the nail fold, this frequently represents a distal phalangeal shaft or metaphyseal open fracture through the nail bed. Point tenderness to palpation over the phalanx should raise suspicion. Angulation likely represents a fracture or fracture-dislocation, more easily detected if the patient can flex the digits (see Fig. 51.8). Open wound over the radial or ulnar neurovascular bundle mandates two-point discrimination sensory testing along that side of the fingertip. Perfusion of the digit should also be verified in settings of injuries with more energy imparted or displacement (crush injuries).

**Differential diagnosis:** Dislocations of the MCP or interphalangeal (IP) joints may be mistaken for phalangeal fractures; clarified using plain radiographs

**Diagnostics:** PA and lateral plain radiographs of the digit should suffice for determination of fracture pattern and differentiation from joint involvement.

**Treatment:** **Potentially open fracture or nonperfused digit** requires emergent surgical treatment and withdrawal from competition. Otherwise, the injured digit may be bandaged in a bulky dressing together with the other digits to allow return to play. Formal radiographic and clinical evaluation of closed perfused injuries should be performed within 48 hours. **Nondisplaced stable fractures** can usually be treated with custom splinting and gentle active assist ROM exercises over 2–3 weeks. **Displaced fractures** must be reduced; digital block anesthetic is usually sufficient. Anything other than complete anatomic reduction of a transverse fracture is unstable and will require pin or plate and screw fixation. Tuft fractures can be managed without splinting, which allows load bearing as tenderness subsides.

**Prognosis and return to play:** Primary complications usually include stiffness and angular deformity. Return to play is dictated by fracture stability (or fixation rigidity), union, and participation requirements. If fixation requires pin placement across a joint, the athlete should not be allowed to participate in any sport requiring forceful grip or ball handling until the pin has been removed. Similarly, even the most stable plate and screw constructs are prone to failure under such loads, and return to activity before at least early callus formation after the third week risks fixation failure and deformity.

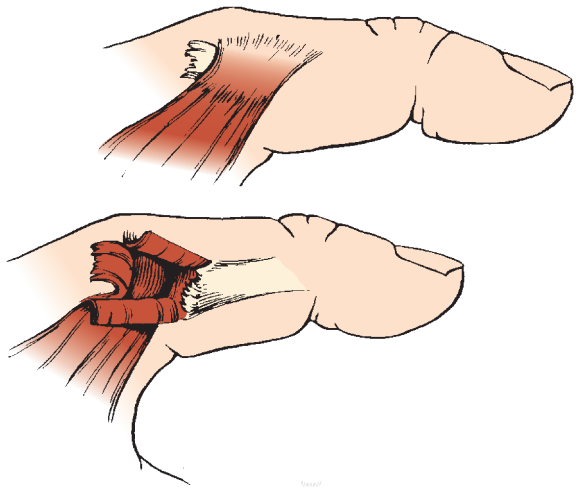
## Thumb Metacarpophalangeal Joint Ligament Injuries

**Description:** Often referred to as "skier's" or "gamekeeper's" thumb (for ulnar collateral ligament [UCL] failure) and "reverse gamekeeper's" thumb (for radial collateral ligament [RCL] failure), these injuries can result in chronically impaired and painful grip when left untreated. UCL failure can occur anywhere along the length of the ligament; detachment usually occurs with or without avulsion fracture at the site of insertion onto the base of the proximal phalanx (Fig. 51.9). The tendon or aponeurosis of the adductor pollicis can become interposed between the torn ligament and its insertion on the phalanx, thus preventing healing; this is called a *Stener lesion* (see Fig. 51.9).

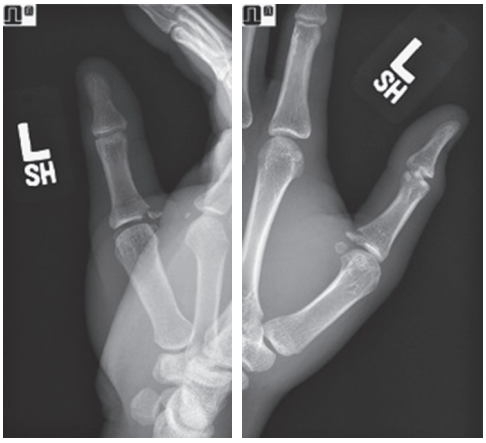
**Mechanism of injury:** UCL injuries (10 times more frequent than RCL injuries) result from sudden valgus force to the thumb, often after a fall onto the thumb or impact from a ball. RCL injuries result from sudden varus force to the thumb and are often underappreciated and underdiagnosed.

**Presentation:** Painful, impaired grip; usually, the MP joint is diffusely swollen in the subacute phase; resting angular deformity may or may not be visible at the MP joint. In RCL injuries, as swelling subsides, metacarpal head may appear prominent because of "sagging" of the radial aspect of the joint from capsuloligamentous incompetence.

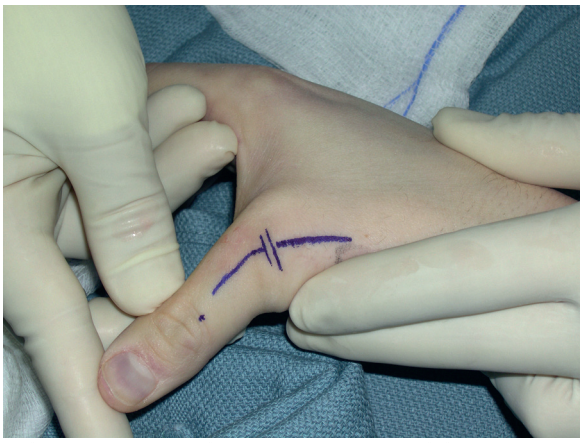
**Physical examination:** In acute and subacute phases, tenderness to direct palpation over the injured ligament is present. Assess UCL stability with passive valgus stress of the extended proximal phalanx while stabilizing the metacarpal with the other hand



Schematic of Stener lesion




Radiograph showing UCL avulsion fracture



Valgus MP stress test demonstrating UCL instability

**Figure 51.9.** Thumb metacarpophalangeal (MCP) joint ligament injuries. (From DeLee J, Drez D, Miller M. *DeLee & Drez's Orthopaedic Sports Medicine: Principles and Practice*. 2nd ed. Philadelphia: Saunders, Elsevier; 2002.)

(see Fig. 51.9). Although it has been suggested that pain and deviation of >30 degrees are indicative of a UCL tear, this laxity should be compared with the contralateral uninjured thumb. Several patients with inherent ligamentous laxity will have that degree of mobility in that joint; repeating the maneuver with the phalanx in 30 degrees of flexion will isolate the UCL and eliminate the stabilizing effect from an intact volar plate. RCL is evaluated in a similar manner, except the phalanx is passively deviated in the ulnar direction while the metacarpal is stabilized.

**Differential diagnosis:** Metacarpal head or phalangeal base fractures

**Diagnostics:** PA and lateral plain radiographs required to evaluate for fractures. In RCL injuries, the lateral view may reveal sagging of the proximal phalanx relative to the metacarpal. Magnetic resonance imaging (MRI) may be helpful in clarifying both UCL and RCL injuries. Stener lesions may be evident on MRI, which is an important feature in determining treatment.

**Treatment:** For UCL injuries, the degree of tear, avulsion fracture displacement, and presence or absence of a Stener lesion is considered. Partial tears with minimal relative laxity can usually be managed by thumb spica cast (including the IP joints) immobilization for 4–6 weeks, followed by a 2-week period of splinting and ROM exercises. A Stener lesion requires surgery to remove the interposed tendon and repair the ligament to its insertion bed. Controversy surrounds complete tears wherein a Stener lesion is not apparent. Several experts advocate direct primary repair, whereas others may recommend casting. There is no consensus regarding management of acute RCL injuries. However, if sagging of the radial side of the joint is present clinically or on radiographs, surgical repair of the radial collateral complex (including associated capsular hood tear) over immobilization alone is preferred.

**Prognosis and return to play:** If the ligament complex heals, return to full activity within a couple of months; however, participation with a cast may begin as soon as the wound is stable. A small proportion of patients may continue to have pain with forceful grip, even with a clinically stable repair, thus requiring more prolonged splinting. If untreated, symptomatic instability persists and eventual osteoarthritis may develop.

## Metacarpophalangeal (MCP) Dislocation (Thumb or Finger)

**Description:** Complete dislocation of thumb or finger proximal phalanx (usually dorsal) in relation to the corresponding metacarpal, classified as either *simple* or *complex*; simple dislocation can be manually reduced; complex dislocation requires open reduction to remove the interposed tissue. In both simple and complex dislocations, the volar plate usually detaches from its metacarpal attachment and retains its attachment on the proximal phalanx. However, in a complex dislocation, the entire volar plate is interposed in the MCP joint, preventing manual reduction (Fig. 51.10). Apart from the interposed volar plate, the flexor tendons and tendons from intrinsic musculature can pass around opposite sides of the metacarpal head, creating a "noose effect" that blocks reduction when traction is applied.

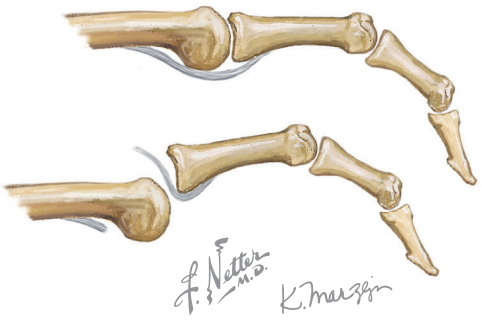
**Mechanism of injury:** Hyperextension injury to a digit at the level of the MCP joint

**Presentation:** Acute pain, swelling, and hyperextension of the MCP joint; border digits are most commonly affected

**Physical examination:** Differentiate between a simple and complex dislocation. Affected joint is typically hyperextended to approximately 70–90 degrees in a simple dislocation, whereas less hyperextension is seen in a complex dislocation. A volar skin "dimple" near the region of the A1 pulley is a pathognomonic finding of a complex dislocation. If a dimple is present, one must take care to evaluate the overlying skin for threatened skin or pressure necrosis. A careful neurovascular examination of the digit should be performed.

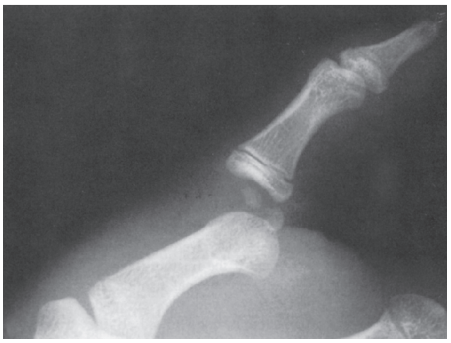
**Differential diagnosis:** A fracture at the level of the MCP joint may occur in conjunction with a dislocation.

**Diagnostics:** Physical examination is the mainstay in initial diagnosis. While reduction may be attempted on the field, *eventual* PA, lateral, and oblique radiographs are required because up to



Metacarpophalangeal (MCP) joint dislocation is typically dorsal, and frequently the volar plate becomes incarcerated dorsally.

Thumb MCP dislocation is clinically apparent with shortened thumb and fullness in thenar eminence.



Complex thumb MP dislocation, showing joint space widening and entrapped sesamoids within joint

**Figure 51.10.** Metacarpophalangeal (MCP) dislocation.

50% of such injuries have an associated fracture. Before reduction or following failed attempted reduction, simple dislocations will demonstrate the proximal phalanx hyperextended 70–90 degrees; complex dislocation may demonstrate excessive joint widening or entrapped sesamoids (see Fig. 51.10).

**Treatment:** Closed reduction is the initial treatment strategy, taking care to avoid converting a simple dislocation to a complex one. Technique includes wrist flexion (to relax flexor tendons), gentle traction, and volar translation of the base of the proximal phalanx relative to the metacarpal head to slide the phalanx into position. Excessive hyperextension and axial traction should be avoided because it may convert a simple to a complex dislocation by flipping the volar plate, lumbrical tendon, thenar tendon (in the thumb), or flexor tendon dorsal to the metacarpal head. For an irreducible dislocation, an open reduction must be performed, using either a dorsal or volar approach. Following reduction of thumb MCP dislocations, grade-III UCL tears with associated gross instability may be better managed with direct ligament repair, similar to skier's thumb (see above).

**Prognosis and return to play:** Simple dislocations in fingers should be protected from hyperextension with a splint for at least 2 weeks while allowing early active flexion. Depending on the specific sport, consider prolonged protection against hyperextension with splint during participation. Consider slightly prolonging the protection of complex repairs. Thumb MCP dislocations mandate a more guarded approach to mobilization and return to play. Stability is a greater priority than mobility here. Rigid immobilization for 4 weeks is recommended with protection against forced hyperextension/hyperabduction for another 2 weeks. Following surgical ligament repair, such protection against impact should be considered for 2–3 months.

## 2nd–5th Carpometacarpal (CMC) Dislocation

**Description:** Complete dislocation of the metacarpal in relation to the adjacent carpus; metacarpals dislocate dorsal relative to carpus; often associated with fractures along the carpal joint margin, which significantly increases instability following reduction.

**Mechanism of injury:** High-energy axial load on the metacarpals; fifth and fourth rays may dislocate in isolation because of a more mobile articulation with the hamate. Index and long carpometacarpal (CMC) joints typically dislocate only in high-energy injuries wherein all four CMC joints are dislocated.

**Presentation:** Significant dorsal swelling and apprehension to grip

**Physical examination:** Inspection for an open injury; careful palpation of each digit is required to look for associated fractures. A neurovascular examination is imperative. Tense swelling in setting of high-energy injuries should raise concern for hand compartment syndrome.

**Differential diagnosis:** Metacarpal fractures, severe contusion, and carpal dislocations

**Diagnostics:** AP, lateral, and oblique radiographs are the mainstay. Radiographic evidence of dislocation is rather obvious when all four CMC joints are involved; however, radiographic findings are more subtle when dislocations occur in isolation. A "true lateral" radiograph of the involved digit must be obtained. Lateral views of small and ring fingers require approximately 30 degrees of pronation at the wrist; lateral views of the index and long finger may require more supination. Distraction or traction views may be useful. Computed tomography (CT) scans can be particularly helpful in cases of fracture-dislocations.

**Treatment:** These injuries are inherently unstable although isolated dislocations may be stable after a closed reduction maneuver is performed. Reduction maneuver is performed by applying axial traction in conjunction with a palmar-directed force on the dorsal base of the affected metacarpal. Most injuries require closed or open reduction combined with internal fixation (usually temporary pins).

**Prognosis and return to play:** Because of the unstable nature of these injuries, return to play involving gripping or hand contact occurs after 6–8 weeks to allow for sufficient healing. In an isolated CMC dislocation that is stable upon closed reduction, return to play may be earlier with protective bracing or casting.

## Scaphoid Fracture

**Description:** A common injury in sports; a precarious retrograde vascular supply renders the scaphoid prone to slow healing and nonunion, particularly in fractures of the waist or proximal pole. If left untreated, scaphoid fractures in adolescents or young adults invariably progress to symptomatic nonunion and secondary arthritic collapse (Fig. 51.11). In isolation, this fracture results in no visible deformity and often no more pain than a minor joint sprain. As such, the diagnosis is frequently missed or the athlete often does not complain of wrist pain until weeks or months later.

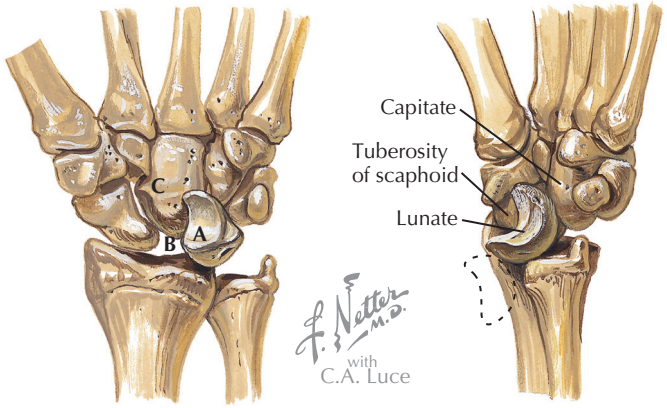
**Mechanism of injury:** Frequently results from a fall on the outstretched hand with an extension load across the distal radius and carpus. The proximal pole is locked in the scaphoid fossa of the radial articular surface, resulting in failure of the waist or proximal pole of the scaphoid. A less frequent mechanism is an axial load through the carpus.

**Presentation:** Radial-sided wrist pain, usually following a fall or sudden axial load through the wrist. Usually no visible deformity, including lack of swelling. Patient will often still be able to

Radiograph of scaphoid nonunion with collapse and secondary osteophyte formation

Normal x-ray of acute fracture

**Figure 51.11.** Scaphoid fractures.




Palmar view shows (A) lunate rotated and displaced volarly, (B) scapholunate space widened, (C) capitate displaced proximally and dorsally.

Lateral view shows lunate displaced volarly and rotated. Broken line indicates further dislocation to volar aspect of distal radius.

**Figure 51.12.** Volar lunate dislocation. Note that in *perilunate* dislocation, the lunate remains in place relative to the radius while the capitate dislocates relative to the lunate (see Fig 51.13).

move the wrist in the mid-ROM, particularly in more delayed presentations.

**Physical examination:** Tenderness to palpation directly over the fracture; in waist fractures, this is in the anatomic snuffbox. However, the proximal pole should be assessed by palpation at the scapholunate joint just distal to the dorsal radial tubercle. The distal scaphoid pole can be palpated at the scaphotrapezial joint, deep to the intersection of the flexor carpi radialis tendon and wrist flexion crease. Forced passive wrist extension also usually produces pain at the fracture site.

**Differential diagnosis:** Distal radius fractures, injuries to the scapholunate ligament, and perilunate or lunate dislocations (markedly more painful with visible deformity)

**Diagnostics:** PA, oblique, lateral, and clenched-fist/ulnar deviation radiographs are essential. If plain radiographs are negative, bone scan or MRI may demonstrate the presence of a nondisplaced fracture, although MRI may offer more specificity (see Fig. 51.11). However, simple repeat radiographs 2–3 weeks later following cast immobilization may reveal bone changes around a previously unrecognizable fracture. To determine displacement and potential surgical planning, CT scan offers the best bony detail.

**Treatment:** Displaced fractures are managed with arthroscopic or ORIF, as are fractures associated with other carpal fractures or ligament injuries. With displacement, the union rate precipitously decreases with immobilization alone. Controversy surrounds management of truly nondisplaced scaphoid waist fracture because similar union rates and functional results have been demonstrated with immediate cast immobilization compared to immediate percutaneous screw fixation. Immediate fixation may allow players to return to competition sooner while casted than without internal fixation. Proximal pole fractures, even when nondisplaced, are often primarily managed with screw fixation owing to the poor vascularity of that portion of the bone and high nonunion rate.

**Prognosis and return to play:** Union rate with immobilization of nondisplaced or impacted distal pole fractures is >90%. Union rate of nondisplaced waist fractures with immobilization ranges from 85% to 95%. Proximal pole fracture union rates drop significantly. Return to play is dictated by the fixation stability and the degree of fracture union. With anatomic reduction and screw fixation, a player may be able to return to play *in a cast* within a few weeks or once the wounds have adequately healed. Until union occurs, the wrist must be protected against forced flexion or extension.

## Perilunate Injuries

**Description:** ***(scapholunate tears, perilunate, and lunate dislocations)*** Injuries involving combinations of ligaments and bones surrounding the lunate (see Fig 51.12)

**Mechanism of injury:** Wrist hyperextension and ulnar deviation during axial loading

**Physical examination:** Localized wrist pain, point tenderness, limited motion, and occasionally median nerve dysfunction; with lunate dislocation, bone may be palpable at the wrist flexion crease

**Differential diagnosis:** Scaphoid fracture and distal radius fracture

**Diagnostics:** PA, lateral, and scaphoid radiograph of wrist; any malalignment of the lunate relative to the radius or capitate on the lateral view is evidence of perilunate injury (see Fig 51.13). With normal radiographs and profound carpal tenderness or swelling, MRI is indicated.

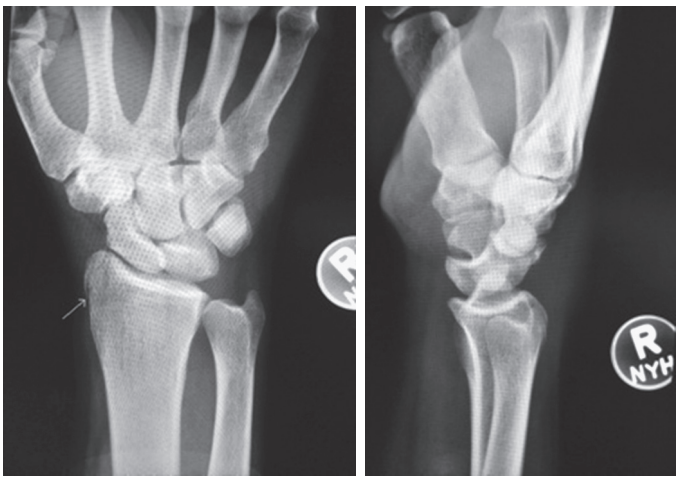


Figure 51.13. Radiographs of *perilunate* dislocation.

**Treatment:** All acute perilunate fractures and dislocations require surgical repair of the ligaments and/or bony elements. Combinations of K-wires and screws are used.

**Prognosis and return to play:** K-wires are removed at 8 weeks, and cast immobilization is required for 12 weeks. Most injuries take 4–5 months before ROM and strength stabilize.

## Distal Radius Fractures

**Description:** One of the most common upper limb injuries seen in orthopedics; in an athlete with open physes, fractures through the growth plate are particularly common following falls onto the outstretched hand; seen commonly in snowboarders. Fracture patterns may be limited to the metaphysis or also involve radiocarpal or ulnocarpal articulations. Degree of comminution is determined by the quality of the bone and energy imparted to it.

**Mechanism of injury:** Fall onto the outstretched hand is the most common mechanism.

**Presentation:** Nondisplaced fractures may have no visible deformity other than mild swelling with complaints of wrist pain following a fall. The more common dorsally displaced patterns may demonstrate a "dinner fork" deformity just proximal to the carpus. Patients will be apprehensive regarding any motion of the wrist. These injuries are typically more painful than scaphoid fractures; hence, most patients will be forced to withdraw from play.

**Physical examination:** Assessment of skin integrity and neurovascular status; open fractures, seen with higher-energy injuries with greater degrees of displacement at the fracture site, which fortunately are uncommon in athletic competition. With greater dorsal displacement at the fracture site, there is a higher risk of traction injuries to the median nerve. Sensation of the radial three and a half digits should be assessed before any manipulation. In nondisplaced fractures, the patient will reliably be tender to palpation directly over the fracture site.

**Differential diagnosis:** Carpal fractures and ligament injuries should always be considered; usually differentiated by direct palpation because patient will usually be tender over the fractured radial metaphysis as opposed to the carpus. Nevertheless, these injuries can occur in combination, and tenderness over the scaphoid as well as the radius should lead to assessment with appropriate plain radiographs.

**Diagnostics:** PA and lateral plain radiographs usually sufficient; CT may be useful for surgical planning for fractures with intra-articular displacement or comminution

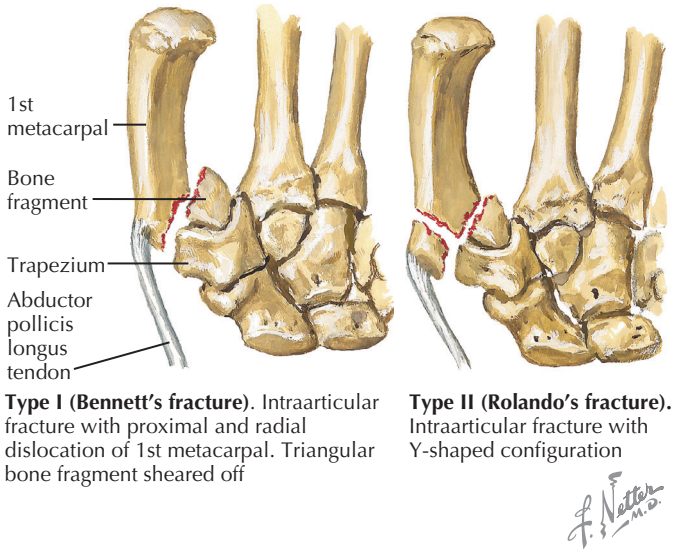



Figure 51.14. Bennett's and Rolando's (first metacarpal base) fractures.

**Treatment:** Wrist is splinted in the neutral position; for fractures with obvious clinical displacement, there is little to be gained from immediate manipulation or attempted reduction before obtaining plain radiographs or having adequate analgesia. Supportive splinting of the wrist should be applied until then. Common guidelines for accepted displacement of extra-articular fractures in skeletally mature individuals of ≤10 degrees of dorsal tilt of the articular surface. For intra articular patterns, most practitioners accept ≤2 mm of gap or a 1-mm step-off of the joint surface. If the reduction cannot be maintained with closed manipulation and splinting, ORIF is recommended. For nondisplaced or minimally displaced fractures, ORIF may allow earlier ROM and use of a removable splint.

**Prognosis and return to play:** Participation in contact sports or load bearing of the fractured radius is usually precluded until stable union is evident, minimum of 4–6 weeks, depending on patient age and fracture pattern. Restoration of normal anatomy ensures optimal prognosis in skeletally mature individuals, whereas younger patients with open physes may still require some remodeling.

## Bennett's and Rolando's (First Metacarpal Base) Fractures

**Description:** Intra-articular fractures of the base of the thumb metacarpal. A Bennett's fracture pattern includes a constant piece on the volar–ulnar aspect of thumb metacarpal base that is held in normal anatomic position to the adjacent trapezium by the strong *deep anterior oblique (beak) ligament*, while the remainder of the metacarpal base subluxates or dislocates dorsally. A Rolando's fracture pattern has the same volar–ulnar fragment and an associated dorsal fragment as well; thus, it is often described as a T- or Y-shaped thumb metacarpal base fracture (Fig. 51.14).

**Mechanism of injury:** Results due to an axially directed force through the shaft of the thumb metacarpal

**Presentation:** Acute pain and swelling localized to the base of the thumb metacarpal after a contact or impact injury; pain is associated with any attempt to move the thumb.

**Physical examination:** Pain and swelling localized to the base of the thumb metacarpal

**Differential diagnosis:** Thumb metacarpal shaft fracture and thumb trapeziometacarpal dislocation must be considered.

**Diagnostics:** Accurate injury classification requires quality radiographs. True AP views of the thumb CMC joint, with the wrist pronated and dorsum of the first metacarpal placed flat against the plate combined with lateral view, should suffice. Traction views may also be helpful.

**Treatment:** These fractures are unstable and surgical management is recommended to maintain CMC joint congruity. Bennett's fractures are often successfully managed with closed reduction and percutaneous pin fixation, whereas Rolando's fractures typically require ORIF techniques.

**Prognosis and return to play:** Overall prognosis is good if articular congruity and length of the metacarpal is restored. A typical course of 6 weeks of immobilization is required before gradual return to play with protective bracing depending on sport-specific demands.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eAppendix 51-1, Hook of Hamate Fracture
- eAppendix 51-2, Injuries to the Triangular Fibrocartilage Complex (TFCC)

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

### eAppendix 51-1 Hook of Hamate Fracture

**Description:** The hook of the hamate projects volarly into the palm, serving as the ulnar border of the carpal canal and ulnar insertion point of the transverse carpal ligament. The prominent palmar position makes it vulnerable to impact (see eFig. 51.1).

**Mechanism:** "Ball and stick" athletes (baseball, golf, tennis, etc.) frequently sustain such an injury as the heel of the bat, racquet, or club frequently impacts the hamate hook.

**Presentation:** Frequently delayed as there is little visible swelling or deformity; patient complains of palmar pain aggravated by grip or impact. Ulnar nerve paresthesia may also occur.

**Physical examination:** Localized palmar tenderness over the hamate hook is usually present. Pain may also occur with resisted ring and small finger flexion with the wrist in ulnar deviation as the tensioned tendons press against the fracture. Pain may be lessened as the wrist is radially deviated while maintaining resisted digital flexion.

**Differential diagnosis:** Palmar contusion, fracture of body of hamate, and ulnar artery thrombosis

**Diagnostics:** Specialized "carpal tunnel view" (see eFig. 51.1) offers the best profile view of the hamate hook. If this is negative in the setting of the aforementioned presentation and findings, CT scan is recommended.

**Treatment:** Acute nondisplaced fractures may be managed with cast immobilization. However, unclarified union rate and potential prolonged immobilization time have led numerous athletes to undergo primary surgical excision of the fractured fragment with favorable results.

**Prognosis and return to play:** Once tenderness has resolved following either nonsurgical immobilization or surgical excision, the athlete may return to play. Following excision, most athletes report full participation at 6 weeks. One particular complication associated with an untreated hook fracture is delayed rupture of the ring and small FDP tendons.

### eAppendix 51-2 Injuries to the Triangular Fibrocartilage Complex (TFCC)

**Description:** The triangular fibrocartilage complex (TFCC) comprises the triangular fibrocartilage disk, dorsal radioulnar ligament (DRUL), volar radioulnar ligament (VRUL), extensor carpi ulnaris tendon subsheath, and the meniscal homologue (eFig. 51.2). It is the primary structure that provides stability to the distal radioulnar joint (DRUJ) throughout the full range of supination and pronation.

**Classification:** Traumatic (type I) and degenerative (type II) tears

**Mechanism of injury:** Most acute injuries result from a fall onto the outstretched hand; degenerative tears of the central portion of the disk generally occur in the absence of a single inciting event.

**Presentation:** Persistent ulnar-sided wrist pain, often with mild swelling in the ulnar fovea, since the event; swelling is often evident in the ulnar fovea. Significant disruption to the ligaments or their deep insertion at the base of the ulnar styloid may result in marked DRUJ instability with visible prominence of the ulnar head. Chronic, untreated, partially traumatic tears or degenerative tears may present as recurring ulnar-sided wrist pain during activities requiring vigorous wrist motion or loading (push-ups, golf, tennis, and cycling).

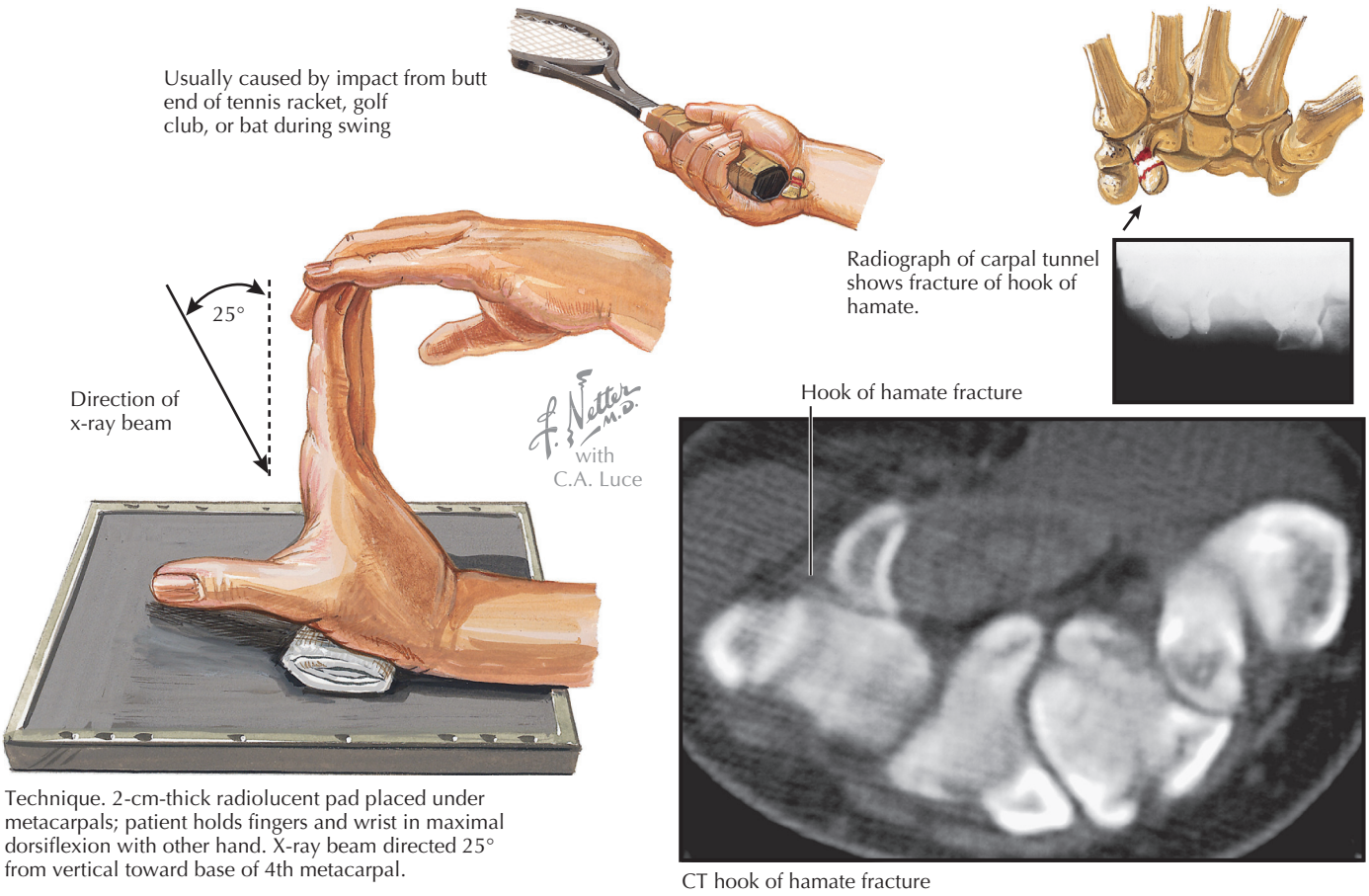
**Physical examination:** Pain or limitations with supination and pronation; palpation tenderness is usually present over the ulnar fovea and perhaps also over the dorsal aspect of the TFCC, just distal to the ulnar head (see eFig. 51.2). DRUJ should be passively stressed by holding the distal radius in one hand and the distal ulna in the other while translating them relative to each other in neutral rotation, supination, and pronation. Pain and/or palpable "click" with the maneuver may also be noticed. Dorsal tenderness over the lunotriquetral ligament may suggest an injury to that structure. Passive ulnar deviation of the pronated wrist that results in ulnar-sided pain is also suggestive of a TFCC tear.

**Differential diagnosis:** Carpal fractures, lunotriquetral ligament injuries, extensor carpi ulnaris tendon instability, and ulnocarpal synovitis without actual associated TFCC tear can also result in pain in this region.

**Diagnostics:** Plain PA and lateral radiographs are useful to identify fractures of the distal ulna or carpus, subluxation of the DRUJ, calcification of the TFCC (suggestive of pseudogout), degenerative changes of ulnar head and ulnar corner of lunate (suggestive of ulnocarpal impaction), and ulnar variance. Positive ulnar variance occurs with the ulna relatively longer than the radius and has a greater association with ulnocarpal impaction and degenerative TFCC tears. MRI may be useful. Arthrogram may reveal tears in the complex but is being employed less frequently. The current gold standard for TFCC evaluation is arthroscopy, which is obviously more invasive but also has the potential for offering treatment at the time of diagnosis. Isolated deep tears of the TFCC insertion may not be visible on arthroscopic evaluation and may be better seen with an MR arthrogram.

**Treatment:** Several TFCC tears, both traumatic and degenerative, may be minimally symptomatic and may not require treatment. If the athlete is in season and does not want to be out for possibly 6–10 weeks following a TFCC repair, a steroid injection into the TFCC through the ulnar fovea may offer enough pain relief to allow continued participation until potential surgical intervention. In stable joints, pain may resolve with time and observation alone. Symptomatic instability of the joint is best treated with surgical repair. Peripheral traumatic tears occur in the vascularized portion of the triangular fibrocartilage and are usually reparable. Central and degenerative tears are relatively avascular (similar to the meniscus in the knee) and do not heal following repair. Degenerative tears may respond to debridement with associated shortening (either arthroscopic or ulnar shaft osteotomy) of the positive or neutral ulna.

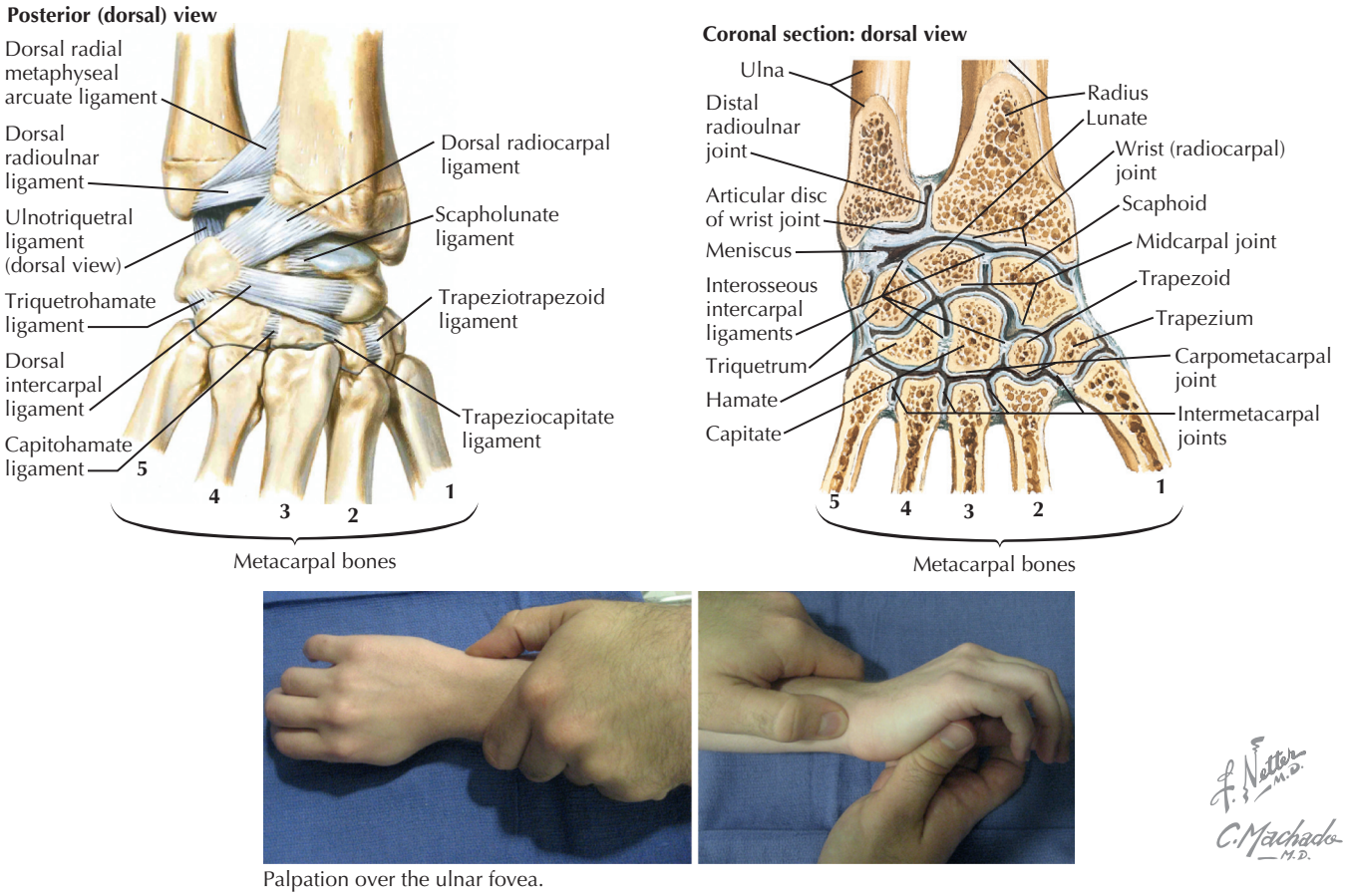
**Prognosis and return to play:** Athletes may be able to return to play if a steroid injection ameliorates their symptoms and there is absence of instability. Repair of the torn TFCC will require a minimum of 4 weeks of cast immobilization followed by a period of gradual motion exercises and progressive strengthening over the next 2–4 weeks. Return to competition in that setting is dependent on the demands of the particular sport.




Technique. 2-cm-thick radiolucent pad placed under metacarpals; patient holds fingers and wrist in maximal dorsiflexion with other hand. X-ray beam directed 25° from vertical toward base of 4th metacarpal.

CT hook of hamate fracture

**eFigure 51.1.** Hook of hamate fracture.






Palpation over the ulnar fovea.

**eFigure 51.2.** Injuries to the triangular fibrocartilage complex (TFCC).

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# 52 THORAX AND ABDOMINAL INJURIES

Sameer Dixit • Cindy J. Chang

## GENERAL PRINCIPLES

### Overview

- Injuries to the thorax and abdomen are more often seen in sports involving sudden deceleration and impact (football, ice hockey, skiing, and snowboarding).
- Early recognition and management of these potentially life-threatening injuries is imperative. Repeated assessment and a high index of suspicion are essential for accurate evaluation. Once a severe injury is recognized, fundamentals of emergency treatment and stabilization should be initiated until transfer to a hospital occurs.
- Torso injuries can overlap with injuries to the skeletal system (e.g., traction apophysitis of the iliac crest presenting as lower abdominal pain; shoulder conditions can radiate to the thorax; similarly, thoracic and abdominal conditions can radiate to the extremities, confusing the source of symptoms).

### Anatomic and Physiologic Issues

- Combination injuries in the upper abdomen can be divided into three regions.
  - Midline region: Left lobe of the liver, pancreas, duodenum, transverse colon, small bowel and mesentery, aorta, inferior vena cava, sternum, lower ribs, and heart
  - Right region: Liver, kidney, adrenal gland, hemidiaphragm, lung, pneumothorax or hemothorax, and ribs
  - Left region: Other paired organs, but the spleen instead of the liver
- In sports, these organs can suffer damage usually resulting from compressive forces (e.g., tackle or bicycle handlebar) that push a solid or viscus organ against the fixed spine.
- Deceleration forces and penetrating injuries are more uncommon in athletics, although "almost penetrating" injuries are possible (e.g., hockey stick or ski pole) without causing a wound.
- Abdominal organs in children are more susceptible to injury from trauma because of their relative position (more anterior and lower due to the more horizontal nature of the diaphragm), the still developing abdominal musculature, and the pliable nature of cartilaginous ribs.
- **"Getting the wind knocked out"** is a more common occurrence than significant trauma to a visceral organ. An unguarded blow to the epigastric region causes a temporary reflex spasm of the diaphragm. Loosening of restricting garments and flexion at the knees and hips usually restores normal respiration. Owing to the risk of intra-abdominal injury, careful observation and follow-up are necessary.

### Epidemiology, Injury Statistics, and Sports-Specific Issues

Sport-specific epidemiologic data are limited.

**Myocardial injury:** May occur in up to 76% of patients sustaining blunt trauma to the chest, with direct compression of the heart between the anterior chest wall and vertebral column

**Abdominal injury:** A 1993 study evaluating serious pediatric sports injuries found that abdominal injuries accounted for 7% of hospitalized cases, whereas fractures were the most frequent reason for hospitalization (77%). The most frequent cause of abdominal injury in children is a bicycle accident.

**Thoracic and pulmonary injuries:** According to a study of male professional rugby players, 8.3/1000 player-hours were lost because of match injuries involving the upper back, sternum, and ribs in comparison with 45.8/1000 player-hours lost because of lower limb injuries. Blunt chest trauma is the most common cause of both cardiac and pulmonary contusions; >90% of such injuries result from motor vehicle accidents (MVAs). Pneumothoraces, with or without concomitant rib fractures, are uncommon injuries in sports but are being increasingly reported. Spontaneous pneumothorax caused by the strenuous weightlifting has been reported.

### History and Physical Examination (PE)

#### History

- An accurate account of events leading to an injury is important in establishing the diagnosis.
- History and physical examination (PE) are sometimes unreliable, particularly in children and if there is an altered level of consciousness.
- A seemingly minor trauma can cause delayed splenic rupture or other injuries; careful history-taking including past injuries, surgeries, and illnesses is paramount.
- Previously undiagnosed preexisting condition such as inflammatory bowel disease, liver hemangioma, and infectious mononucleosis can cause major clinical symptoms after trauma to an affected organ.
- Detailed history of the patient's pain and the use of a pain scale upon initial presentation and during serial evaluations are useful; examples include the PQRST principles of evaluation of pain, and a visual analog pain scale (Table 52.1)

#### PE

- See Chapter 35 for a discussion of the cardiovascular examination; see Chapter 4 for a discussion of the initial evaluation of injuries to the thorax and abdomen.

#### General Appearance and Vital Signs

- If thoracic and abdominal injuries both present, thoracic injuries are usually more symptomatic and will distract attention from abdominal pain, which is usually less localized and specific.
- Abdominal pain can be vague and diffuse or localized to a quadrant. Abdominal pain is sensitive but not specific to the presence of injury; 50% of individuals with pain have no significant abdominal injury. Always examine the chest and spine when evaluating an abdominal complaint, and consider examining the inguinal and pelvic regions.
- Frequent monitoring of vital signs, including orthostatics, is important to gauge the cardiovascular status; in addition, respiratory rate, rhythm, and use of accessory respiratory muscles should be observed. If difficult to obtain blood pressure by auscultation, deflate the cuff until palpable return of the brachial or radial pulse; the systolic pressure obtained by auscultation is approximately 10 mmHg higher.

#### Inspection

- Observe the effort of breathing; listen for abnormal breathing sounds.
- Look for asymmetry, deformity, swelling, bruising, lacerations, and scars.

**TABLE 52.1 PQRST EVALUATION OF PAIN**

| | |
|---|---|
| **P** | **Palliative/provoking**<br>What causes it?<br>What makes it better?<br>What makes it worse? |
| **Q** | **Quality**<br>How does it feel, look, or sound?<br>How much of it is there? |
| **R** | **Radiation**<br>Where is it?<br>Does it spread? |
| **S** | **Severity**<br>Does it interfere with activities?<br>How does it rate on a severity of 1 to 10? |
| **T** | **Timing**<br>When did it begin?<br>How often does it occur?<br>Is it sudden or gradual? |

- Confirm that the trachea is in the midline and the chest has a normal anteroposterior (AP) diameter.
- Evaluate the abdominal contour and signs of increasing abdominal girth; observe for peristaltic or pulsating movements.
- Observe for splinting or guarding of torso and upper extremities (UEs) or any change in the neck position.

### Auscultation, Percussion, and Palpation

- Auscultate both the posterior and anterior chest, comparing for asymmetry. If breath sounds are decreased, the normal lung has been displaced by air (suspect pneumothorax) or fluid (hemopneumothorax or pleural effusion).
- Percuss the posterior and anterior chest, comparing both sides, and for normal diaphragmatic excursion with inspiration (symmetrical 3–5 cm). If hyperresonant, suspect pneumothorax; if dull, suspect fluid. Percuss all four quadrants; percuss the liver span (range 6–12 cm), and check for splenic enlargement.
- Auscultate before palpating because bowel sounds can change with manipulation. Listen for bruits over the aorta and renal and iliac arteries. Start with gentle palpation to check for areas of tenderness, noting facial expression and any guarding. Follow with deep palpation to further delineate areas of pain or presence of abdominal masses. Include a complete evaluation of the genitourinary system.
- For bony and soft tissue injuries of the thorax, perform neck, thoracic spine, and shoulder range of motion (ROM) and strength tests and palpate for crepitus, deformities, or tenderness.

## SPECIFIC INJURIES AND PROBLEMS

### Chest Wall Injuries

#### Sternal Fracture

**Description:** The incidence of associated intrathoracic trauma is high in acute injury, particularly rib fractures and soft tissue contusions. Incidence may be increasing: in a recent case series of 22 patients, 11 injuries were related to sports.

**Mechanism of injury (MOI):** High-impact injuries and acute hyperflexion of the cervicothoracic spine

**Presentation:** Chest pain (CP), localized tenderness over the sternum, and shortness of breath (SOB)

**PE:** Bruising, swelling, localized pleuritic pain palpable, or visible defect suggesting displacement

**Differential diagnosis/associated injuries:** Stress fracture of the sternum (develops when great stress placed on the upper body during wrestling or golf), manubriosternal joint dislocation, sternoclavicular or costochondral injury, or sternal contusion; associated injuries include myocardial contusion, injury of internal mammary vessels, retrosternal and mediastinal hematoma, pulmonary laceration or contusion, and rib and thoracic vertebrae fractures

**Diagnostics:** The lateral chest film is best to evaluate a fracture (upper fragment usually displaced anteriorly over lower fragment); posteroanterior (PA) radiograph to evaluate possible pneumothorax or widened mediastinum; cervical, thoracic, and lumbar spine radiographs if flexion/compression mechanism; computed tomography (CT) scan (axial cuts alone are not as sensitive; sagittal and coronal reconstruction views needed); routine CTs important to evaluate for cardiac and aortic injuries, which, although rare, are highly lethal if missed. Ultrasound (US) helps confirm radiologic diagnosis, but its use remains limited in the United States. If intrathoracic trauma is suspected, electrocardiogram (ECG) and chest X-ray (CXR) should be performed; consider repeat ECG in 24 hours.

**Treatment:** If displaced, reduction possible by lying supine and then lifting both arms above the head while hyperextending the thoracic spine at a level just below the scapular spines; **because of a high incidence of associated intrathoracic trauma, observation in hospital with a cardiac monitor advisable during reduction and for at least 24 hours after injury**. Displaced fractures may require open reduction and internal fixation (ORIF), particularly if respiration is compromised.

**Prognosis and return to play (RTP):** Nonunion of sternal fractures rare, but suspect when pain persists over the sternum; if an isolated fracture with no underlying thoracic injuries, progressive return to sport as limited by pain; avoid contact sports until the fracture healed and pain resolved (range 6–12 weeks); if the risk of re-injury is high, consider a chest protector during the sport

#### Dislocation of the Sternoclavicular Joint (SCJ)

**Description:** Relatively infrequent, constituting <1% of somatic dislocations; only 50% of the clavicular joint surface contacts the sternal articular surface; the posterior sternoclavicular ligament is stronger than the anterior ligament; anterior disruption more common than posterior disruption (ratio ranges from 9:1 to 20:1); superior dislocation rare

**Classification:**

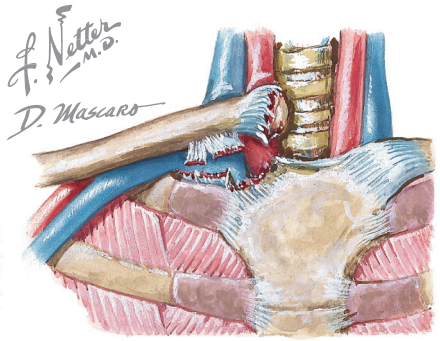
- **Type I:** Sprain with no ligamentous damage or instability
- **Type II:** Stretch or partial rupture of sternoclavicular and costoclavicular ligaments; joint is partially displaced
- **Type III:** Dislocation with gross disruption of capsule and ligaments

**MOI:** Caused by direct or indirect trauma to the shoulder girdle; injury most often seen in contact sports (e.g., martial arts, football, and rugby); indirect trauma can be seen in gymnastics; for anterior dislocation, force applied at the anterolateral aspect of the shoulder or along the abducted arm is transmitted along the clavicle to the SCJ, compressing and rolling the shoulder back and displacing the clavicle. For posterior dislocation, force applied to the posterolateral aspect of the shoulder when the arm is adducted and flexed is transmitted to the SCJ, compressing and rolling the shoulder forward and displacing the clavicle (Fig. 52.1). Posterior dislocation can also result from a direct blow on the anterior aspect of the medial end of the clavicle.

**Presentation:** Severe pain, particularly with any arm movement; pain exacerbated by coughing, sneezing, or deep breathing; because neck muscles spasm, the head is tilted toward the injured side. Increased discomfort in supine position. Other symptoms include hoarseness, dysphagia, dyspnea, numbness, and weakness or venous engorgement of the ipsilateral arm.

**PE:** Bruising, pain, and significant swelling at the joint; noticeable prominence of the medial end of the clavicle in anterior dislocation; in posterior dislocation, loss of normal prominence but

For posterior dislocation, force is applied to posterolateral aspect of shoulder when arm is adducted and flexed.




Posterior dislocation of sternoclavicular joint. Serious because of probable injury to trachea or vessels

**Figure 52.1.** Posterior dislocation of sternoclavicular joint.

often missed because of swelling; obtain vital signs; observe ease of respiration and neurologic/vascular status of the affected UE to rule out pressure on adjacent vital structures.

**Differential diagnosis/associated injuries:** SCJ sprain or subluxation; fracture of the medial clavicle, fracture of the medial physeal growth plate of the clavicle (fusion occurs between 22 and 25 years of age). In patients younger than 25 years, SCJ dislocations are classified as Salter–Harris type I or II fractures. **More serious injuries are associated with posterior SCJ dislocations; a 30% incidence of injury to vital structures traversing thoracic outlet, including major vessels of neck, brachial plexus, dome of pleurae, trachea, esophagus, and larynx; a 12.5% mortality rate in this group of injuries**

**Diagnostics:** Plain radiographs with a 40-degree cephalic tilt view ("serendipity" view); tube distance for children, 45 inches; for thicker-chested athletes, 60 inches; CXR to rule out pneumothorax; axial CT images (3-mm cuts) have greater sensitivity and specificity and are an imaging modality of choice for the SCJ; can differentiate fractures from dislocations, and allow assessment of adjacent mediastinal structures if intravenous (IV) contrast used; coronal plane paraxial CT reconstruction if superior component of dislocation is suspected; the role of US in the diagnosis of SCJ injury is equivocal as the reported sensitivity is low; occasionally, arteriography/venography may be needed.

**Treatment:**

- **Type II injuries:** Avoid stress to joint for at least 3–4 weeks for adequate healing; goal to avoid increased symptomatic mobility at joint
- **Type III injuries:** Immediate closed reduction for posterior dislocations with impending airway or bleeding complications; otherwise, reduce in the operating room under anesthesia in the presence of a cardiothoracic surgeon. Closed reduction method has a reported 80% success rate. Anterior dislocations can be reduced in outpatient settings by applying gentle pressure over the displaced medial aspect of the clavicle. If closed reduction fails, open reduction can be considered in severe cases; however, an anteriorly displaced medial clavicle often becomes relatively asymptomatic with activities of daily living (ADLs). Chronic joint instability may cause pain and persistent functional limitation in active patients; this is an indication for surgical intervention.
- **For chronic SCJ dislocation:** Check for hypermobility of surrounding structures (including acromioclavicular and glenohumeral joints). Consider a limited course of corticosteroid injections for symptomatic patients.

**Prognosis and RTP:** Despite 6–8 weeks of immobilization after successful reduction, healing can be inadequate and a mild SCJ instability may persist, with a propensity for recurrent subluxation. Osteomyelitis of the clavicle can be a late complication. Operative stabilization can be difficult with unpredictable results. In cases of recurrent stabilization failure or painful arthroses, resection of the medial clavicle as salvage procedure.

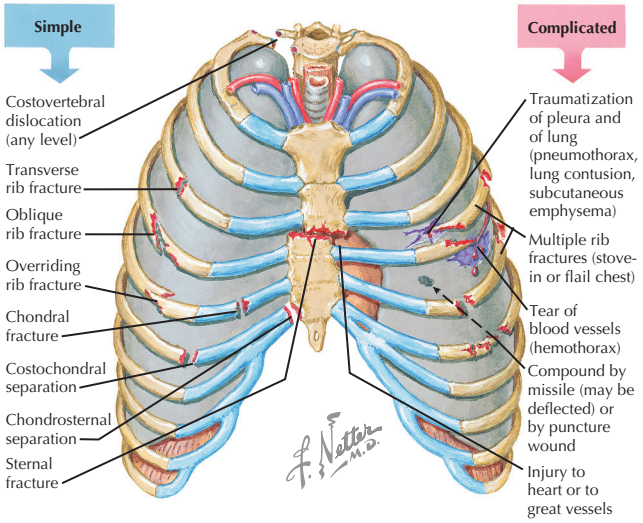



**Figure 52.2.** Thoracic cage injuries.

## Rib Fractures

**Description:** Most common serious injury of chest; can be complete, incomplete, or stress fractures (Fig. 52.2); often associated with other injuries, including other fractures and organ trauma; nondisplaced fractures more common; if displaced, other injuries include laceration of intercostal artery and pneumothorax; uncommon in children because thorax is more pliable

**MOI:**

- Blunt trauma: Force usually applied in the AP plane; fractures located at the posterior angles of 5th–9th ribs
- Direct force over a small area of the chest wall leads to fracture beneath the point of impact.
- Violent muscle contraction: **Floating rib** or **avulsion fractures** of attachments of external oblique muscle to lower three ribs; reported in baseball pitchers and batters. Forceful contraction, against significant resistance, of other muscles that attach to the ribs can also result in fractures.
- **Fracture of 1st rib:** Direct external trauma is rare cause because of protection of shoulder girdle. Other causes are indirect trauma from falling on outstretched arm, violent muscular pull (e.g., hyperabduction of arm), or repetitive stresses.
- **Stress fractures of the ribs:** Caused by excessive forceful muscular traction at the attachments to ribs. Chronic

opposing pulls of scalene muscles and upper digitations of serratus anterior may fracture the **1st rib** at its thinnest and most anatomically weak segment, where the subclavian artery crosses (subclavian sulcus), as with **weightlifting, pitching, and other throwers**. Anterolateral stress fractures of **4th** and **5th ribs,** and other ribs have been reported in **rowers** because of excessive action of serratus anterior muscle (see Chapter 85, Rowing).

**Presentation:** History of traumatic event with intense localized pain over involved rib. **If 1st rib injured, may complain of shoulder, scapular, or neck pain; may complain of abdominal pain if lower ribs (11th and 12th) involved**. With stress fractures, insidious onset of pain associated with specific activities, and possible radiation of pain. If fracture unstable, pain is acute and knifelike. Pain aggravated by deep inspiration, coughing, or sneezing and with twisting or side flexion (causing tension on fractured rib); may report dyspnea

**PE:** Localized tenderness, ecchymosis, and edema; crepitus over fracture site; palpable deformity of rib if fracture displaced; shallow and rapid breathing; with AP and transverse compression of rib cage, pain at the site of suspected injury; subcutaneous emphysema with pleural injury

**Differential diagnosis/associated injuries:**

- Severe rib contusion, costochondral separation, muscle strain (e.g., forceful contraction of thoracic muscles during tennis serve), and other medical causes of CP (e.g., pneumothorax, pleurisy, and herpes zoster)
- The more ribs that fracture, the greater the incidence of intrathoracic injuries
- **If the 1st rib fracture displaces posteriorly, check for vascular injury** (e.g., subclavian artery and aorta).
- **Fracture of lower two ribs may damage** the **kidneys, liver, or spleen**; splenic trauma reported in up to 20% of left lower rib fractures; liver trauma in up to 10% of right lower rib fractures
- **Flail chest:** Fracture of at least three consecutive ribs, each in two locations, causing free-floating segment of chest wall; high risk of internal injury, particularly lung and thoracic aorta. Paradoxical chest wall movement results in impaired ventilation and respiratory failure.

**Diagnostics:** CXR establishes diagnosis in 90% of cases if acute fracture; can also exclude complications, such as pneumothorax. Oblique views may detect anterior and lateral fractures. Bone scan if stress fracture suspected; US more sensitive than conventional radiography; CT scan most appropriate imaging modality if suspect posterior displacement of 1st rib; if cardiac complications possible, follow-up with ECG and echocardiogram. If upper thoracic ribs fractured, angiography may be indicated. If renal injury is suspected, IV pyelogram or other imaging modalities may be performed.

**Treatment:** Pain relief using ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and analgesics; bone stimulators have been approved by the Food and Drug Administration (FDA) for certain fractures and fracture sites, but no published data on use for acute rib fractures or stress fractures; intercostal nerve block for relief of severe pain; after aspirating, infiltrate just below the lower border of the rib, in close approximation to the intercostals vessels and nerve. The level of fracture along with two ribs above and below is infiltrated with 3–5 mL of lidocaine or bupivacaine; risk of causing pneumothorax; multiple fractures may require ORIF. Encourage deep breathing to prevent atelectasis and pneumonia; avoid/minimize rib belt or taping. Activity modification until symptoms resolve, then gradual resumption of training; changes in technique (if thought to contribute to cause of fracture); nutritional evaluation and pertinent laboratory tests if bone insufficiency suspected.

**Prognosis and RTP:** RTP when no pain on palpation, no use of analgesics, full ROM of thoracic cage, and ability to sprint/twist without significant discomfort; usually minimum 3 weeks and typically 6–8 weeks before return to contact sports; ability to protect fracture site should be considered; early return inadvisable because of danger of pneumothorax; close follow-up essential to avoid delayed complications (e.g., excessive callus formation of 1st rib can cause thoracic outlet syndrome [TOS] or Horner's syndrome)

### Costochondral Sprain and Separation ("Rib-Tip" or "Slipping Rib Syndrome")

**Description:** Frequently occurs in contact sports such as football, ice hockey, wrestling, lacrosse, and rugby; weakness or separation of costal cartilage as it attaches to sternum (sternocostal ligament) or separation of anterior margin of the rib from anterior end of the costal cartilage (costochondral ligament), putting pressure on the intercostal nerve lying between it and the rib above; more frequently involves the 10th rib, followed by 9th or 8th.

**MOI:** Forced compression of the rib cage, twisting injury, or stretching injury to the joint when arm is forcefully pulled to the side; onset sometimes insidious, occurring long after initial trauma because loose ribs can cause further stretching of the supporting ligaments

**Presentation:** Often upper abdominal pain or lower CP; history of feeling a pop; initial sharp discomfort, with severe pain lasting for several days before slowly decreasing in intensity. Pain patterns can include a dull sensation, intermittent unilateral pain in anterior ends of lower costal cartilages, or severe sharp pain during bending maneuvers with a painful click as cartilage and bone override one another. Pain can radiate toward the epigastrium or spine.

**PE:** Localized swelling and tenderness at the involved joint; possible deformity because of cartilage displacement. Reproducible pain and sometimes clicking by hooking fingers under the costochondral junction in question and pulling the rib cage anteriorly ("hooking maneuver")

**Differential diagnosis/associated injuries:**

- **Costochondritis or costosternal syndrome:** Both traumatic and nontraumatic; self-limiting; multiple sites of tenderness (usually 2nd–6th costal cartilages) but without swelling
- **Tietze's syndrome:** Traumatic and nontraumatic; self-limiting; usually only the 2nd or 3rd costochondral junction involved with localized swelling

**Diagnostics:** CXR if chronic to rule out tumors, Paget's disease, or rheumatoid arthritis; US of costal margin during abdominal muscle contraction to demonstrate abnormal mobility

**Treatment:** Ice and NSAIDs; injection of lidocaine or bupivacaine with or without corticosteroid at site of separation; rib block; physical therapy for correction of possible posterior dysfunction at corresponding costovertebral joint; surgical resection of the affected costochondral junction or repair of ribs and cartilage for intractable pain

**Prognosis and RTP:** May take 9–12 weeks to resolve (slow healing); subject to re-injury as complete healing is seldom seen

### Rupture of Pectoralis Major

**Description:** Pectoralis major is the most important adductor and internal rotator of shoulder and cosmetically forms anterior wall of axilla. Ruptures can be partial (grades I and II) or complete (grade III). Excessive tension on muscle causes tear of muscle belly, musculotendinous junction, or tendinous insertion on humerus lateral to the bicipital groove; latter most common. Tears of proximal sternal origin are rare; may be associated with anabolic steroid use (muscle hypertrophy not accompanied by tendon adaptation); increased incidence over past several years

**MOI:** Excessive tension on maximally and eccentrically contracted muscles while the affected UE is externally rotated, extended,

or abducted. Among athletes, the most often is seen in weightlifters during bench press; also reported during waterskiing, wrestling, boxing, football, and other sudden violent deceleration maneuvers with sudden stretching and co-contraction of muscle (attempting to grasp something to prevent fall, punching, and blocking with an outstretched arm)

**Presentation:** History of sudden stress or direct blow to shoulder while arm abducted and extended; sudden onset of extreme pain on medial aspect of the UEs or in the chest wall; tearing, snapping, or popping sensation; significant swelling and ecchymosis; painful limitation of motion; weakness of the involved UE; after resolution of swelling and ecchymosis, complaints of asymmetry and persistent weakness.

**PE:** Swelling and hemorrhage into arm and across the anterior chest wall; weakness and pain during resisted internal rotation, flexion, and adduction of arm; deformity of chest wall and palpable muscle bulge with resisted adduction; with abduction, defect in anterior axillary fold if tendon is avulsed at insertion; shoulder ROM limited by pain

**Differential diagnosis/associated injuries:** Pectoralis muscle tendonitis/tendinosis and congenital absence of pectoralis major muscle

**Diagnostics:** CXR reveals soft tissue swelling and absent pectoralis major muscle shadow but limited in diagnosis and characterization of ruptures; shoulder radiographs rule out bony avulsions/fractures. US shows uneven echogenicity and muscle thinning; useful when clinical examination is in question and prompt magnetic resonance imaging (MRI) not possible; MRI with appropriate sequences accurately defines extent of injury (grade), location, and amount of retraction; guides treatment plan

**Treatment:** Extent of tear may be difficult to diagnose because of ecchymosis, swelling, and extreme pain; serial examinations important; partial tear treated conservatively with initial ice, analgesia, sling for comfort, and activity restriction. Start with early protected ROM and gentle isometric strengthening. Regain full ROM to prevent further injuries, then resisted strengthening exercises by 6–8 weeks. Activities resumed slowly as allowed by pain and function; complete tear surgically repaired in competitive athletes, particularly those who depend on chest and shoulder strength; without surgical repair, weakness can result, particularly adduction and flexion; repair recommended in bodybuilders for improved cosmesis. Several cases present with delayed diagnosis and thus adhesions, muscle retraction, and atrophy, but late repair compatible with significant strength improvement. After surgery, immobilization for 4–6 weeks to protect repair; passive pendulum exercises and passive forward flexion with arm adducted to 130 degrees can begin immediately. Within 6–12 weeks, progress to full passive ROM and add an periscapular and isometric strengthening program (avoiding shoulder adduction, internal rotation, and horizontal adduction); by 12 weeks, resistive strengthening exercises begin; by 6 months, light free weights and push-ups

**Prognosis and RTP:** Surgical repair of distal pectoralis major tears results in almost full recovery of peak torque and work performed (97%). Full recovery of those managed nonsurgically 56%, but normal ADLs are not affected; nonsurgical management recommended for tears at sternoclavicular origin, although delayed repairs for persistent pain successful; RTP after surgery ranges 8 months to 1 year.

### Breast Injuries

**Description:** Contusions, hematomas, runner's/cyclist's nipple, and breast pain

**MOI and presentation:** Contusions caused by direct trauma with resultant bleeding and swelling; common in softball and basketball; nipple chafing, pain, eczema, and occasional bleeding from friction and abrasion by clothing during prolonged activity or evaporation of perspiration over the chest (see Chapter 40, Skin Problems); breast pain often experienced during athletic activity, particularly during running, because of strain on Cooper's ligaments (connective tissue that holds and supports breast on chest wall); query if pain is cyclical, and if so, its duration

**PE:** Examine for additional signs suggestive of other breast conditions, such as a mass, skin changes, or bloody nipple discharge. In runner's nipple, usually bilateral involvement with erythema, edema, oozing, crusting, and occasionally lichenification; systematically examine four breast quadrants in both lying and sitting positions with hands on hips and then above the head. Examine axillary, supraclavicular, and infraclavicular lymph nodes.

**Differential diagnosis/associated injuries:** Pectoralis major muscle strain, costochondritis, rib fractures if significant trauma, fibrocystic breast disease, cyclic mastalgia, contact dermatitis, bacterial or yeast infection of the nipple, Paget's disease, and breast cancer

**Up to 18% of women with newly diagnosed breast cancer had localized breast pain as a presenting symptom.** Individuals with concomitant atopy are predisposed to develop jogger's nipples. For those with breast augmentation and blunt chest trauma, implant rupture can lead to spherical capsular contracture.

**Diagnostics:** In general, diagnostic studies not required if no breast masses palpated and no nipple discharge; US should be considered if focal breast pain; mammography in women at a high risk of breast cancer

**Treatment, prognosis, and RTP:**

- See Chapter 40 for information on runner's nipple.
- **For contusions:** Ice, NSAIDs, and appropriate support; added protective padding. Hematomas rarely require aspiration.
- Can lead to posttraumatic scarring and retraction or thrombophlebitis of superficial veins (Mondor's disease); follow-up closely to differentiate from breast carcinoma
- Premenarchal athletic injuries to the breast bud (Tanner stages I–II; ages 10–11 years) can cause appreciable breast asymmetry (as much as one cup size or more).
- Hormonal therapy may help diminish breast tenderness during phases of menstrual cycle.
- Nicotine may increase breast pain; it increases epinephrine levels, which stimulates cyclic AMP, a regulator of mammary tissue metabolism.
- During pregnancy, specialized breast support is imperative as breasts can enlarge by 800 mL.

## Lung Injuries

### Pulmonary Contusion

**Description:** Blood and protein leak into alveoli and interstitial spaces, leading to atelectasis and consolidation (Fig. 52.3).

**MOI:** Blunt trauma

**Presentation:** CP, SOB, cough, and hemoptysis (see Fig. 52.3)

**PE:** Hypoxemia (hallmark clinical sign), tachypnea, rales, wheezing, and diminished breath sounds; PE may be normal.

**Differential diagnosis/associated injuries:** Other causes of CP and hemoptysis (pulmonary embolism and pneumonia); with history of chest trauma, injury to pulmonary parenchyma most likely; flail chest greatly associated with pneumothorax or hemothorax; also pulmonary contusion and aortic rupture

**Diagnostics:** Initial CXR may not show severity of injury; nonsegmental patchy infiltrates or consolidation evident 4–6 hours and occasionally up to 48 hours after injury (see Fig. 52.3). In addition, CXR underestimates the degree of contusion. CT scan highly sensitive; pulmonary contusions detected twice as frequently with CT compared with CXR. However, CXR

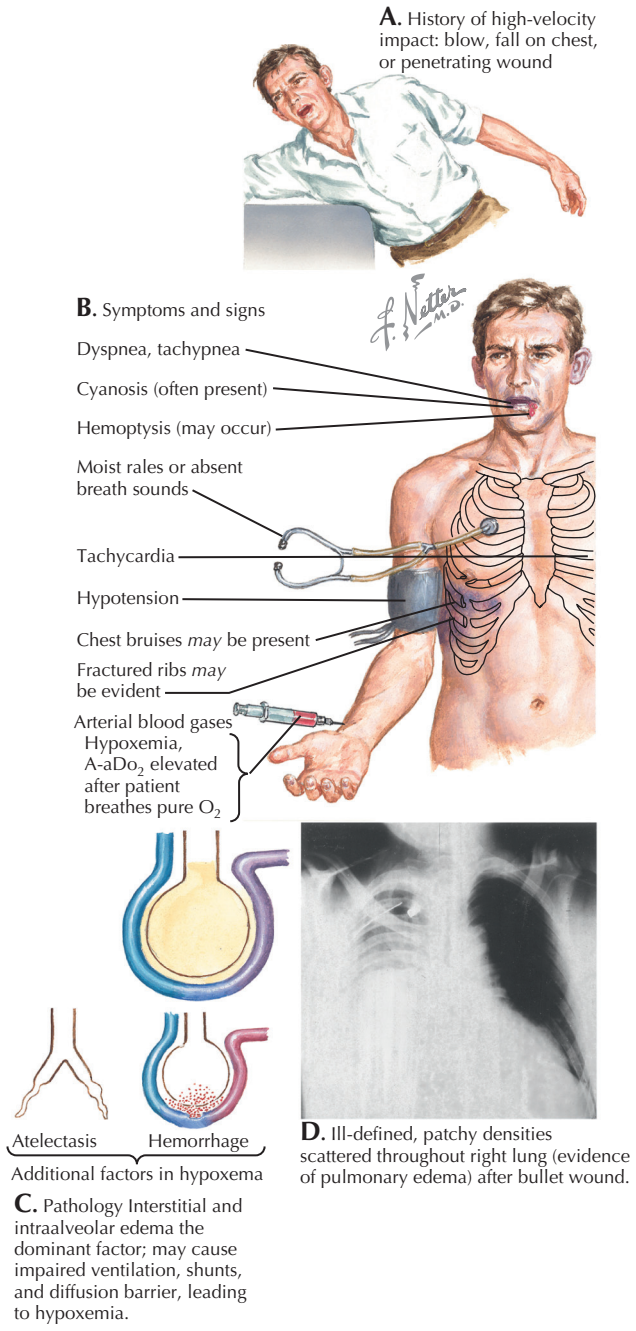



**Figure 52.3.** Pulmonary contusion.

provides clinically valuable information at minimal cost, and should be considered primary imaging tool. Pulse oximeter or blood gas analysis helpful

**Treatment:** Assisted ventilation; endotracheal intubation if necessary. Watch for onset of pneumonia and acute respiratory distress syndrome (ARDS). Severe thoracic or abdominal trauma represents a major risk factor.

**Prognosis and RTP**: No sport-specific guidelines exist. With mild pulmonary contusion and no CXR findings, gradual return to progressive activity after symptoms resolve (within 2–10 days). Traumatic pseudocysts can develop; follow-up imaging to avoid an infection and possible surgical excision. Consider flak jacket for contact sports. Those who recover even from severe pulmonary contusions do not suffer any significant late respiratory problems.

## Pneumothorax

**Description:** Refers to air within chest cavity in pleural space (separation of the visceral and parietal pleura), which leads to collapse of lung; classified as **spontaneous** or **traumatic**; described by the approximate percentage of hemithorax occupied by free air (e.g., 10%, 50%)

**MOI:** Blunt trauma most common cause, usually associated with rib fracture(s); spontaneous pneumothorax occasionally precipitated by strenuous physical activities, particularly in tall, thin, young males who smoke; the risk increases by defects in lung periphery; these bullae usually located in the lung apex

**Presentation:** Gradual or sudden pleuritic CP and dyspnea (depending on size and rate of collapse of lung); pain referred to shoulder tip

**PE:** Shallow, rapid respirations; cyanosis; tachycardia; hyperresonance to percussion and decreased or absent breath sounds over affected lung; tracheal shift to contralateral side; possible subcutaneous emphysema

**Differential diagnosis/associated injuries:** Traumatic rupture of left hemidiaphragm with herniated stomach bubble mistaken for loculated pneumothorax; skin folds act as "Mach bands" and presumed to be visceral pleural lines

**Hemothorax:** Blood accumulates in the pleural space as a result of bleeding of intercostal or mammary blood vessels and lung parenchyma injury; massive bleeding with aortic or myocardial rupture and injuries to the hilar structures; usually associated with pneumothorax. Symptoms include dullness to percussion, decreased breath sounds, and hypotension.

**Pneumomediastinum:** Excessive intra-alveolar pressures (from exacerbation of asthma, coughing, vomiting, seizures, Valsalva maneuver) lead to rupture of perivascular alveoli; air escapes into surrounding connective tissue, with dissection into the mediastinum. Symptoms include CP, persistent cough, sore throat, and substernal CP radiating to the back, neck, or shoulders. Subcutaneous emphysema is the most consistent physical finding.

**Diagnostics:** Upright PA CXR shows absence of lung markings in the periphery and increased density of the collapsed part of the lung. The white visceral pleural line is evident. A lateral width of 1 cm corresponds to a 10% pneumothorax. Inspiration and expiration radiographs may facilitate clear visualization of small pneumothorax lesions; a small pneumothorax on the nondependent side more easily detected in the lateral decubitus view; mediastinal shift seen with large pneumothorax; based on emergency medicine (EM) literature, thoracic US more sensitive than supine CXR and as sensitive as CT scan in detecting traumatic pneumothoraces

**Treatment:** If minimal (15%–30%), stable, and asymptomatic, observation with serial examinations and CXR; avoid unnecessary physical exertion; if large enough to cause SOB and discomfort, transport to hospital for possible insertion of chest tube. In **open pneumothorax** with chest wall defect, ambient air enters the injured hemithorax during inspiration, and mediastinum shifts to the uninjured side. In expiration, the mediastinum swings back to the injured side, and expiratory air from the normal lung enters the collapsed lung. Place foil, cloth, or other item over the wound, securing it only on three sides to avoid development of tension pneumothorax.

**Prognosis and RTP:** No vigorous activities for 2–3 weeks after chest tube removal; gradual and monitored return to activity. Educate regarding the risk of further episodes (reported in up to 50% of cases of primary spontaneous pneumothorax). No published data to suggest an increased risk for recurrence of traumatic pneumothorax. Review appropriate breathing techniques during the sport.

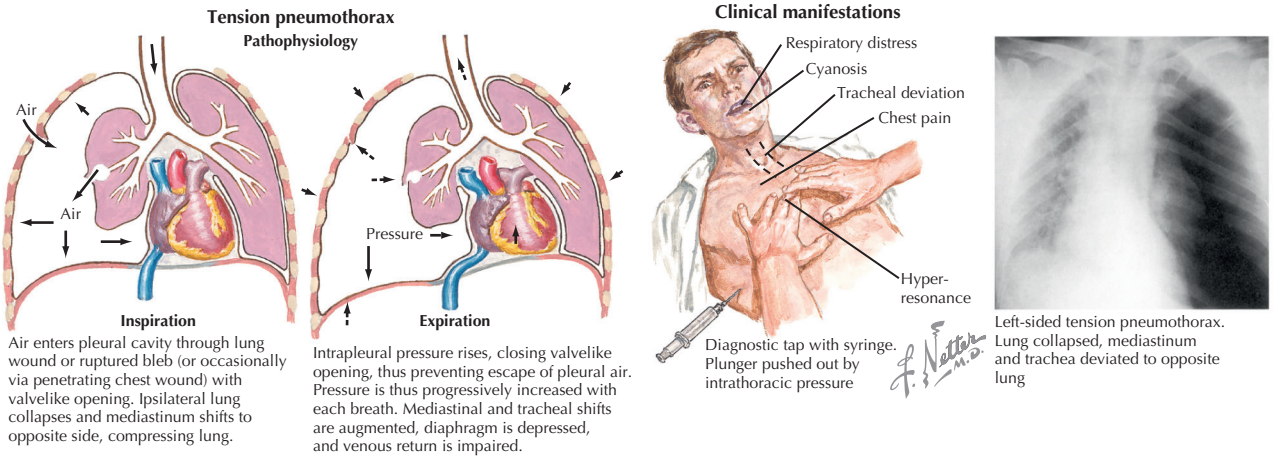



**Figure 52.4.** Tension pneumothorax.

## Tension Pneumothorax

**Description:** Progressive enlargement of pneumothorax because of communication between airways or exterior and interpleural space (Fig. 52.4); **flap valve effect** created; with inspiration, air is drawn into pleural cavity, whereas with expiration, air stays trapped. Positive intrapleural pressure develops, shifting the mediastinum, and further impairs ventilation of the compressed noninjured lung. **Absolute medical emergency**; progressive hypoxia and hypotension lead to death.

**MOI:** Similar to pneumothorax

**Presentation:** Rapidly increasing SOB, asymmetry of respiration (see Fig. 52.4)

**PE:** Distended neck veins, cyanosis, hypotension and tachycardia, dyspnea and tachypnea, shift of trachea away from injured side, and absent breath sounds on the involved side; hyper-resonance on percussion of the involved side

**Diagnostics:** CXR (only if immediately available) shows a distinct shift of the mediastinum to the contralateral side and flattening of the ipsilateral hemidiaphragm.

**Treatment:** Usually emergently treated before confirmatory chest radiograph with needle decompression; large-bore needle (14–16 gauge) inserted into the 2nd intercostal space in the midclavicular line on the affected side, just over superior aspect of the rib to avoid intercostal vessels; transport to a hospital, and chest tube placement for definitive treatment. If breathing is spontaneous, hemodynamically stable, and no evidence of respiratory compromise, obtain portable CXR, then place a chest tube once diagnosis confirmed

**Prognosis and RTP:** No specific RTP guidelines; treated as discussed previously, with a gradual increase in progressive activities

## Cardiac and Great Vessel Injuries

### Myocardial Contusion

**Description:** Most common cardiac complication of blunt chest trauma; can lead to impaired circulation, arrhythmia, or bleed into pericardium, resulting in cardiac tamponade at the time of injury or as a late complication several weeks after injury; in general, myocardial contusion refers to structural damage of the heart as described in this section; commotio cordis refers to a sudden disturbance in the heart rhythm via mechanical impact in the absence of structural damage (see Chapter 35, Cardiac Disease in Athletes).

**MOI:** Blunt trauma causes "bruising" of cardiac muscles; most commonly reported from baseball or hockey puck

**Presentation:** Crushing and sudden deceleration injuries; impact to the anterior chest wall and sternum; difficult to diagnose because signs typically transient and vary with degree of myocardial damage; athletes may experience minor CP or sudden cardiac arrest. CP is nonpleuritic; relieved by oxygen but not nitroglycerin

**PE:** Tachycardia, arrhythmias, and signs of decreased cardiac output; cardiac examination usually normal but may show friction rub or murmur

**Differential diagnosis/associated injuries:** Differential diagnosis includes acute coronary occlusion; may be obvious external chest wall injury such as sternal fractures

**Diagnostics:**

- **ECG:** Findings are nonspecific, but 70%–85% show abnormalities of ST segment and T-wave changes, sinus tachycardia, and intraventricular conduction disturbances. Seen on initial evaluation and subsequent 3 days after injury; usually transient
- **Biomarkers:** A negative troponin I assay in addition to a normal ECG is required to exclude myocardial injury; CK-MB is neither sensitive nor specific; no longer recommended
- **Holter monitor:** If arrhythmias present, most are premature ventricular contractions; atrial fibrillation and other supraventricular arrhythmias also noted
- **Radionuclide angiogram:** Depressed ejection fraction and segmental ventricular wall motion abnormalities; also nonspecific and not predictive of cardiac complications
- **Echocardiography:** Transthoracic echocardiogram (TTE) can narrow differential diagnosis of blunt chest trauma in the setting of rising cardiac markers; if TTE is limited owing to other injuries sustained in chest trauma, use transesophageal echocardiography. Echocardiography may be a more effective tool to follow suspected contusion and manage myocardial decompensation.
- **Cardiac catheterization:** Differentiate between myocardial contusion and coronary artery occlusion

**Treatment:** For mild contusion, admit for observation and watch for dysrhythmias (more likely to occur in initial 24 hours following injury); more severe contusion associated with other injuries may require invasive monitoring and inotropic medications

**Prognosis and RTP:** Gradual return to activity when stable and showing signs of fully healing from a diagnostic and clinical standpoint. Manufacturers have developed softer baseballs in an attempt to decrease incidence of morbidity and mortality. Use of chest protector, particularly during batting, is advocated.

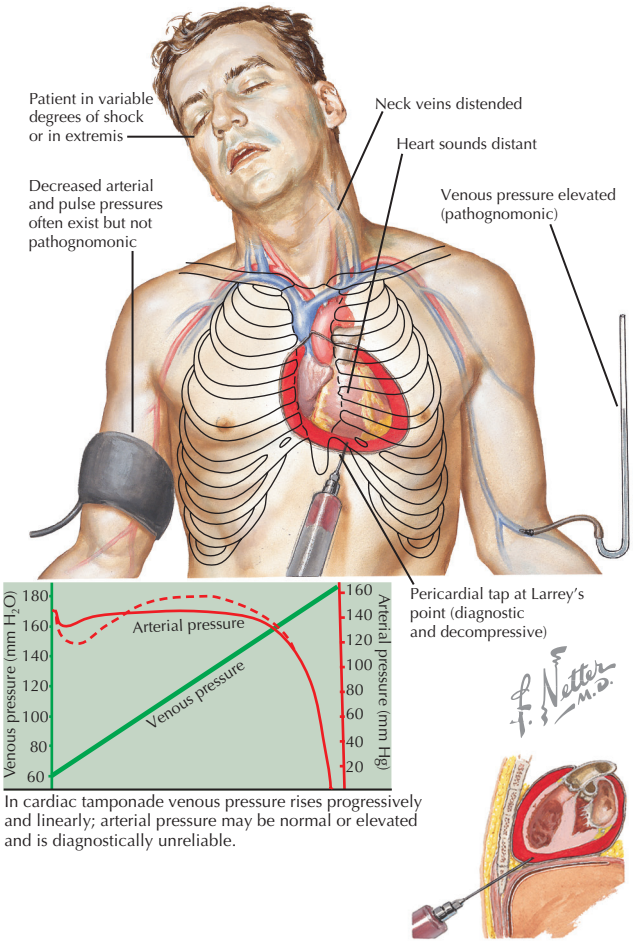



**Figure 52.5.** Cardiac tamponade.

## Cardiac Tamponade

**Description:** Accumulation of blood or edematous exudate into pericardial sac; volume and rate of accumulation of fluid determine symptoms; tension created within pericardial sac limits venous inflow and diastolic filling, and cardiac output diminished (Fig. 52.5); shock and death can rapidly evolve without early recognition and treatment.

**MOI:** Blunt trauma to chest, most commonly from high-energy collisions

**Presentation:** Symptoms can be variable but in acute cases, will present with dyspnea, tachycardia, and tachypnea; cold and clammy extremities caused by hypoperfusion

**PE: "Beck's triad"—hypotension, jugular venous distention, and distant heart sounds**, **pulsus paradoxus** (fall in blood pressure of >10 mmHg on inspiration)—tachycardia and weak pulse

**Diagnostics:**

- **Radiography:** Cardiomegaly, water bottle-shaped heart, pericardial calcifications, or evidence of chest wall trauma
- **ECG:** low voltage most common, also sinus tachycardia, electrical alternans, and PR segment depression
- **Echocardiography:** Can visualize pericardial effusion, but cardiac tamponade is the clinical diagnosis

**Treatment:** IV fluids and **urgent transport to a hospital**; pericardiocentesis guided by echocardiography, and emergent thoracotomy (see Fig. 52.5)

**Prognosis and RTP:** No evidence-based guidelines; decisions should involve a cardiologist and/or a cardiothoracic surgeon

## Coronary Artery Dissection and Occlusion

**Description and MOI:** Uncommon result of blunt trauma (e.g., high-speed vehicular collisions) resulting in a rapid deceleration of body; creates enormous shearing forces that may result in intimal dissection or disruption of coronary artery near its origin; intraluminal thrombus can form adjacent to injured arterial wall; can be asymptomatic; may cause angina, myocardial infarction, or death

**Presentation:** Dyspnea, diaphoresis, severe CP with or without radiation of pain, nausea

**PE:** Sinus tachycardia, hypotensive or hypertensive, may hear murmur particularly if injury affects valvular function or wall motion

**Diagnostics:**

- ECG: may show myocardial ischemia
- Echocardiography: evaluate wall motion abnormality
- Coronary angiogram: evaluate coronary vasculature

**Treatment:** Conservative, because lesion may heal with medical management: IV morphine, nitroglycerin; anticoagulation with heparin and acetylsalicylic acid; beta-blocker, statin, or later ACE inhibitor. Watch for postinfarction complications, including complete heart block, ventricular arrhythmias, left ventricular failure, ventricular aneurysm, and pulmonary emboli. With ongoing ischemia, coronary angiography can confirm diagnosis and assess extent of injury with possible percutaneous coronary intervention (PCI).

**Prognosis and RTP:** Dependent on extent of injury and treatment used; decisions should be made in conjunction with a cardiologist.

## Traumatic Aortic Rupture

**Description:** Approximately 80%–90% fatality rate at the accident scene; disruption extends through full thickness of aortic wall, with rapid exsanguination into mediastinum and pleural spaces; rupture sufficiently contained by adventitia to allow long enough survival to reach medical attention in only 20%; potentially higher risk with **Marfan syndrome** (cystic medial necrosis causes aortic dilatation); such athletes are restricted from contact and high-exertion sports.

**MOI:** High-speed, deceleration-type injuries (e.g., motor sports, bicycling, skiing, snowboarding); tremendous torques that result from sudden deceleration affect junction of fixed and mobile parts of great vessels, as between aortic arch and fixed descending aorta

**Presentation:** Acute onset anterior chest or interscapular pain that can migrate, dyspnea, hoarseness, occasionally, neurologic deficits seen; can be difficult to detect initially

**PE:** Hypertension in UEs with pulse pressure widening; with **ascending aortic injury,** aortic diastolic murmur radiating to back because of incompetence of aortic valve; acute coarctation syndrome in up to 25% of **descending aortic injuries:** caused by partial obstruction of aortic lumen, UE hypertension, diminution of femoral pulses and leg blood pressure, and systolic murmur

**Diagnostics:**

- **CXR:** Abnormalities present at time of admission in 75%–90%: widening of superior mediastinum and obscuring of aortic knob shadow most consistent findings

  Others: deviation of trachea to right, depression of left main stem bronchus, left apical extrapleural density, and left pleural effusion
- **ECG:** May show left ventricular hypertrophy or myocardial ischemia or infarction
- **TEE:** May be both sensitive and specific enough to plan management without aortogram
- **CT scan with contrast enhancement:** Often used in emergency department settings

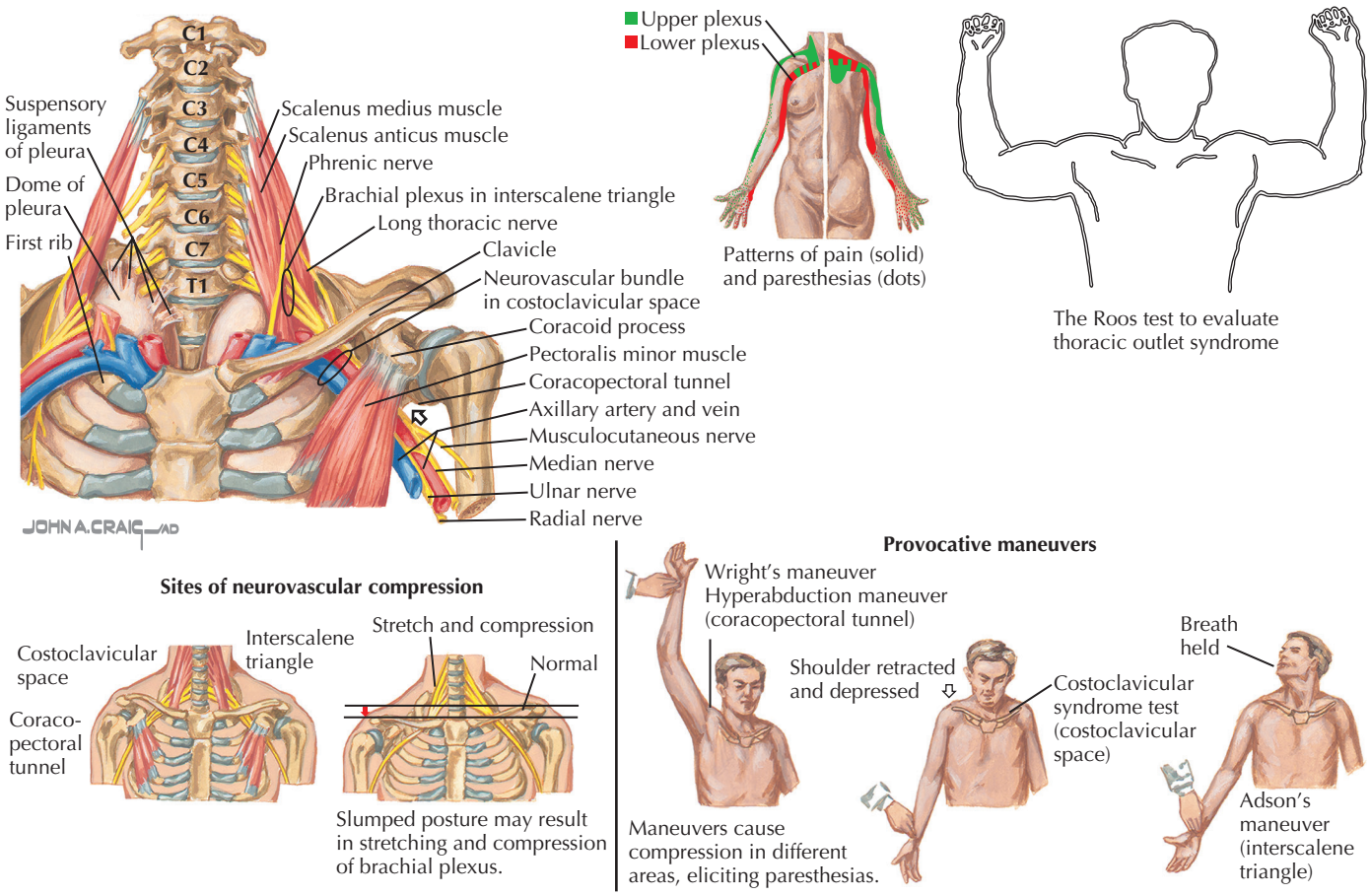



**Figure 52.6.** Thoracic outlet syndrome.

**Treatment:** Medical therapy to avoid sudden rupture, including antihypertensive agents and beta-blockers; emergent operative repair results in 75%–90% survival.

**Prognosis and RTP:** Mortality and morbidity rates for repair of this condition are among the highest in cardiovascular surgery; 80%–85% of patients die before arrival to the hospital; RTP decisions should be made with consultation of a cardiologist and/or cardiothoracic surgeon.

## Thoracic Outlet Syndrome (TOS)

**Description:** Spectrum of signs and symptoms resulting from compression of neurovascular bundle (brachial plexus and subclavian and axillary arteries and veins) in the interval between intervertebral foramina and axilla (Fig. 52.6); clinical presentations differ according to different degrees of compression. Greater incidence in women (male : female ratio, 4 : 1), perhaps secondary to lower position of scapula and changing shoulder posture with larger breasts

**MOI**: Related to areas of compression:

- **Supraclavicular region:** Interscalene triangle bordered by anterior and middle scalene muscles that attach to the 1st rib

  Contributing factors: hypertrophy of scalene muscles; long transverse process of C7, cervical ribs, or other rib anomalies; and fibrous bands changes in alignment and angulation of the 1st rib, as may occur with age and postural changes
- **Subclavicular or costoclavicular region:** Between the "mobile" clavicle and the "fixed" 1st rib; changes in shape and mobility of the clavicle, such as callus from fracture or alteration in shoulder motion, can narrow the interval; the subclavius muscle lies behind the clavicle and just anterior to the subclavian vein.
- **Infraclavicular region:** At the coracoid process of scapula; compression by pectoralis minor, which inserts at coracoid, during full abduction; subcoracoid area thickens the costocoracoid membrane; another contributing factor is any lesion involving the pleura, such as neoplasm.

**Presentation:**

- **Neural compression symptoms:** Pain from the root of neck to the shoulder and diffusely down the arm; if lower trunk of brachial plexus compressed (most common), paresthesias involving medial aspect of elbow, forearm, and hand, particularly little and ring fingers; weakness and occasionally atrophy of affected hand; sometimes just a vague ache and heaviness in the shoulder, upper arm, and upper anterior and posterior chest, including trapezius and suboccipital region; if the patient sleeps with arms above head, may awaken with symptoms at night
- **Arterial compression symptoms:** Hand feels cold and arm becomes numb and fatigued with rapid overhead movement
- **Venous compression symptoms:** Swelling and discoloration of arm after exercise and prominent superficial venous pattern over ipsilateral the shoulder and chest

**PE:** Careful examination of neck, cervical spine, shoulder, elbow, and hand; check supraclavicular fossa for masses or bruits; careful neurologic examination

- **Adson's maneuver:** Arm abducted and externally rotated while head extended and turned to side of lesion; radial pulse monitored while deep breath is taken and held; positive test equals diminution or total loss of pulse; positive in up to 30% of asymptomatic population

- **Wright's maneuver:** Similar to Adson's except arm is hyperabducted, with hand brought over the head with the elbow and arm in the coronal plane; 70%–90% sensitivity and 29%–53% specificity
- **Military brace position or costoclavicular syndrome test:** Shoulders are retracted and pulled down to narrow interval between clavicle and the 1st rib and reproduce symptoms, including absence of radial pulse; an effective test in those with symptoms when wearing backpack
- **Overhead exercise test (Roos test):** Arms are abducted to 90 degrees, shoulders externally rotated, and elbows flexed to 90 degrees (see Fig. 52.6); hands opened and closed slowly for 3 minutes to reproduce symptoms of fatigue, cramping, numbness and tingling, or coolness or paleness of extremity; false-positive rate as high as 47% in normal individuals

**Differential diagnosis/associated injuries:** Shoulder instability and "dead arm syndrome"; cervical spine pathology; peripheral neuropathies; Raynaud's phenomenon (can be present with both TOS and collagen vascular diseases); complex regional pain syndrome; Pancoast's tumor or other space-occupying lesion in thoracic outlet; **myofascial syndrome** (periscapular region, base of neck, and chest wall are common areas of pain and fatigue; trigger points found in supraspinous fossa near rhomboids, levator scapula, and infraspinatus can cause pain to radiate down arm)

**Diagnostics:** Cervical spine radiographs to assess for cervical ribs; CXR to evaluate lung apex; electrodiagnostic studies (difficult to accurately determine nerve conduction velocity at thoracic outlet; perform with arm in provocative position; helpful in ruling out peripheral nerve entrapment); CT angiography and venography have supplanted conventional venography or arteriography to evaluate vascular structures. US can be useful to evaluate for arterial and venous TOS, but is operator dependent; MRI is optimal for brachial plexus nerve compression and can be combined with MR angiography to evaluate vascular findings.

**Treatment:** Conservative management results in 50%–90% recovery. Strengthen shoulder girdle suspensory muscles; stretch scalenes, lateral neck flexors, and pectoral muscles. Other measures include weight reduction, posture training, appropriate brassiere support. Avoid hyperabduction of shoulder and carrying heavy packages in the affected hand. Surgery considered only if diagnosis is firm and conservative treatment has failed (3–4 months), with symptoms of intractable pain or major neurological or vascular complications. First rib resection is most dependable, but surgical procedure varies depending on the anatomical basis for symptoms.

**Prognosis and RTP:** Conservative treatment generally results in recovery from injury; RTP generally guided by the patient's clinical symptoms

### Vascular Injuries of Subclavian and Axillary Veins

**Description: "Effort thrombosis"** (Paget–Schroetter syndrome), or primary thrombosis, describes traumatic thrombosis of the subclavian or axillary vein

**MOI:** May occur after single traumatic event around shoulder or clavicle (clavicular fracture, axillary hematoma, injury to axillary or subclavian vein); **more commonly associated with repetitive overhand motions causing trauma to a vessel** (e.g., hyperabduction, external rotation); primary thrombosis is related to inherent anatomic structure of thoracic outlet and axillary region, with compression at various points causing damage to vein walls.

**Presentation:** Pain and diffuse swelling in arm; numbness, heaviness, and easy fatigability; distention of superficial arm veins with cyanosis (bluish discoloration) of skin; onset of symptoms commonly within 24–72 hours of activity

**PE:** Increased girth of the affected UE, and perhaps weakness of the UE, particularly if venous occlusion long-standing; symptoms often reproduced with exercise test of the affected UE or by putting arms into extreme hyperabduction

**Differential diagnosis/associated injuries:** Rule out secondary causes of thrombosis (sarcoidosis, infection, drug use, hypercoagulable states, and metastatic tumor) and poor circulation; arterial occlusion, including **aneurysms of subclavian or axillary arteries**, also reported in athletes; classic symptom is early fatigue during act of throwing; other symptoms: absent pulses, cyanosis, decreased skin temperature, and finger ischemia secondary to digital embolization. If UE thrombosis is confirmed, watch for signs of pulmonary embolus (incidence approximately 12%).

**Diagnostics:** Venogram, compression ultrasonography or duplex Doppler studies (most accurate noninvasive tests), CXR, and cervical spine radiography (AP view); given the frequent bilateral presentation, consider evaluation of both UEs.

**Treatment:** Simultaneous anticoagulation with IV or low-molecular-weight heparin and subsequent warfarin therapy for 1–3 months; thrombolysis with fibrinolytic agents such as streptokinase or urokinase; thrombectomy and surgical correction of involved thoracic outlet and axillary structures if documented external compression; high incidence of late morbidity, such as swelling, pain, fatigability, and numbness, particularly with conservative therapy or anticoagulation alone; can become asymptomatic if compensatory collateral veins develop; promising short-term results with thrombolysis alone or in combination with surgery

**Prognosis and RTP:** No participation in contact sports while on oral anticoagulants

## Abdominal Injuries

### Rectus Sheath Hematoma

**Description:** Major muscle groups of abdominal wall are rectus abdominus muscles, external and internal obliques, and transverses.

**MOI:** Direct blow to abdominal wall, causing hemorrhage into muscle; may damage either epigastric artery or intramuscular vessels, causing hematoma within sheath, which usually self-tamponades; with violent stretching movements, the inferior epigastric artery can rupture and hemorrhage without associated indirect injury to muscle tissue.

**Presentation:** Sudden abdominal pain particularly with trunk flexion or rotation, local tenderness, rapid swelling, greatest comfort in supported flexed position; may have nausea and vomiting. Rectus sheath hematoma can mimic an acute abdomen.

**PE:** Abdomen may be somewhat rigid with guarding, increased tenderness over rectus; fixed (within the rectus sheath) palpable mass most often below umbilicus in sitting or lying position; other signs: bluish discoloration around periumbilical region 72 hours after injury (Cullen's sign) and pain with resisted trunk or hip flexion; hyperextension of spine causes pain in the anterior abdominal wall

**Diagnostics: Laboratory studies:** Complete blood count (CBC), international normalized ratio (INR), protime (PR), and partial thromboplastin time (PTT). Based on Emergency Medicine (EM) literature, US can be a useful diagnostic tool. MRI or CT scan may be used to assist in differentiation between intra-abdominal injury and hematoma (Fig. 52.7).

**Treatment:** Ice, activity modification, and NSAIDs; local heat fermentation after 48–72 hours. Avoid activities requiring rotation, stretching, or flexion of trunk or lower extremities. Rehabilitation concentrates on restoring flexibility, strength, and endurance of all abdominal muscles. If the hemorrhage

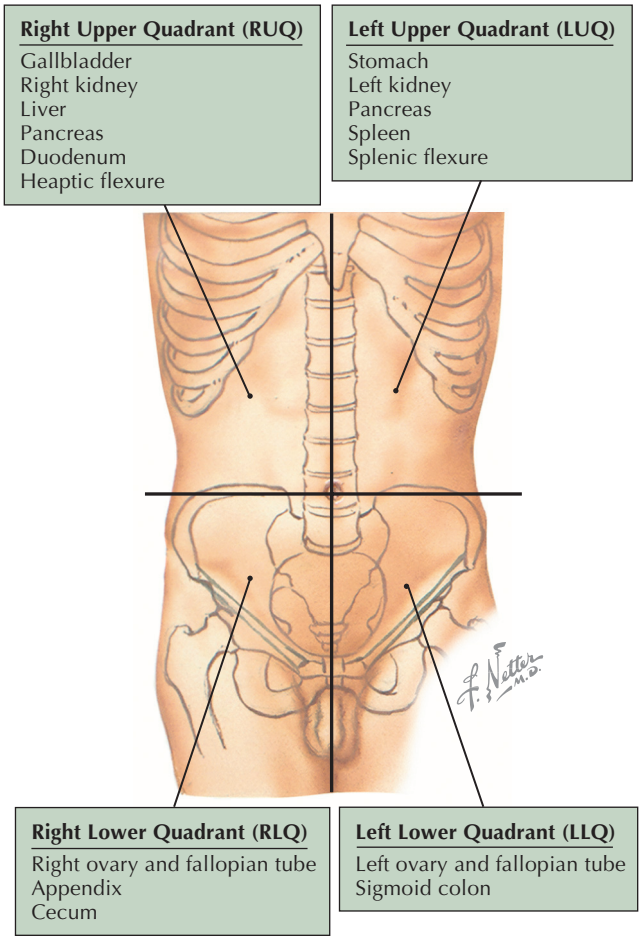



Figure 52.7. Quadrants of abdomen.

extensive and superficial epigastric artery is torn, may require operative evacuation of hematoma and ligation of artery

## Rupture of Diaphragm

**Description:** Herniation of abdominal contents into chest (Fig. 52.8); four times more common on the left side with blunt trauma (there is some degree of protection from liver on right); easily overlooked because of delayed onset of symptoms

**MOI:** Blunt chest or abdominal trauma

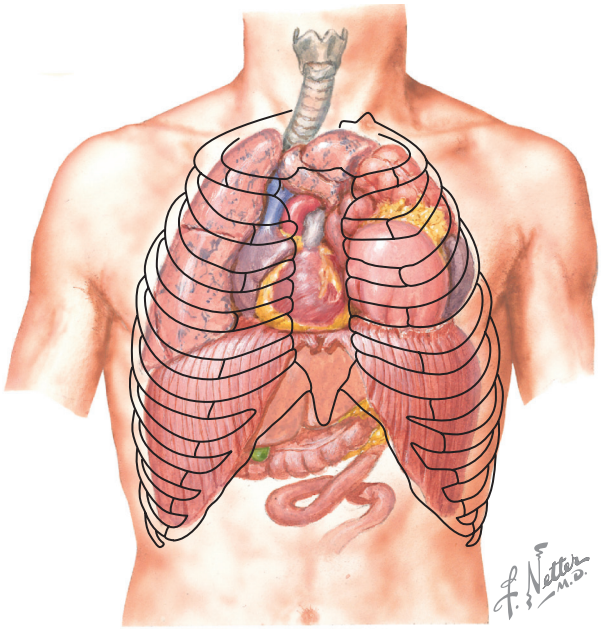
**Diagnosis:** CP, SOB, and intestinal obstruction

**PE:** Decreased breath sounds, excessive percussion of tympany in chest; bowel sounds in chest and scaphoid abdomen

**Differential diagnosis/associated injuries:** Hemothorax, pneumothorax, pulmonary contusion, blunt thoracic aortic tear, and elevated hemidiaphragm due to other reasons; splenic rupture in 25% of patients with blunt diaphragmatic rupture, liver lacerations in 25%, pelvic fracture in 40%, rib fracture in 52%, and thoracic aortic tears in 5%

**Diagnostics:** CXR and abdominal films show dilated stomach in the lower chest. Presence of nasogastric tube terminating in air space is confirmatory. Abdominal CT with reformatted images: for left-sided hernias, 78% sensitivity and 100% specificity; for right-sided hernias, 50% sensitivity and 100% specificity; MRI with clinical suspicion and indeterminate chest radiography and CT findings

**Treatment:** Immediate surgical repair; left-sided injuries explored transabdominally because of high incidence of associated intra-abdominal injuries; right-sided injuries explored transthoracically because of location of liver; watch for complications resulting from pneumonia and abscess



May result from blunt impact or compression or from penetrating wound. Stomach and other abdominal viscera herniated into left thorax; left lung collapsed, right lung compressed; mediastinum shifted and trachea deviated to right.

Figure 52.8. Rupture of diaphragm.

**Prognosis and RTP:** Complications include missed diaphragmatic injuries; delayed diagnosis may result in intestinal herniation, ischemia, and necrosis; no RTP literature

## Splenic Rupture

**Description:** The spleen is the most commonly injured organ in sport and the **most frequent cause of death related to abdominal injury in sport.** Although rib cage offers a certain amount of protection, rib fractures can leave spleen more vulnerable to injury. The spleen's capacity for encapsulating bleeding delays overt signs and symptoms of rupture; may be days before clinical deterioration. Spleen can enlarge and weaken during certain illnesses (infectious mononucleosis and sarcoidosis), making it more susceptible to rupture.

**MOI:** Direct trauma to the left lower chest from falls, sporting injuries, or MVAs

**Presentation:** Initial sharp pain in left upper abdomen, then continuation of dull, left-sided flank pain; abdominal distention; referred pain to either right or, more commonly, left shoulder (**Kehr's sign**) from free intraperitoneal blood irritating the diaphragm. Neck pain may be referred from phrenic nerve pressure (**Seagasser's sign**).

**PE:** Generalized abdominal tenderness; may have rebound tenderness and rigid abdomen; may have tenderness over 10th, 11th, or 12th ribs; tachycardia, hypotension, diaphoresis, and rapid respirations suggest internal bleeding; fixed dullness in the left flank (**Ballance's sign**).

**Differential diagnosis/associated injuries:** Left-sided 11th and 12th rib fractures and abdominal contusions

**Diagnostics: Imaging studies:** Flat-plate abdominal radiographs may show fading splenic outline and growing splenic shadow. CT scan with contrast (greater sensitivity and specificity and greater anatomical detailing of spleen and surrounding structures than radionuclide scan) is the current diagnostic imaging standard; US; arteriogram; peritoneal lavage (useful but can miss subscapular tear)

**Laboratory studies:**
- Decreased hemoglobin and hematocrit levels
- Markedly elevated white blood count if subcapsular hematoma has developed
- Diagnostic peritoneal lavage is positive unless bleeding is encapsulated (10 mL of free blood, >100,000 red blood cells/$mm^3$, or >500 white blood cells/$mm^3$)

**Treatment:**
- If splenic injury suspected, immediate transport to hospital; if hypotensive, give bolus of IV fluids; keep flat or in modified Trendelenburg position to direct blood flow to heart
- Various grading systems, based on anatomic location of splenic injury on CT scan, help guide treatment and predict outcomes. If hemodynamically stable and splenic injury is minimal or subcapsular, nonsurgical management with observation in critical care setting is the preferred treatment; splenic preservation is preferred over splenectomy, particularly in pediatric population.
- Exploratory laparoscopy/laparotomy indicated if continuing hemodynamic instability or if require >4 units of blood during a 48-hour period; first choice: repair of capsular lacerations (splenorrhaphy); splenectomy only for extensive injury and uncontrolled hemorrhage; after splenectomy, vaccinate for encapsulated organisms (*Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitides*).
- Beware of **"delayed rupture"** of spleen; occurs >7 days after initial negative CT scan; need high index of suspicion and liberal utilization of other imaging techniques

**Prognosis and RTP:** After 3 months in nonsurgical patients (may be longer depending on results of follow-up studies; interval studies useful in predicting RTP); after 3–6 months in postsplenectomy patients; postsplenectomy patients often return before those treated conservatively; may return to full activity when surgical scars healed and able to tolerate activity

## Liver Laceration

**Description:** Relatively rare in contact sports; usually results from high-speed accidents during motor racing and skiing; capsular hematoma most common liver injury in athletes

**MOI:** Blows to right upper quadrant (RUQ)

**Diagnosis:** Pain in RUQ, right shoulder, or neck pain

**PE:** Pain and tenderness to palpation over RUQ; tachycardia and hypotension; may be associated with right lower rib fractures

**Differential diagnosis/associated injuries:** Pancreatic injury; obtain serum amylase levels

**Diagnostics:** CT scan with contrast enhancement, US, arteriogram, liver enzymes (AST or ALT 130 IU/L indicates liver injury), and peritoneal lavage

**Treatment:** Grading systems based on anatomical location of lesions on CT scan help determine treatment and prognosis; 50%–80% of liver injuries stop bleeding spontaneously. Rest, observation, and IV fluids for hemodynamically stable patients with no signs of peritoneal irritation and no other intra-abdominal injuries that may require surgical repair; laparotomy may be necessary to control bleeding.

**Prognosis and RTP:** Length of time varies; interval radiographic studies help predict; RTP guidelines not established; athletes must show anatomic and functional healing before participation.

## Rupture of Stomach and Intestines

**Description:** Such injuries are rare.

**MOI:** Kicks or blows to abdomen; falls off horse or against equipment, as in gymnastics; pile-ons and spearing in football; handlebar accidents in cycling; diving decompression accidents

**Presentation:** Persistent abdominal pain with signs of chemical or bacterial peritonitis, including fever, nausea, and vomiting; referred shoulder pain from irritation of diaphragm; may have blood in stool if intramucosal hemorrhage present

**PE:** Localized pain, guarding, rebound tenderness, absence of normal bowel sounds, rigid abdomen; clammy, sweaty skin; hypotension and tachycardia; absence of normal respiratory motion of abdomen; gross or occult blood on digital rectal examination

**Associated injuries:** Intramural hematoma of duodenum may manifest as gastric outlet or high small bowel obstruction

**Diagnostics:** Plain radiograph with upright and decubitus abdominal views shows free air under diaphragm or along abdominal wall; keep athlete in appropriate positions for 3 minutes before views are taken; peritoneal lavage: not helpful for duodenal or large intestine injuries because of retroperitoneal position; nasogastric tube placement to check for blood if damage to stomach suspected; meglumine diatrizoate (Gastrografin) swallow; CT scan

**Treatment:** Urgent transport to hospital if increasing pain, signs of peritonitis, or circulatory collapse develop; infuse IV fluid until transport arrives; serial examinations, nasogastric tube, and IV fluids; abdominal exploration and repair

**Prognosis and RTP:** Length of time may vary; RTP guidelines not established; decisions should be made with a general surgeon

## Pancreatic Injury

**Description:** Injuries rare; pancreas is relatively immobile and in the protected retroperitoneum; injuries to pancreas most often occur with direct contact; reported injuries include lacerations and contusions to the body and duct of pancreas

**MOI:** Similar to injuries to stomach and intestine

**Presentation:** Abdominal pain; can diminish within initial 2 hours after injury then subsequently increase in the following 6–8 hours

**PE:** Abdominal or epigastric pain, abdominal wall ecchymosis; rebound tenderness is rare

**Diagnostics:** Often, normal amylase immediately after trauma; amylase neither sensitive nor specific for pancreatic injuries, but serial monitoring found to be more specific; lipase more sensitive; Hgb and Hct generally normal because blood loss usually minimal
- **Peritoneal lavage:** Not helpful because of retroperitoneal position of pancreas
- **Contrast-enhanced multislice CT:** Efficient screening modality for pancreatic trauma
- **Focused sonography for abdominal trauma (FAST):** Often used in trauma centers as screening in blunt abdominal trauma while checking for intraperitoneal fluid
- **Endoscopic retrograde cholangiopancreatography (ERCP):** Evaluates pancreatic duct; highly sensitive; risk of pancreatitis, hemorrhage, and gastrointestinal tract perforations
- **Magnetic resonance cholangiopancreatography (MRCP):** Noninvasive and accurate method of imaging pancreatic duct

**Treatment:** Principally supportive, IV fluid hydration, management of metabolic complications, fasting to avoid pancreatic stimulation, parental or enteral jejunal feedings

**Prognosis and RTP:** Higher morbidity and mortality when pancreatic injuries not recognized in initial 24 hours; no RTP guidelines; progressive RTP following anatomic and functional healing

## Hernias

**Description:** Three most common hernias in adults are indirect inguinal (50%–70%), direct inguinal (men >40 years), and femoral (women). Hernias involving anterior abdominal wall include incisional, periumbilical, and linea alba defects (spigelian hernias); potential for **incarceration** (irreducible hernia) and **strangulation** (twisting of hernia) causing bowel obstruction and toxicity

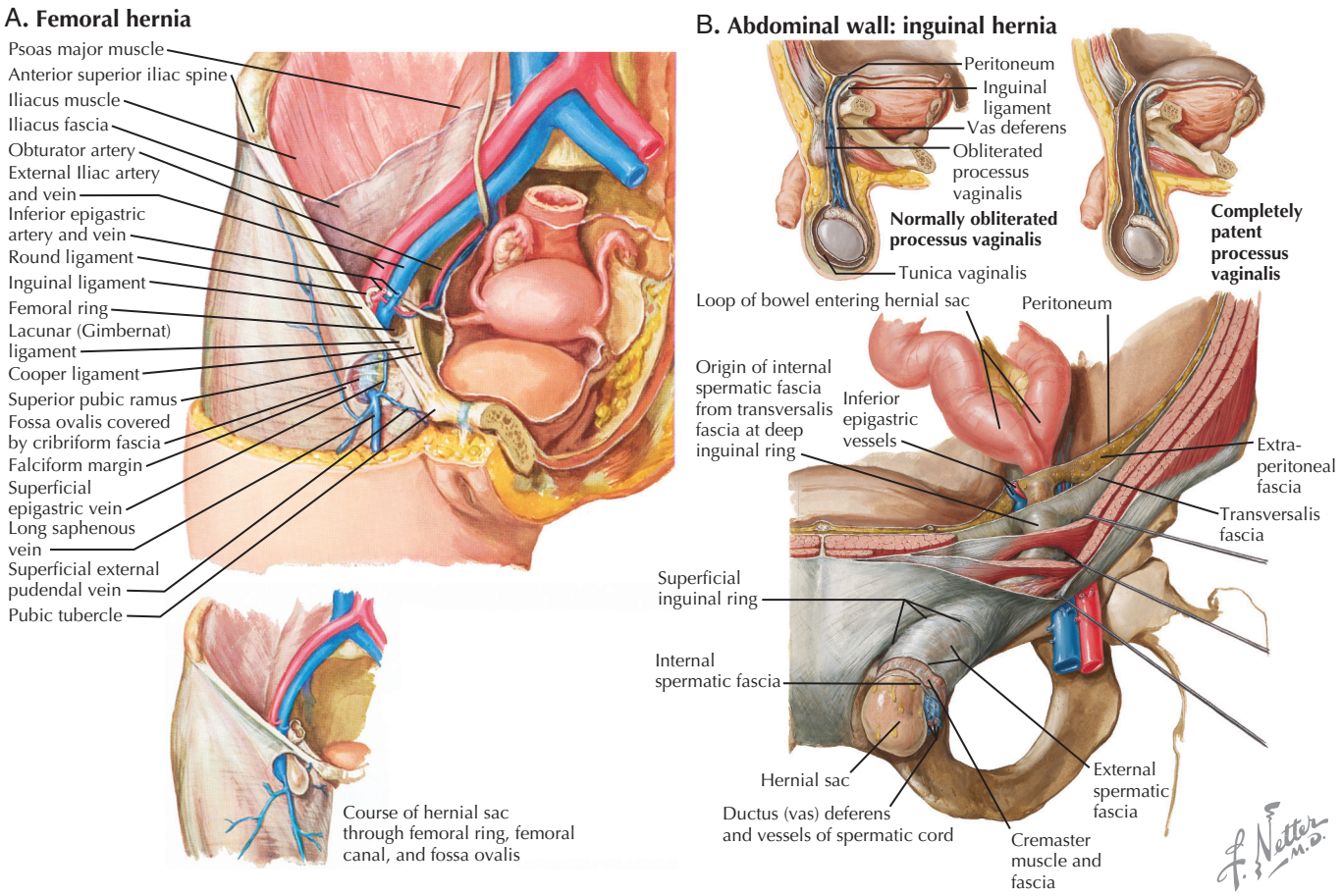



**Figure 52.9.** Hernias.

**MOI:** Repetitive heavy lifting activities cause increased intra-abdominal pressure, which can contribute to development of hernias, particularly with predisposing weakness of abdominal muscle and fascia; hernias also reported secondary to trauma, such as impact from bicycle handlebar on abdominal wall

**Presentation:** Aching sensation and occasionally tender swelling in area of hernia; may be scrotal swelling with indirect hernia as the sac extends into the inguinal canal

**PE: Indirect and direct inguinal hernias** are palpated by invaginating the scrotum with a finger to palpate external inguinal rings and inguinal canals. Athletes asked to do Valsalva maneuver; elliptical mass descending along the spermatic cord and bulging against the tip of finger is indirect hernia (Fig. 52.9); globular mass close to pubis that bulges against bottom of finger is direct hernia; **femoral hernias** occur below inguinal ligament, two fingerbreadths medial to femoral artery (see Fig. 52.9); hernias more prominent when athlete stands or increases intra-abdominal pressure

**Diagnostics:**

- **Herniography:** Intraperitoneal injection of contrast material to diagnose occult hernia sacs; 84% incidence of inguinal hernia by herniography in soccer players with groin pain; only 8% had hernias detectable by PE
- Bone scan: Increased uptake in musculature in initial phase
- MRI: Examine soft tissues in groin region
- CT scan: Contrastographic medium combined with CT scan
- US: Helpful in hands of experienced clinician

**Differential diagnosis/associated injuries:**

- **Iliopectineal/iliopsoas bursitis:** Groin pain reproduced by passive hip flexion caused by inflammation of bursa between pectineus and psoas muscle; pain can be localized over the lesser trochanter area; position of hip flexion and external rotation is most comfortable
- **Osteitis pubis inflammation of the pubic symphysis and surrounding muscle insertions thought to be secondary to repetitive microtrauma and/or shearing forces**
- **Posterior inguinal wall weakness (sports hernia, groin insufficiency)** presents as gradually worsening, poorly localized groin pain that is aggravated with activity. Herniography shows bulging or areas of weakness of wall. Dynamic US can demonstrate weakening of pelvic floor musculature with Valsalva. Surgical exploration reveals tears in the floor of the inguinal ring (transversalis fascia). Ilioinguinal nerve is occasionally trapped in the scar tissue within torn aponeurosis.
- Other: Muscular strain, hydrocele, or varicocele

**Treatment:** Treatment for athletic pubalgia is discussed in Chapter 54. Prompt surgical repair for large or symptomatic hernias; avoid activities that stretch or pull abdominal muscles for 2–4 weeks after repair; then gradually resume progressive exercise and conditioning; by 4 weeks, begin abdominal, back, and pelvic strengthening.

**RTP:** Return to noncontact sports after 6–8 weeks for indirect hernia repairs: contact sports at 8–10 weeks; more extensive repairs of direct inguinal, femoral, and anterior abdominal wall hernias require longer recovery: contact sports at 12 weeks

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# THORACIC AND LUMBOSACRAL SPINE INJURIES

# 53

Brock E. Schnebel

## GENERAL PRINCIPLES

- With an increased number of adults and adolescents participating in fitness programs and competitive sports, there has been an increase in thoracic and lumbar spinal problems.
- Most injuries are soft tissue injuries, and appropriate training and avoidance of aggravating activities may allow participation while the pain resolves.
- Treatment of an athlete can be complicated by his or her competitiveness and the fact that the athlete will be returning to the activity that precipitated the injury.
- The primary treatment objective is the protection and preservation of the nervous system.
- Spine injuries occur with a reported incidence of 7%–27% among sports-related injuries.

### Anatomy

- The spine is a mechanical structure consisting of bones, joints, ligaments, and muscles surrounding and distributing neural elements.
- The thoracic spine is more stable than the lumbar and cervical spine and is less mobile because of the thoracic cage.
- The spinal column is composed of 33 vertebrae divided into five sections: cervical (7), thoracic (12), lumbar (5), sacral (5), and coccygeal (4) (Fig. 53.1).

#### Osseous

**Vertebral body:** Large, strong, anterior weight-bearing structure (see Fig. 53.1)

**Posterior vertebral arch:** Semicircular-shaped structure surrounding the central canal, above and below the **vertebral foramen** through which the roots pass; composed of **pedicles,** which project dorsally off the body, one on each side, and the **lamina,** which connects the pedicles; from the pedicles and lamina, project the **transverse process**; from the lamina, project the **spinous process**; these are locations for muscular attachments. Each lamina articulates with the lamina above and the lamina below through **facet joints**. These **synovial-lined** facet joints are formed by a **superior articular process** from the lamina below and an **inferior articular process** from the lamina above and are surrounded by a facet capsule or ligament (see Fig. 53.1).

**Pars interarticularis:** Literally, the "part between the joints" or the area between these facet joints; relevant because it is the weak link anatomically and is susceptible to injury and stress fracture

**Sacrum:** Coalesced lower segments of the spine that articulate with the pelvis

**Costovertebral joints:** Area of articulation of the thoracic vertebrae with its rib

**Disc:** Major ligament connecting each vertebral body; composed of two types of tissue: annulus fibrosus (outer layer of fibers that functions to hold the nucleus pulposus and restrains the vertebral bodies) and nucleus pulposus (gelatinous structure located in the annulus)—both are responsible for load bearing

#### Ligaments

**Anterior longitudinal ligament:** Strong bond of fibrous tissue that runs the entire length of the spine along the anterior vertebral bodies (see Fig. 53.1)

**Posterior longitudinal ligament:** Weaker than the anterior ligament; runs along the posterior surface of the vertebral bodies

**Ligamentum flavum:** Very strong ligament attaching the lamina above to the lamina below

**Facet capsule:** Connects each superior articulating process with its corresponding inferior articulating process

**Interspinous ligament:** Connects each spinous process to the one below

**Supraspinous ligament:** Runs along the dorsal surface of the tips of the spinous processes

**Costotransverse ligaments:** Thoracic area

#### Muscles and Fascia

**Thoracolumbar fascia:** Investing tissue that separates the muscular compartments and fuses with the aponeuroses of several muscles (see Fig. 53.1)

**Anterior groups of muscles:** Anterior to the transverse processes; include psoas, intertransversalis, quadratus, and levator costae

**Posterior groups of muscles:** Posterior to the transverse process; include superficial muscles, erector spinae (semispinalis, longissimus, and iliocostalis), deep muscles, multifidus, rotares, and interspinalis

**Accessory group of muscles:** Includes abdominal muscles, latissimus dorsi, rhomboids, and gluteus maximus

#### Neural Elements

- Neural elements of the spine include the spinal cord from occiput to about L1, the conus medullaris or lower portion of the cord thickened by the mass required to innervate the lower extremities located from T11 to L1, and the cauda equina from L1 to the sacrum; the nerve roots exit at each level on both sides of the canal via the vertebral foramina (see Fig. 53.1).

#### Vascular Elements

- Vascular elements of the spine include a complex system of intradural and extradural arteries and veins that supply the neural elements.

### Biomechanics

- The spinal column has five principal functions:
  - Support of the head
  - Support of the abdominal contents and pelvic girdle
  - Point of attachment for the thoracic cage and muscles
  - Protection of the spinal cord and the neural elements within while allowing motion
  - The biomechanical transfer of the weight and bending movements of the head and trunk to the pelvis
- Understanding several basic concepts of biomechanics helps us understand how specific activities can exacerbate symptoms in certain syndromes:
  - In the spine, flexion of the lumbar spine increases the size of the intervertebral canal and the intervertebral foramina.
  - Extension decreases the size of the intervertebral canal and the intervertebral foramina.
  - Flexion increases dural sac and nerve root tension.
  - Extension decreases dural sac and nerve root tension.
  - Front flexion, axial loading, and an upright posture increase intradiscal pressure: pressure is greater while sitting, less while standing, and least while lying down.

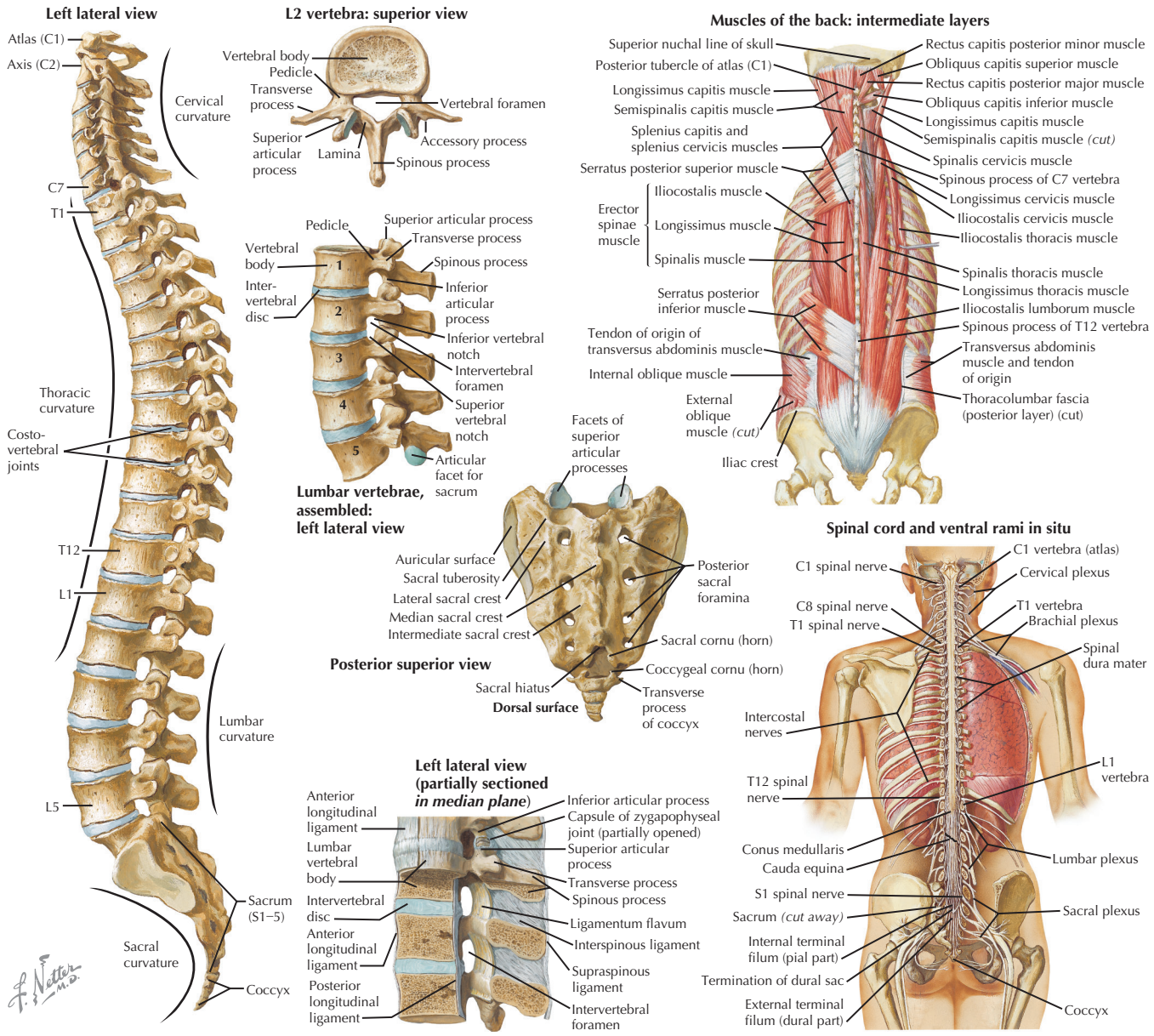



**Figure 53.1.** Anatomy of the spine.

- With flexion, the annulus bulges anteriorly.
- With extension, the annulus bulges posteriorly.
- Nuclear shift in an injured disc is poorly documented, but the disc probably shifts in the direction of the annular bulge.
- Rotation and torsion produce annular tears and disc herniations.

## History and Physical Examination

### History

- Information obtained during history taking and physical examination is critical. The following questions are particularly relevant.
  - **When and how did your symptoms start?** Mechanism of injury can help locate the damage. Onset can be telling because a gradual onset can be more related to a stress injury and a rapid severe onset may indicate a more acute injury.
  - **What is the location of the pain?** Neurologic involvement is likely when a patient perceives more pain in the leg than in the back. The percentage of back to leg pain is relevant. Associated neurologic symptoms such as numbness, dysesthesia, weakness, or spasticity are important to note. There are multiple nociceptive sources in the spine.
  - **When does it hurt?** Night pain may be more ominous and associated with tumors. Pain with motion and relieved by rest implies mechanical pain. Constant pain unaffected by rest may indicate an inflammatory component.
  - **What makes it worse?** Discogenic pain is usually worse with flexion or prolonged sitting and increases with a Valsalva maneuver (cough or strain with defecation). Pain from a spondylolysis is worse than that with hyperextension. Pain from spinal stenosis (pseudoclaudication) is worse with ambulation or prolonged standing. Sacroiliac (SI) joint dysfunction can be worse with hyperextension.

**TABLE 53.1 COMMON NERVE ROOT FINDING**

| Root | Strength | Sensation, Dermatomal Distribution | Reflex |
|---|---|---|---|
| L1–L2 | Iliopsoas | Inguinal area and upper two-thirds of anterior thigh | |
| L3 | Quadriceps | Oblique band of distal third of anterior thigh immediately above patella | |
| L4 | Tibialis anterior and quadriceps | Medial side of lower leg and foot | Patellar tendon |
| L5 | Extensor hallucis longus, and tibialis anterior | Anterior aspect of lower leg and dorsum of foot | Not reliable (posterior tibialis reflex present in only 40%–50% of population) |
| S1 | Peroneus longus and brevis gastrocnemius | Lateral aspect of lower leg and lateral foot | Achilles tendon reflex |

- **What makes it better?** Discogenic pain can be improved by lying down with knees and hips flexed (opens foramina and unloads disc). Spinal stenosis symptoms can improve by bending forward (as in walking over a grocery cart) or by sitting.
- **How do you train?** This may reveal a voluntary restriction that helps with a diagnosis or reveals a training error compatible with a specific syndrome.
- **Do you have any of these factors?** Bowel, bladder, or sexual dysfunction may indicate significant neurologic involvement of the cord, conus, or cauda equina. Weight loss, anorexia, night pain, and pain at rest may suggest neoplasia. Fever may indicate infection. A visceral type of pain with referral may indicate kidney, prostate, bowel, or vascular pathology.

### Physical Examination

- Should address inspection, palpation, and percussion and identify
  - Exact location of tenderness, dysesthesias, or numbness
  - Maneuvers that reproduce the pain
  - Presence of neural tension signs
  - Deficits in range of motion (ROM)
  - Any neurologic deficit

**Inspection of posture and stance:** Patients in a sitting position may tend to tripod or lean back to unload the spine. When standing, a list to one side may suggest nerve root compression. The appearance of a flat back with a vertical sacrum may be seen in advanced spondylolisthesis. Scoliosis may be detected in a standing position.

**Inspection of gait:** Myelopathic patients may walk with spasticity. A forward flexed posture can be seen in stenosis. Patients with discitis may have a rigid short-stride gait.

**Inspection of ROM:** May be limited by painful conditions; extension may be limited in stress fractures and facet syndromes. Discogenic pain may limit flexion.

**Palpation:** May detect muscle spasm, deformity with a palpable mass, scoliosis, spondylolisthesis, or a gibbus deformity; tenderness may present at the sciatic notch.

**Percussion:** May elicit tenderness in trauma or infection and costovertebral angle tenderness may imply a kidney problem

**Neurologic:** Most critical portion of the examination (Table 53.1 and Fig. 53.2); **upper motor neuron findings** of spasticity, weakness, numbness, hyper-reflexia, and clonus may indicate spinal cord pathology; **lower motor neuron findings** of flaccid muscles, weakness, numbness, hyporeflexia may indicate cauda equina or root injury. Sensory levels on the trunk will help determine the level of spinal cord injury, if present. A rectal and cremasteric reflex examination is recommended with any spinal cord injury.

## Special Tests

### Pain From Neural Source

**Straight leg raise (SLR) test:** Tension test that indicates nerve irritation in the sciatic nerve if positive with radicular pain at <60–70 degrees of leg elevation (Fig. 53.3)

**SLR test with foot dorsiflexion (Lasègue's test):** Tension test suggesting sciatic nerve irritation if painful

**SLR test while sitting:** Positive when extending the knee while patient is sitting causes radicular pain; patient may lean back to gain relief. A positive and a negative SLR test while sitting can be inconsistent and suggest other etiologies.

**Crossed SLR test:** Positive test may be pathognomonic for a herniated disc. With the patient sitting, the examiner lifts the unaffected leg; in the presence of a herniated disc, this may produce pain in the affected leg.

**Femoral stretch test:** In a side-lying position with knee flexed 90 degrees, the hip is extended, stretching the femoral nerve. Pain on the anterior thigh suggests femoral nerve or upper lumbar root irritation.

### Pain From Structural Source

**Jackson's one-legged standing hyperextension test:** If painful, it suggests a stress fracture (spondylolysis), a facet syndrome, or SI joint dysfunction.

**Flexion, abduction, and external rotation of hip (FABER) or Patrick's test:** Can suggest hip joint or SI joint dysfunction

**Supine-to-long sitting test:** Examiner tests leg length at the medial malleoli with the patient first supine then sitting with both legs extended at the knees. A change in relative leg length may indicate SI joint dysfunction.

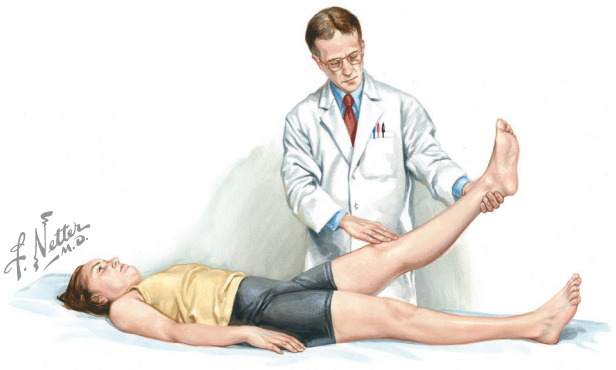
### Radiographic and Ancillary Testing

**Plain radiographs:** May be unnecessary in the acute phase in a nonathletic clinic setting, but in high-velocity and collision sports, early radiographic evaluation may be needed. Plain radiographs may reveal an acute fracture or spondylolysis that may alter the treatment. Plain radiographs may also reveal spondylolisthesis, degenerative changes, signs of infection in discitis (endplate irregularities), or congenital anomalies. Anteroposterior (AP) and lateral radiographs of the lumbar spine may be augmented by right and left obliques if a spondylolysis is suspected.

**Clinical Features of Herniated Lumbar Nucleus Pulposus**

| Level of Herniation | Pain | Numbness | Weakness | Atrophy | Reflexes |
|---|---|---|---|---|---|
| L3, L4, L5, S — **L3-4 disc; 4th lumbar nerve root** | Lower back, hip, posterolateral thigh, anterior leg | Anteromedial thigh and knee | Quadriceps | Quadriceps | Knee jerk diminished |
| L3, L4, L5, S, L5 — **L4-5 disc; 5th lumbar nerve root** | Over sacroiliac joint, hip, lateral thigh and leg | Lateral leg, web of great toe | Dorsifexion of great toe and foot; difficulty walking on heels; foot drop may occur | Minor | Changes uncommon absent or diminished posterior tibial reflex |
| L4, L5, S, S1 — **L5-S1 disc; 1st sacral nerve root** | Over sacroiliac joint, hip, posterolateral thigh and leg to heel | Back of calf; lateral heel, foot and toe | Plantar flexion of foot and great toe may be affected; difficulty walking on toes | Gastrocnemius and soleus | Ankle jerk diminished or absent |
| L4, L5, S, L5, S1, S2, S3, S4, S5, Coccygeal — **Massive midline protrusion** | Lower back, thighs, legs, and/or perineum depending on level of lesion; may be bilateral | Thighs, legs, feet, and/or perineum; variable; may be bilateral | Variable paralysis or paresis of legs and/or bowel and bladder incontinence | May be extensive | Ankle jerk diminished or absent |

**Figure 53.2.** Dermatomal distribution of nerves.



**Straight leg raising test**
Perform with the knee extended. Flex hip until resistance and/or pain is noted. Test places sciatic nerve, as well as hamstrings on stretch. Patients with herniated disc causing compression of sacral nerve roots often experience back pain radiating to lower extremity.

A *crossed straight leg raising test* is pain in the affected extremity when the contralateral leg is raised and is highly specific for nerve root entrapment.

**Figure 53.3.** Straight leg raise test.

**Bone scan:** May be used to reveal lesions that are not yet visible on plain radiographs (e.g., stress fractures in the pars interarticularis); because a positive bone scan indicates increased metabolic activity, this scan may reveal bone tumors or infection as well as occult fractures. It may take up to 5 days after the onset of symptoms for a bone scan to be positive.

**Computed tomography (CT) scan:** To evaluate osseous lesions and to help stage stress reactions

**Magnetic resonance imaging (MRI):** To help detect discogenic lesions such as degenerative disease with desiccation, disc herniations, spinal cord lesions, fractures, stress reactions, and compressive lesions

**Myelography and CT myelography:** May also be used to show compressive lesions

**Electromyography (EMG) and nerve conduction velocity (NVC):** May help in localizing the location of a nerve compression lesion; it may take up to 3 weeks before denervation changes occur such that they are revealed on an EMG.

**Laboratory studies:** Can be useful in diagnosing discitis, inflammatory spondylitis, or neoplasia, but these studies are rarely needed

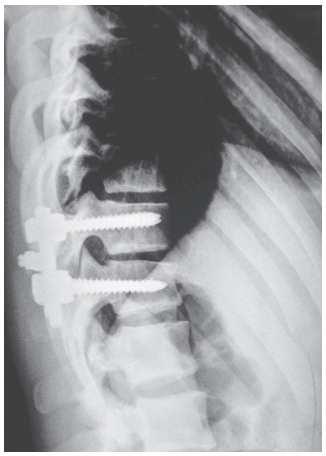
## SPECIAL INJURIES AND PROBLEMS

### Traumatic Injuries to the Thoracic or Lumbar Spine

**Description:** Motor sports, high-velocity sports, and collision sports are capable of producing significant forces to the thoracic and lumbar spine. Fractures, fracture dislocations, and dislocations can occur with and without neurologic injury (Fig. 53.4). Blunt trauma may produce rib fractures and transverse process fractures. Any of the ligamentous structures may be injured, requiring very specific treatments.

**A.** CT shows facet dislocation.

**B.** MRI shows edema in cord, kyphotic deformity, and rupture of the ligamentum flavum.



**C.** Plain film shows internal fixation postoperative.

**Figure 53.4.** NCAA football player sustained an accidental injury in the weight room resulting in a fracture dislocation of T11 on T12, with a conus medullaris spinal cord injury.

**Mechanism of injury:** Trauma
**Presentation:** Pain, limited motion, and possible neurologic findings with neurologic distribution of pain
**Physical examination:** Local tenderness, possible deformity, neurological findings with lower motor neuron findings with a lumbar injury (canda equina) or upper motor neuron findings with a cord injury
**Differential diagnosis:** Be aware of injuries at multiple levels.
**Diagnostics:** Imaging techniques, CT, MRI
**Treatment:** After on-field management and transfer to a care facility, further treatment plans can be made depending on examination and imaging. Certain traumatic injuries can be managed nonsurgically but surgical intervention may be required in others.
**Prognosis and return to sport:** Dependent on specific injury and neurological involvement

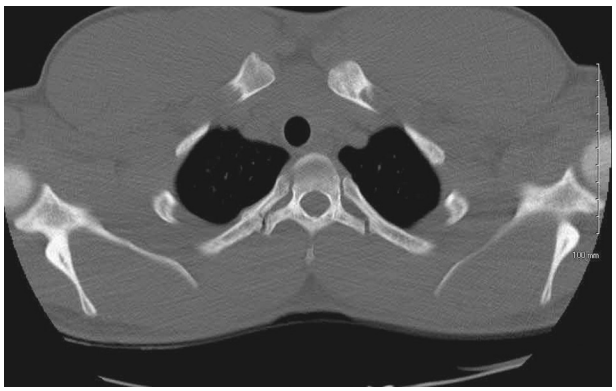


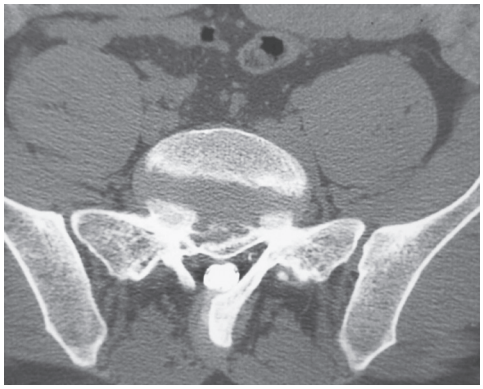
**Figure 53.5.** CT scan performed after a bone scan of a NCAA football player shows a posterior rib fracture that enters the costotransverse joint; this may manifest as a thoracic disc herniation.

## Thoracic Disc Herniation

**Description:** The extrusion of nucleus pulposus out of its contained position in the annulus fibrosus; nuclear material may cause a protrusion in the annulus (contained) or escape from the annulus, termed an *extrusion* or *free fragment*
**Mechanism of injury:** Annular tears occur with torsional loads under pressure; through these tears, the nucleus can extrude gradually or suddenly. Most herniations occur at the posterolateral portion of the annulus, where the annulus fibrosus merges with the posterior longitudinal ligament.
**Presentation:** Thoracic herniations may present as chest wall pain, thoracic back pain, exiting root pain along a rib, or as myelopathy if the cord is involved. Upper thoracic herniations may present as a cervical problem with pain radiating to the medial brachium. Lower thoracic herniations may present as lumbar disease.
**Physical examination:** If the patient is myelopathic, he or she will have upper motor findings of spasticity, weakness, clonus, positive Babinski. Exiting nerve root distribution of numbness or hyperesthesia may be present.
**Differential diagnosis:** Cervical and lumbar herniated discs, tumors (benign, primary, metastatic, neural, bone, or malignant), and fractures (both stress and traumatic) of the spine or rib (Fig. 53.5)
**Diagnostics:** Diagnosis is confirmed with MRI or myelogram with CT.
**Treatment:** Nonoperative treatment is with medications, and physical therapy with a stabilization program; surgical interventions indicated for significant progressive myelopathy and unrelenting radiculopathy
**Prognosis and return to sport:** Variable; return should be slow and cautious with intense rehabilitation

## Apophysitis

**Description:** Injury or inflammation of the ring apophysis of the spine
**Mechanism of injury:** Caused by repetitive traction on the anterior longitudinal ligament or repetitive compressions with end-plate microfractures
**Presentation:** Presents as back pain that increases with activity and is relieved by rest
**Physical examination:** Normal; unlike Scheuermann's disease there will not be a kyphosis
**Differential diagnosis:** Stress fractures, traumatic fractures, and spondyloarthropathy
**Diagnostics:** Plain radiographs will reveal irregularities of the ventral apophysis; bone scan may have increased activity.



**A.** Axial CT with bone remodeling

**B.** Sagittal cut showing protrusion of disc and calcified slipped apophysis at superior S1 and Schmorl's nodes at anterior L4 and L5

**Figure 53.6.** Slipped apophyseal ring: CT scan of a junior high basketball player with back and leg pain shows a slipped apophyseal ring at superior aspect of S1 and anterior Schmorl's nodes at L4 and L5.

**Treatment:** Rest
**Prognosis and return to sport:** Excellent; return is variable depending at length of symptoms

## Slipped Apophyseal Ring

**Description:** Posterior fracture of the ring apophysis with protrusion of the bone rim and disc into the canal (Fig. 53.6)
**Mechanism of injury:** Same as a herniated disc but in skeletally immature
**Presentation:** Can produce back pain only, particularly if the disc herniates anteriorly or into the vertebral body producing a Schmorl's node, which is very common (see Fig. 53.6); can present with leg pain if the protrusion enters the canal and will behave like a herniated disc
**Physical examination:** May have same findings as a herniated disc: list, weakness/numbness, tension findings, and reflex deficit
**Differential diagnosis:** Herniated disc, stress fracture, and SI joint pain
**Diagnostics:** MRI, myelogram with CT, EMG, and NCV
**Treatment:** Similar to herniated nucleus pulposus (see later section)
**Prognosis:** Same as herniated nucleus pulposus (see later section)

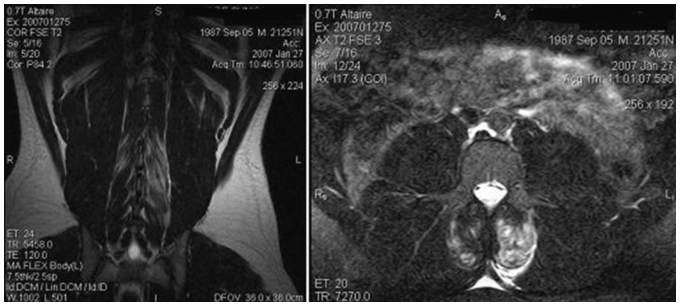


**Figure 53.7.** Lumbar myonecrosis with potential paravertebral compartment syndrome: MRI shows edema in paravertebral muscles.

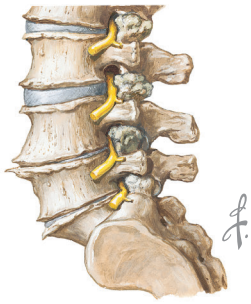
## Acute Lumbar Strain

**Description:** Muscle or muscle tendon unit strain
**Mechanism of injury:** Fatigue failure or over stretch injury to a muscle tendon unit about the lumbar spine; usually with bending, rotation, and/or inappropriate lifting
**Presentation:** Acute onset, back pain, no neurologic complaints
**Physical examination:** Localized tenderness, with or without spasm
**Differential diagnosis:** Stress reaction or fracture, and SI joint pain
**Diagnostics:** Radiographs may not be required by history or examination but will be normal or show a straight spine related to the spasm.
**Treatment:** Conservative care with rest, modalities, and occasional use of medications; to prevent recurrences a rehabilitation program with spine stabilization exercises will be helpful; this is based on core strength and teaches the principle of muscle fusion; use of core muscles to brace the spine. The goal is to find the neutral position or position of least pain and use it protectively. Overall, flexibility and strength is stressed through a well-defined program. Progressive aerobic training and sport-specific training is added as skills improve.
**Prognosis:** Excellent

## Lumbar Myonecrosis and Potential Compartment Syndrome

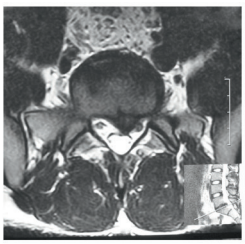
**Description:** Rare reports of compartment syndrome of the lumbar paraspinal muscles; more recently, events of lumbar myonecrosis with rhabdomyolysis have been reported; associated with sickle cell trait in athletes and can be significant
**Mechanism of injury:** Overuse, possibly associated with sickle cell trait
**Presentation:** After aggressive exercise, back pain, muscular, spasm, cramping sensation, dark urine occurs if rhabdomyolysis significant; back pain more severe and not motion related compared with lumbar strain
**Physical examination:** Posturing, spasm, and neurologically intact
**Differential diagnosis:** Lumbar strain, acute fracture, and acute stress fracture
**Diagnostics:** MRI will show edema in paraspinous muscles and possible enlargement (Fig. 53.7); laboratory studies (creatine kinase); sickle cell testing
**Treatment:** Rest when it occurs, observe to check for progression of rhabdomyolysis; treatment of potential causes of rhabdomyolysis, hospitalization if needed; long-term management with appropriate guidance
**Prognosis:** Excellent if detected early and protective measures taken

**A.** Radiograph of thoracic spine shows narrowing of intervertebral spaces and spur formation.

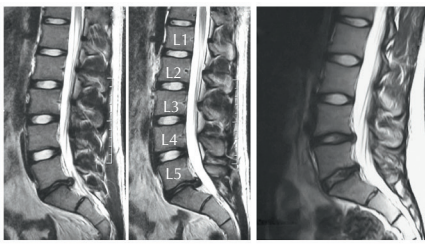


**B.** Degeneration of lumbar intervertebral discs and hypertrophic changes at vertebral margins with spur formation. Osteophytic encroachment on intervertebral foramina compresses spinal nerves.

**C.** Lumbar disc herniation. Schematic cross section showing compression of nerve root.



**D.** Axial and sagittal MRI of NCAA baseball player shows a herniated disc at L5–S1compressing the S1 nerve root.




**E.** MRI shows disc degeneration at two levels in a NCAA softball player.

**Figure 53.8.** Discogenic syndromes.

## Discogenic Syndromes

### Annular Tears

**Description:** A tear of the annulus fibrosus; nucleus material may or may not be extruded or displaced. As the tear occurs at the periphery, it is more likely to be painful and may be associated with referred radicular pain or a chemical neuritis.

**Mechanism of injury:** Rotational stress or rotation with compression

**Presentation:** Pain, local back and possible dermatomal distribution of referred pain, often with spasm and postural changes; limited motion

**Physical examination:** Neurologically normal examination, but may be posturing, with spasm

**Differential diagnosis:** Early herniated nucleus pulposus (HNP), stress fracture, acute lumbar stain, fracture, and SI joint pain

**Diagnostics:** Plain radiographs may be normal or show degenerative changes, straight spine if spasm significant; MRI may be normal but may show an area of increased signal intensity in the annulus, particularly at the outer annulus on T2-weighted images.

**Treatment:** Modalities: possible nonsteroidal anti-inflammatory drugs (NSAIDs) or Medrol dose pack and physical therapy with a spine stabilization program

**Prognosis:** Excellent, but may cause prolonged and often recurrent symptoms

### Herniated Nucleus Pulposus (HNP)

**Description:** The nucleus pulposus partially or completely extrudes through the annulus fibrosus, protruding into the neural elements or extruding into the canal and possibly being sequestered away from the annulus (Fig. 53.8). Herniation usually occurs to one side or the other of the posterior longitudinal ligament, which is a restraint, but may exit centrally through the posterior ligament. It can also exit far lateral to involve the root in the foramen. It is most common at the lower segments and during the 3rd or 4th decade of life.

**Mechanism of injury:** Repetitive flexion and rotation increases the load on the disc; nachemson has measured the intradiscal pressures in various positions, and this pressure is increased with flexion.

**Presentation:** May present as back pain as the annulus tears in the process of the disk extruding; as it extrudes and as the nerve becomes involved the back pain will become associated with buttock pain or extremity pain. The back pain may lessen as the radicular pain, numbness, and dysesthesias progress along a dermatomal distribution. Weakness is possible. Pain is usually worse with prolonged sitting, flexion, or Valsalva maneuvers. If severe compression occurs, part or all of the cauda equina may be involved, leading to weakness, bowel, bladder, and sexual dysfunction (cauda equina syndrome).

**Physical examination:** Patient may have a list, may tripod, may have limited ROM, and may have spasms. Tension tests are positive, and a neurologic examination may reveal numbness and weakness.

**Differential diagnosis:** Stress fracture to lumbar spine, sacrum, or pelvis, annular tear with referred pain, plexopathy, peripheral neuropathy or nerve injury, SI joint pain, and fracture (see Figs. 53.6 and 53.7)

**Diagnostics:** Plain radiographs may be normal or show a narrow disc space. MRI will show the lesion (see Fig. 53.8). Myelogram and myelo-CT will also reveal the lesion but are more invasive. An MRI with gadolinium may be required to distinguish post-surgical scarring from a recurrent herniated disc. EMG and NCV studies may be helpful to rule out a peripheral neuropathy that can masquerade as a radiculopathy. This may be needed because 32% of imaging techniques may be positive in asymptomatic subjects. A positive study is not diagnostic unless it confirms the clinical picture.

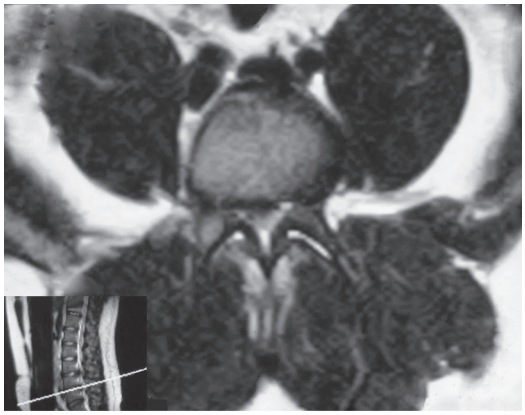
**Treatment:** Conservative with medications, possible Medrol dose pack, followed by NSAIDs; epidural steroid treatment may be used. Surgery may be required for intractable pain, progressive neurologic deficit, or lack of improvement after 8–12 weeks. A cauda equina syndrome requires urgent surgical care.

**Prognosis and return to sport:** Several patients completely recover with nonsurgical care; this, followed by aggressive rehabilitation and a prolonged care program, can lead to return to sport. In certain studies, 70%–90% of athletes treated surgically were able to return to an elite level. Strength programs and lifting as a part of training should be appropriately adjusted.

## Spinal Stenosis

**Description:** Narrowing of the space available for the nerves; may be congenital with short pedicles or a narrow interpedicular distance or acquired related to trauma or degeneration. It may be central (spinal canal) or foraminal.

**Mechanism of injury:** Congenital, traumatic, or degenerative; may be caused by a synovial cyst or herniated disc



**A.** Axial image shows congenitally short pedicles and triangular narrowed canal.

**B.** Sagittal image shows degeneration with acquired changes exacerbating a congenital stenosis.

**Figure 53.9.** Spinal stenosis: MRI of NCAA football player shows spinal stenosis.

**Presentation:** Neurologic symptoms, radicular pain, neurogenic claudication, and postural radicular pain
**Physical examination:** May be normal; if severe, may have neurologic findings
**Differential diagnosis:** Herniated disc, stress fracture, fracture, SI joint pain, and peripheral neuropathy
**Diagnostics:** Imaging by MRI or myelo-CT; EMGs and NCV may be useful (Fig. 53.9).
**Treatment:** Specific to cause; if symptoms are related to a synovial cyst, then aspiration or injection may be useful. If congenital, then surgical decompression may be required; if acquired degenerative, then conservative measures such as medication and epidural steroids may help prolong an athletic career
**Prognosis and return to sport:** Variable

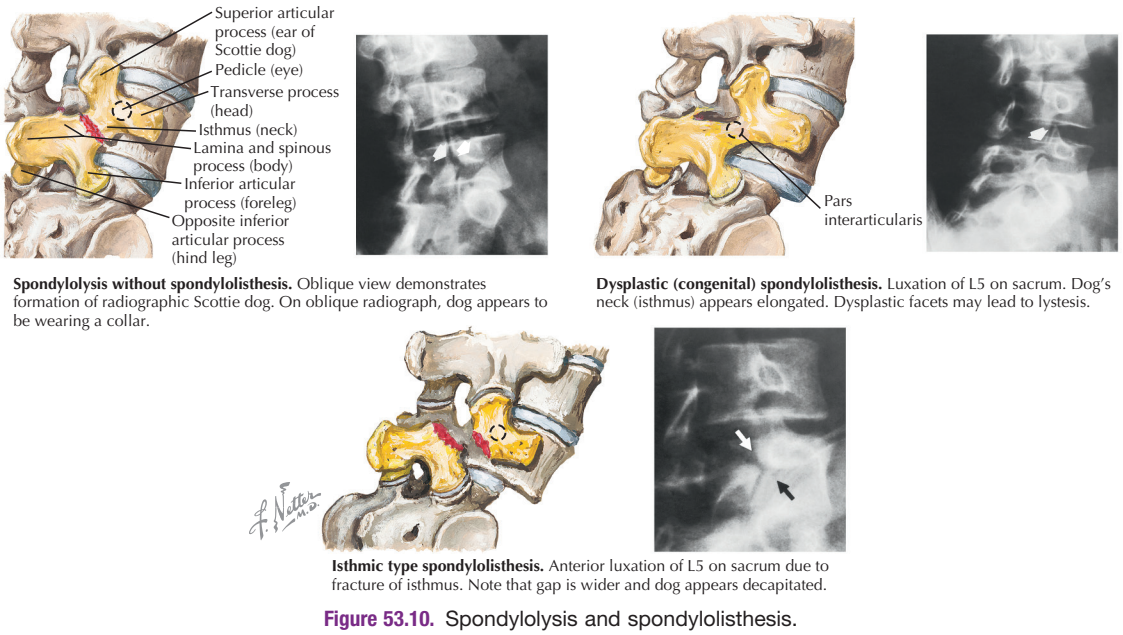
## Stress Reaction, Stress Fracture, Spondylolysis

**Description:** A spectrum of overuse injuries to the pars interarticularis or pedicle area of the bone; very common in athletes
**Mechanism of injury:** Repetitive shear forces, worse with repeated extension and hyperextension maneuvers that load this portion of the bone
**Presentation:** Back pain, occasionally with some radicular pain; worse with extension; present as a bone stress reaction without a fracture on one or both sides, or as a nonunion of a stress fracture, known as *spondylolysis* (spondylo = spine, lysis = defect), on one or both sides (Fig. 53.10). Pain can be present on one or both sides, worse with motion or running; often has onset in strength and conditioning workouts
**Physical examination:** Extension and one-legged extension tests are positive; neurologic examination is negative; tension tests are negative. Pain may occur with rotation or side bending.
**Differential diagnosis:** Annular tear, HNP, SI joint pain, and stress fractures; in the sacrum or pelvis, facet syndrome
**Diagnostics:** Plain radiographs include AP, lateral, and oblique views. They may be normal early in a reaction and certain defects are not clearly seen. Oblique radiographs can show a defect as "the neck of the Scotty dog" (see Fig. 53.10). Sometimes, the defect can be seen on a lateral radiograph. If plain radiographs are negative, nuclear imaging will be helpful. A whole-body conventional biplanar bone scan can not only visualize the pars area but also rule out a sacral or pelvic stress fracture that mimics a lumbar injury. A bone scan combined with a single photon emission computerized tomography (SPECT) will improve sensitivity and localization of small abnormalities by screening out other tissues. A positive scan indicates an acute lesion and suggests bone healing potential.
**Treatment:** Guided by symptoms and images; restrictions are controversial, particularly bracing
- Normal plain radiograph, positive scan indicates an acute lesion; a positive scan indicates metabolic activity and some potential for healing. A modified Boston orthosis may be indicated. Limit activities for 4–8 weeks; rehabilitation exercises to begin as symptoms abate. Strength and neutral position are stressed. Occasionally with a positive scan, a thin-slice CT may help evaluate the lesion and help with treatment. If CT scan normal, no brace; if CT scan shows acute fracture line, brace; if CT scan shows fracture with sclerotic margins (chronic lesion), no brace required
- Positive plain radiograph and positive scan indicate a semiacute lesion. A 0-degree antilordotic modified Boston orthosis may be indicated; Micheli reported success with use of this orthosis for 23 hours/day for 12 weeks, but the use is variable. The goal is to be pain free before beginning rehabilitation exercises. The lesion may not heal with bone, so progression is based on symptoms.
- Positive plain radiographs with a negative bone scan indicate a chronic lesion or nonunion. A negative scan suggests minimal metabolic activity and poor healing potential. Start rehabilitation when symptoms abate. Occasionally, surgery is required if pain is intractable.

**Prognosis and return to sport:** Excellent

## Spondylolisthesis

**Description:** One vertebral body is slipping relative to another (spondylo = spine, listhesis = slippage) (see Fig. 53.10); usually, the upper body is slipping forward on the body below, resulting in a kyphotic deformity at the slip and a reactive lordosis above.
- There are five types of spondylolisthesis:
  - Dysplastic (abnormal anatomy)
  - Isthmic (caused by a defect in the pars); this is most common in young athletes
  - Degenerative (caused by lax facet joints and a degenerative disc that allows the slippage)—more common with age
  - Traumatic (high-velocity injuries)
  - Pathologic (tumors or osteopenic bone)
- Isthmic (the most common type in young athletes) is most common from L5 to S1 and can be graded on a scale of I–V




**Spondylolysis without spondylolisthesis.** Oblique view demonstrates formation of radiographic Scottie dog. On oblique radiograph, dog appears to be wearing a collar.

**Dysplastic (congenital) spondylolisthesis.** Luxation of L5 on sacrum. Dog's neck (isthmus) appears elongated. Dysplastic facets may lead to lystesis.

**Isthmic type spondylolisthesis.** Anterior luxation of L5 on sacrum due to fracture of isthmus. Note that gap is wider and dog appears decapitated.

**Figure 53.10.** Spondylolysis and spondylolisthesis.

based on the amount of forward displacement of the higher vertebra on the vertebra below.
- 0%–25% is grade I
- 25%–50% is grade II
- 50%–75% is grade III
- 75%–100% is grade IV
- Complete displacement or spondylolisthesis is grade V

**Mechanism of injury:** Isthmic spondylolisthesis occurs as a result of repetitive overuse at a young age, probably related to extension activities. Progression of slips is most likely to occur between ages 9 and 12 years in girls and 10 and 14 years in boys.

**Presentation:** Patients with grade I and II slips may be asymptomatic and may be participating in sport not knowing about the defect. They may have back pain and occasional radicular pain; pain is worse with extension activities. Patients with higher-grade lesions may have been limited by their pain and face more difficulty with sports because of back pain, stiffness, and hamstring tightness. Localized back pain is common, and radicular pain may develop as a result of nerve root compression by hypertrophic callus or the deformity or instability.

**Physical examination:** With high-grade slips, there will be vertical sacrum, flat buttocks, and compensating tight hamstrings. There may be a palpable step-off at the deformity and a short waist. Neurologic examination is usually normal.

**Differential diagnosis:** Just because a patient has a spondylolisthesis does not mean that it is the origin of the pain. Other possibilities include SI joint pain, degeneration of other levels, and stress fracture at other levels.

**Diagnostics:** Standing plain radiographs usually reveal the abnormality and a lateral projection allows grading. Repeat radiographs should be obtained yearly if the diagnosis is made before the age of 10 years because progression can occur. CT and MRI are useful if other sources are suspected.

**Treatment:** Conservative in most cases and involves trunk stabilization exercises; occasional limits may be required for symptomatic events. Surgical fusion may be required with high-grade slips or with slips associated with neurologic symptoms related to nerve root compression or stenosis.

**Prognosis and return to sport:** For low-grade slips, excellent to good; but high-grade slips can be more problematic and limiting

## Inflammatory Causes of Pain

### Facet Syndrome

**Description:** Pain syndrome emanating from an irritated facet joint

**Mechanism of injury:** This synovial fluid lined joint can become inflamed with injury and have associated hyaline cartilage damage. Synovitis may occur. Facet trophism (asymmetry) may lead to injury.

**Presentation:** Painful with extension and bending to the involved side

**Physical examination:** Pain reproduced with above maneuvers; neurologically intact

**Differential diagnosis:** Stress fracture, fracture, discogenic disease, and SI joint pain

**Diagnostics:** Plain radiographs may show joint changes. Bone scan may reveal increased activity in the joint, and a CT scan may show arthritic changes.

**Treatment:** NSAIDs may be helpful. A facet injection may be diagnostic and therapeutic.

**Prognosis and return to sport:** Participation limited by pain

### Sacroiliac Joint Dysfunction

**Description:** Pain syndrome emanating from an irritated SI joint; commonly confused with low back pain. These joints are very strong and allow limited motion while being constrained with strong anterior and posterior ligaments; possess a synovial membrane

**Mechanism of injury:** Injury, contracture, or inflammation of the SI joint

**Presentation:** Patients may present with a traumatic history or they may be involved in jumping sports that require repetitive single-leg landing. Pain is usually over the SI joint but may also be referred into the leg, suggesting radiculopathy.

**Physical examination:** Local tenderness, a positive supine-to-long sitting test, a painful extension test, a painful FABER test, and pain when stresses are applied to the SI joint

**Differential diagnosis:** Discogenic pain, stress fracture, facet joint pain, and muscle injury

**Diagnostics:** Radiographs normal unless late in the course of severe spondyloarthropathy

**Treatment:** Mobilization exercises, NSAIDs, modalities, and occasionally, corticosteroid injections into the SI joint
**Prognosis and return to sport:** Limited only by pain

## RETURN-TO-PLAY DECISIONS

- Each condition has its own criteria and may allow return to sport at certain but not all levels.
- Consultation with a spine specialist is recommended to help make the decisions and to discuss the risks and benefits of participation with the patient and family.
- The following findings may prohibit participation in contact sports:
  - Intersegmental instability on flexion–extension radiographs (e.g., instability that could potentially lead to neurological impairment)
  - Spinal cord impingement with myelopathy
  - Significant neurologic impairment or risk of impairment with a herniated disc or obstructing lesion
  - Limiting pain
  - A previous spinal fusion
- After treatment, certain athletes can be placed into the following risk categories, and allowed to return to sport with the understanding that there is a level of risk involved.
  - Minimal risk: The increase in risk is small compared with play before the injury.
  - Moderate risk: There is a reasonable chance that symptoms will recur, and the patient is at a risk of permanent injury.
  - Extreme risk: The risk that symptoms will recur and cause a permanent damage is high.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- **eAppendix 53-1 Less Common Thoracic and Lumbosacral Spine Issues:**
  - Degenerative Disc Disease
  - Postural Roundback (Flexible)
  - Scheuermann's Disease
  - Atypical Scheuermann's Disease
  - Scoliosis
  - Ankylosing Spondylitis
  - Infections

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

# eBOOK SUPPLEMENTS

## eAppendix 53-1 Less Common Thoracic and Lumbosacral Spine Issues

### Degenerative Disc Disease

**Description:** Annular tears and herniated discs are part of the spectrum of degenerative disc disease. Degeneration of the discs leads to collapse and then concomitant degeneration of the facet joints and hypertrophy of the facet capsule, which can lead to stenosis.
**Mechanism of injury:** Chronic degeneration of the disc
**Presentation:** May present with an annular tear or herniated disc, but also as a facet syndrome with symptoms from stenosis, either foraminal or central; patients may have back pain, neurologic radicular pain, referred facetal pain, or neurogenic claudication pain. Symptoms may be positional and worse with extension and improved with flexion. Certain athletes may have congenital stenosis with the addition of acquired degenerative changes then become symptomatic (see Fig. 53.8).
**Physical examination:** May have neurologic findings, limited ROM; may have a normal examination; may be seeking postural changes for relief of symptoms
**Differential diagnosis:** Stress fracture, SI joint pain, fracture, and congenital stenosis
**Diagnostics:** Imaging techniques, MRI, myelogram, and CT
**Treatment:** Specific to symptoms; medicines and stabilization protocol for back symptoms. Facet injections for facet syndrome; epidural steroids for stenotic neurogenic symptoms. Occasionally, surgical decompression may be needed for intractable limiting pain or neurologic deficit. However, surgical decompression in an athlete can lead to a weakening of the lamina and predispose the athlete to pars fractures and spondylolisthesis and may limit his or her return to sport.

### Deformities

#### POSTURAL ROUNDBACK (FLEXIBLE)

**Description:** A long, gentle kyphotic deformity is usually seen in older adolescents. It is flexible and can be voluntarily corrected, unlike Scheuermann's.
**Mechanism of injury:** Postural
**Presentation:** No pain; presents as a deformity
**Physical examination:** Gentle round kyphosis
**Differential diagnosis:** Scheuermann's kyphosis or congenital kyphosis
**Diagnostics:** Normal spine has a forward kyphosis of 25–45 degrees. In postural roundback, the kyphosis measured on a standing lateral from the inferior margin of T12 to the inferior margin of an upper level is >40 degrees. A supine lateral radiograph with hyperextension shows correction.
**Treatment:** Postural
**Prognosis and return to sport:** Sports participation is not limited

#### SCHEUERMANN'S DISEASE

**Description:** A fixed rigid thoracic kyphotic deformity with or without pain, also known as *juvenile kyphosis*, and may present between 8 and 12 years of age (eFig. 53.1)
**Mechanism of injury:** It is thought to be caused by necrosis of the ring apophysis, perhaps related to repetitive trauma.
**Presentation:** Usually asymptomatic but certain patients report pain or fatigue. Incidence is high in adolescent rowers and weightlifters. Progression later in life is unusual.
**Physical examination:** Rigid kyphosis accentuated with forward flexion; it tends to produce a more acute angle deformity at midthorax compared with a postural deformity.
**Differential diagnosis:** Postural roundback or congenital kyphotic deformity
**Diagnostics:** Radiographs will reveal the kyphosis and a lateral radiograph must show three consecutive thoracic vertebral bodies each wedged >5 degrees, with endplate irregularity or with Schmorl's node.
**Treatments:** May include observation, exercises, bracing, or even surgery
**Prognosis and return to sport:** Sports participation is not limited.

#### ATYPICAL SCHEUERMANN'S DISEASE

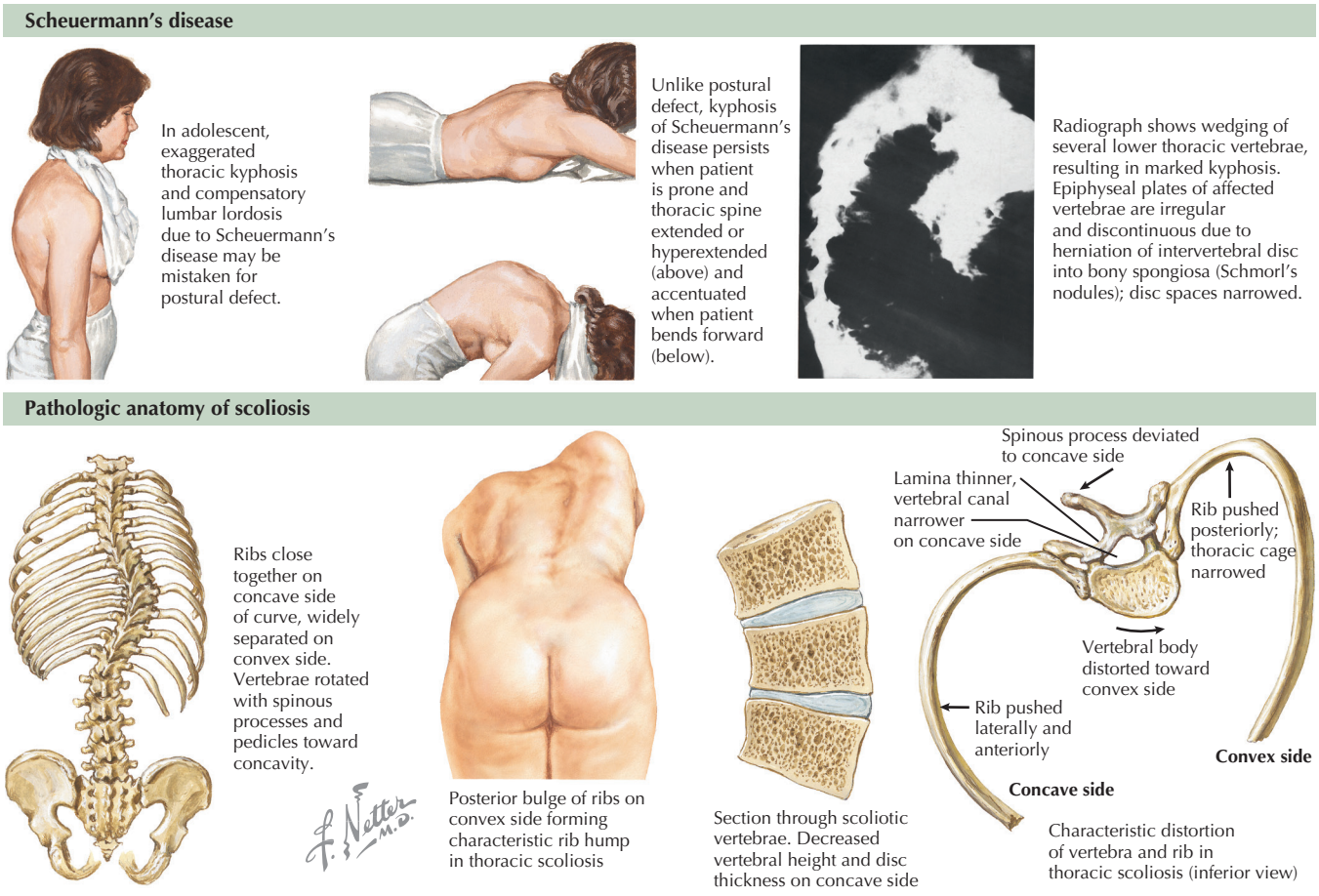
**Description:** Also known as *thoracolumbar* or lumbar *Scheuermann's kyphosis*
**Mechanism of injury:** Same as Scheuermann's kyphosis
**Presentation:** This kyphosis tends to be more painful; associated with sports that have vigorous spine loads (gymnastics, particularly dismounts, and weightlifting)
**Physical examination:** Loss of lumbar lordosis
**Differential diagnosis:** Old fractures
**Diagnostics:** Radiograph shows loss of lordosis; will have vertebral wedging and Schmorl's nodes but may not involve consecutive vertebrae
**Treatment:** Conservative with rest and therapeutic exercises; occasional bracing may be required
**Prognosis and return to sport:** No limits on sports but may occasionally be limited by pain

#### SCOLIOSIS

**Description:** A lateral spinal curvature of >10 degrees; more common in girls than boys and is present to a minor degree in 2%–3% of the population
**Mechanism of injury:** Of scoliosis cases, 80% are of unknown etiology and are called idiopathic. There is a strong familial factor. Less common congenital scoliosis results from a failure of formation or segmentation. Other types may be related to neurologic problems.
**Presentation:** Deformity, otherwise usually asymptomatic; a careful history and examination is required to rule out other causes for the scoliosis, most of which are neurologic.
**Physical examination:** Inspection from behind will reveal the curve and allow to check for balance. Forward bending will allow measurement of rib elevation. Neurologic examination will be normal in idiopathic scoliosis. Skin pigmentation changes, skin defects at the spine, and neurologic defects may suggest other types of scoliosis.
**Differential diagnosis:** Congenital scoliosis or neuropathic scoliosis
**Diagnostics:** AP and lateral views on a 14-by-36 cassette to include the entire thoracolumbar spine and to allow assessment of skeletal maturity (Risser sign)
**Treatment:** Varies from observation to bracing to surgery, depending on the age and type of curve. If the curvature is <20 degrees, no active treatment is required but reassessment should be performed every 4–6 months until skeletally mature. If the curve is ≥25 degrees or if progressing, then bracing may be indicated. Severe curvature may require surgery.
**Prognosis and return to sport:** Most patients are allowed to participate in sports. If surgery is required, then participation in sport may be limited and decisions regarding sport should be made with the surgeon. Athletes need to understand that the fusion type of surgery required will limit flexibility and can alter performance. Fusion surgeries place additional stress on the adjacent spinal segments and can produce pathology at those levels.

### Ankylosing Spondylitis

**Description:** An inflammatory spondyloarthropathy
**Mechanism of injury:** Unknown cause but may be associated with the rheumatoid family of autoimmune diseases
**Presentation:** Can present with spine or SI symptoms, usually at 15–35 years; insidious onset morning stiffness, pain, and




**eFigure 53.1.** Spinal deformities.

insertional tendonitis (enthesopathy); 20% have peripheral joint disease. Eyes, lungs, and heart may be involved.

**Physical examination:** May reveal limited chest expansion (2 inches); spinal flexibility may be limited, as measured by Schober's test

**Differential diagnosis:** Other forms of inflammatory arthropathy, degenerative disc disease, and SI joint disease

**Diagnostics:** HLA-B27 testing may be helpful. Radiographs of SI joints may show erosions, blurring of margins, sclerosis, and narrowing. Spine radiographs may show straightening of the lumbar spine, squaring of vertebral bodies, and with progression of the disease, syndesmophytes will bridge adjacent vertebrae, leading to the appearance of a "bamboo" spine.

**Treatment:** Medications helpful

**Prognosis and return to sport:** Allowed to participate but may be limited by pain

### Infections

**Description:** Adult disc space infections, discitis, and vertebral body osteomyelitis may cause back pain, but are very rare in an athletic population

**Mechanism of injury:** Infection, usually by a hematogenous spread from distant focus

**Presentation:** Insidious with progressively worsening back pain, may be febrile

**Physical examination:** Rigid spine, pain with motion

**Differential diagnosis:** Neoplasia and fracture

**Diagnostics:** Laboratory studies may reveal elevated white blood cell count and sedimentation rate. Blood cultures may be positive. Radiographs may be negative early but may show disc-space narrowing and endplate irregularities later. Bone scan will be positive and an MRI will be valuable to differentiate between a tumor and an infection.

**Treatment:** Specific to infection

**Prognosis and return to sport:** During acute phase, will not be able to participate; late participation will depend on treatment and outcome

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# PELVIS, HIP, AND THIGH INJURIES

# 54

Jorge Chahla • Matthew J. Kraeutler • Cecilia Pascual-Garrido

## GENERAL PRINCIPLES

### Overview

- Understanding of hip pathology has substantially improved recently owing to more specific clinical tests, better imaging diagnosis, and discovery of new entities.
- Hip pathologies include femoroacetabular impingement (FAI), borderline dysplasia, femoral version, hip instability, and femoral head deformities such as slipped capital femoral epiphysis (SCFE) and Perthes disease.
- Understanding hip anatomy, physiology, biomechanics, different pathologies, and treatment options is key to offering high-quality care to patients.

### Bony Anatomy

#### Acetabulum

- The acetabular fossa constitutes the inferior portion of the acetabulum and is surrounded by the lunate surface in its superior and lateral aspects (Fig. 54.1).
- When performing hip arthroscopy, it is critical to understand the location of the pathology. To locate chondrolabral hip lesions more easily, the socket is considered a clock, wherein 12 o'clock represents the most superior aspect of the acetabulum, continuing anteriorly with successive hours. Unlike the knee, the 3 o'clock position is always anterior for both right and left hips.
- Several bony landmarks have been described to help the surgeon with an accurate location during arthroscopy:
  - The superior extent of the anterior labral sulcus (psoas-u) indicates the 3 o'clock position on the acetabular rim; anteriorly, it corresponds to the location of the iliopsoas tendon.
  - The stellate crease is located superior to the apex of the acetabular fossa and corresponds to the 12:30 position (see Fig. 54.1).
- The abduction angle of the acetabulum relative to the horizontal plane averages 45 degrees with 20 degrees of anteversion.

#### Femoral Head

- The femoral head forms roughly two-thirds of a sphere whose surface is completely articular except for the fovea capitis femoris where the ligamentum teres (ligament of the head of the femur) is attached (see Fig. 54.1).
- On an average, the neck shaft angle averages 130 degrees and the femoral neck is anteverted 14 degrees relative to the bicondylar axis at the knee.

### Soft Tissue Anatomy

#### Labrum

- The labrum is a fibrocartilaginous structure attached to the acetabular rim.
- It is responsible for guaranteeing the suction seal, a key function in fluid dynamics, ensuring wider coverage of the femoral head and providing negative intra-articular pressure that provides stability to the hip joint.
- Labral pathology is normally observed in the anterosuperior area of the socket (12 to 2 o'clock position); this area correlates with the location where impingement normally occurs (Fig. 54.2).

#### Capsule

- The hip capsule is formed by the iliofemoral ligament anteriorly, the ischiofemoral ligament posteriorly, and the pubofemoral ligament inferiorly (see Fig. 54.1).
- Near the acetabular origin, the superior and superolateral aspect of the capsule is the thickest portion (3.7–4.0 mm) (Fig. 54.3).
- The capsule is inserted at a mean of 26.2 mm distal to the chondral head–neck junction of the proximal femur. The capsule has a spiral configuration that tightens in terminal extension and external rotation.

### Muscles

#### Hip Abductors

- Gluteal group: Gluteal muscles include the gluteus maximus, gluteus medius, gluteus minimus, and tensor fasciae latae.
- The gluteus medius muscle stabilizes the hip and controls hip motion, particularly during weight bearing. Weakness or insufficiency of this muscle leads to the Trendelenburg gait.

#### Hip Adductors

- Adductor group: Adductor brevis, adductor longus, adductor magnus, pectineus, and gracilis muscles; the adductors originate on the pubis and insert on the medial, posterior surface of the femur, with the exception of the gracilis, which inserts distally on the pes anserine on the tibia. There is a pubic aponeurosis that is a confluence of the adductor and gracilis origins; it is also referred to as *rectus abdominis/adductor aponeurosis*. There is a clinical association between FAI and sports hernia and adductor tendinopathy, also known as *athletic pubalgia*.

#### Hip Flexors

- Iliopsoas group: Composed of the iliacus and psoas major muscles; the iliacus originates on the iliac fossa of the ilium and joins the psoas major muscle that runs from the lumbar bodies (2, 3, 4) and inserts distally into the lesser trochanter.
- The rectus femoris is a weaker flexor with the knee in extension. However, owing to its proximity to the capsule and its complex anatomy, it serves as an important differential diagnosis for hip pain.
- The direct head of the rectus femoris attachment has a shape of a "tear drop" occupying the entire footprint of the superior facet of the anterior inferior iliac spine (AIIS). The indirect head has a broad insertion over the rim (Fig. 54.4).

#### Hip Short External Rotators

- This group consists of the obturator externus and internus, the piriformis, the superior and inferior gemelli, and the quadratus femoris (see Fig. 54.4). The piriformis muscle originates in the anterior surface of the sacrum and inserts into the superior boundary of the greater trochanter; the gemellus superior goes from the ischial spine and joins the piriformis tendon in its insertion at the greater trochanter. The obturators (internus and externus) insert on the medial and lateral surface of the obturator membrane and then travel to the medial surface of the greater trochanter and the trochanteric fossa, respectively. The gemellus inferior muscle inserts on the superior aspect of the ischial tuberosity and inserts distally into the piriformis tendon (indirectly to the greater trochanter). Last, the quadratus

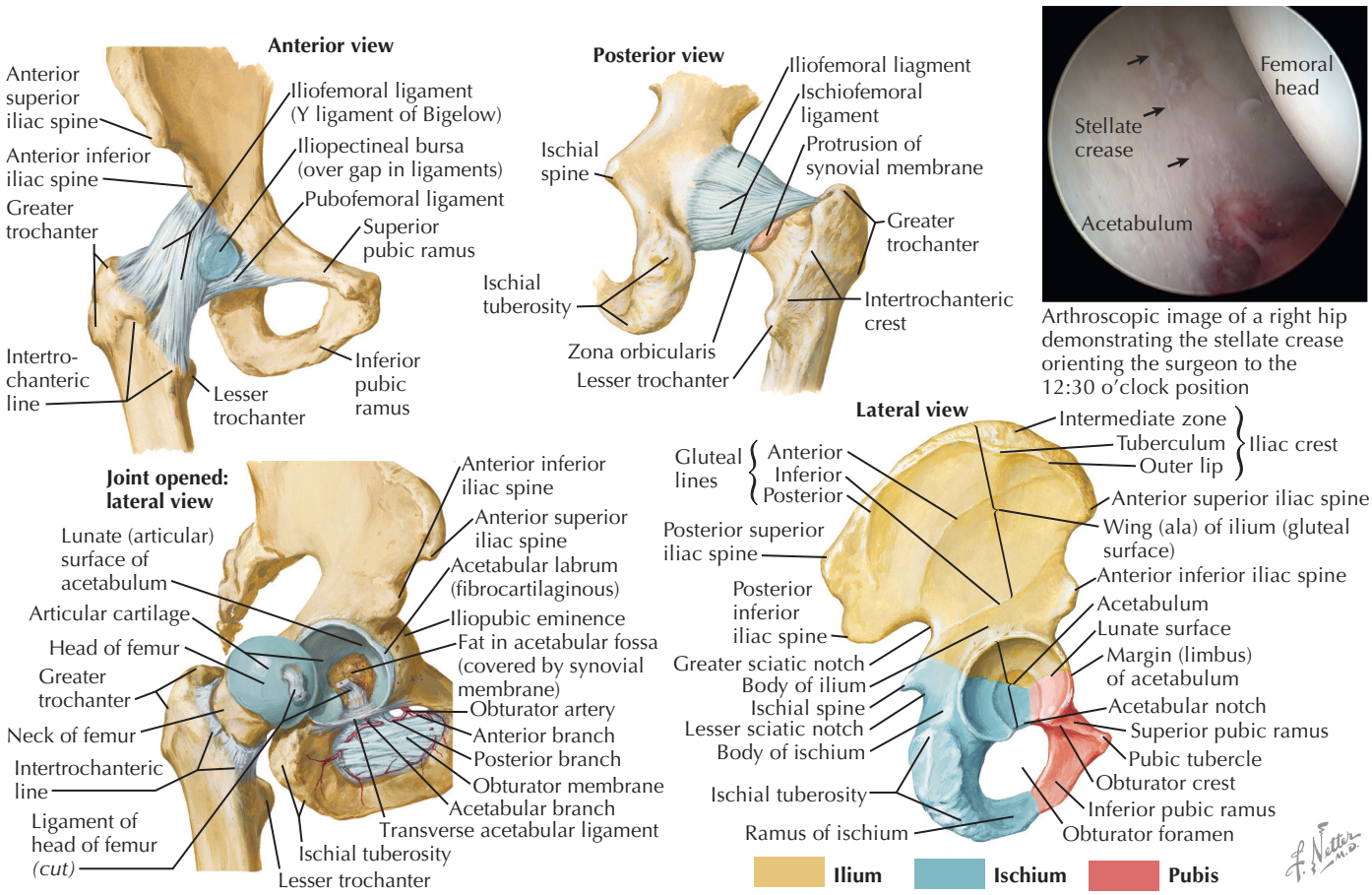



**Figure 54.1.** Bony anatomy of the hip.

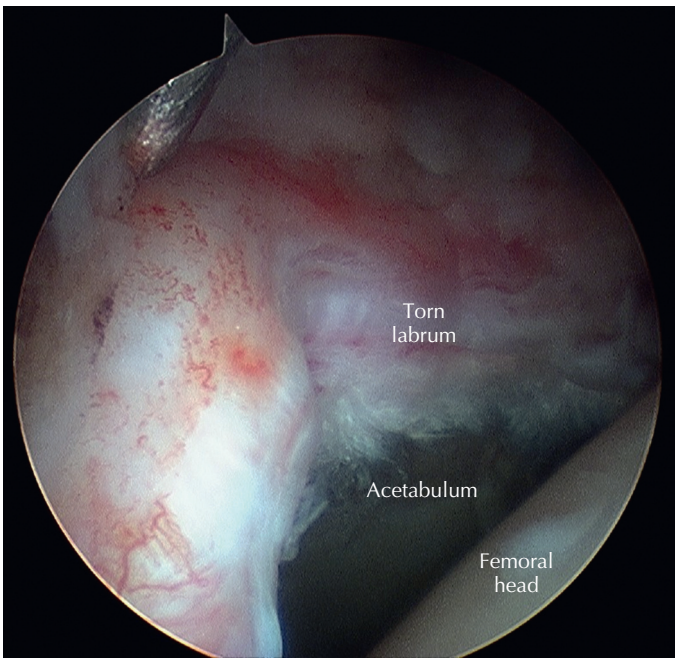



**Figure 54.2.** Arthroscopic image of a left hip demonstrating a tear in the superolateral aspect of the labrum. Note also the inflammation of the labrum confirming a pathologic process.

femoris muscle goes from the lateral edge of the ischial tuberosity to the intertrochanteric crest.

## Neurovascular Structures

- The extracapsular arterial ring at the base of the femoral neck is formed posteriorly by a large branch of the medial femoral circumflex artery (MFCA) and anteriorly by smaller branches of the lateral femoral circumflex artery (LFCA). The superior and inferior gluteal arteries have minor contributions to the irrigation. Retinacular arteries and the internal ring arise from the ascending cervical branches. Finally, the artery of the ligamentum teres is derived from the obturator artery or the MFCA (Fig. 54.5).
- The blood supply to the femoral head is mainly from the deep branch (posterior) of the MFCA. During anterior controlled hip dislocation, this vessel is protected by the obturator externus muscle. The ligamentum teres branch, which is important during developmental phases of the femoral head, does not play an important role in the adult hip.
- The lower extremity receives its innervation from the lumbosacral plexus, which forms the sciatic, femoral, and obturator nerves as well as various smaller branches.
- The hip receives innervation from L2 to S1 nerve roots but principally from L3; this explains the presence of medial thigh pain often accompanying hip pathology because symptoms may be referred to the L3 dermatome.
- The lateral femoral cutaneous nerve, providing sensation to the lateral thigh, exits the pelvis under the inguinal ligament, close to the anterior superior iliac spine (ASIS).

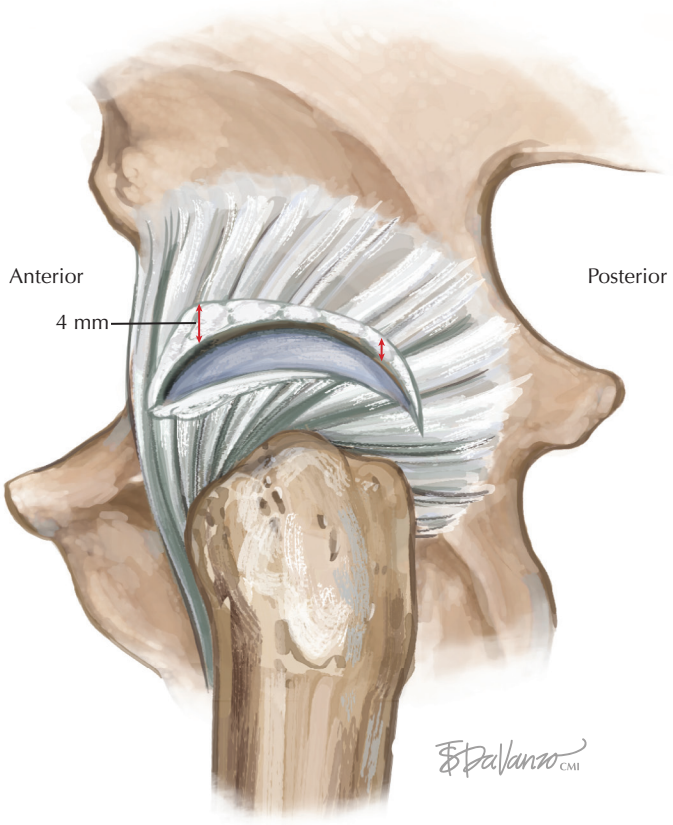



**Figure 54.3. Left Hip With an Intraportal Capsulotomy.** Note the thickness of the capsule at several locations (thickest at its superolateral portion).

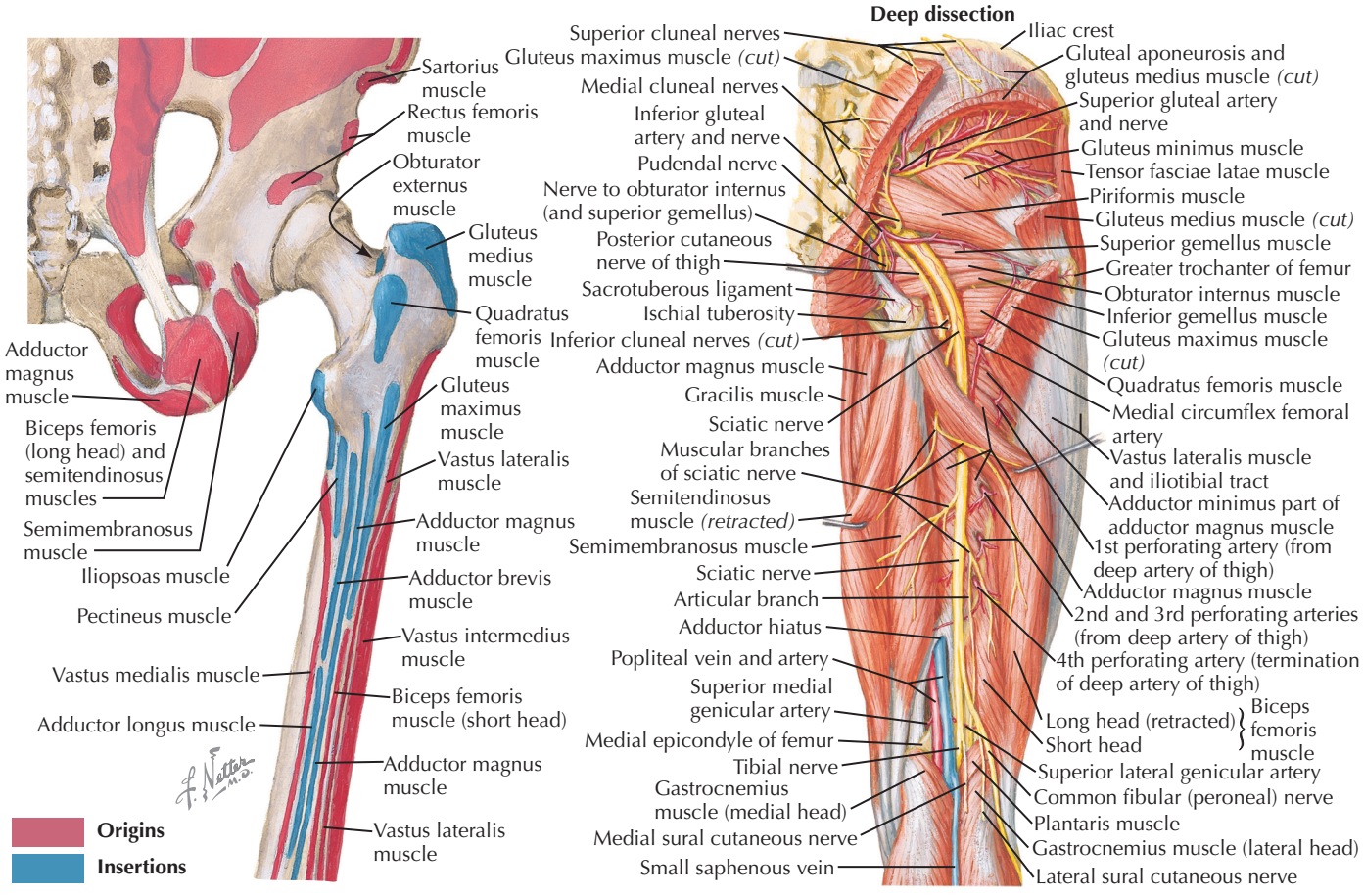



**Figure 54.4.** Muscles and insertions of pelvis, hip, and thigh.

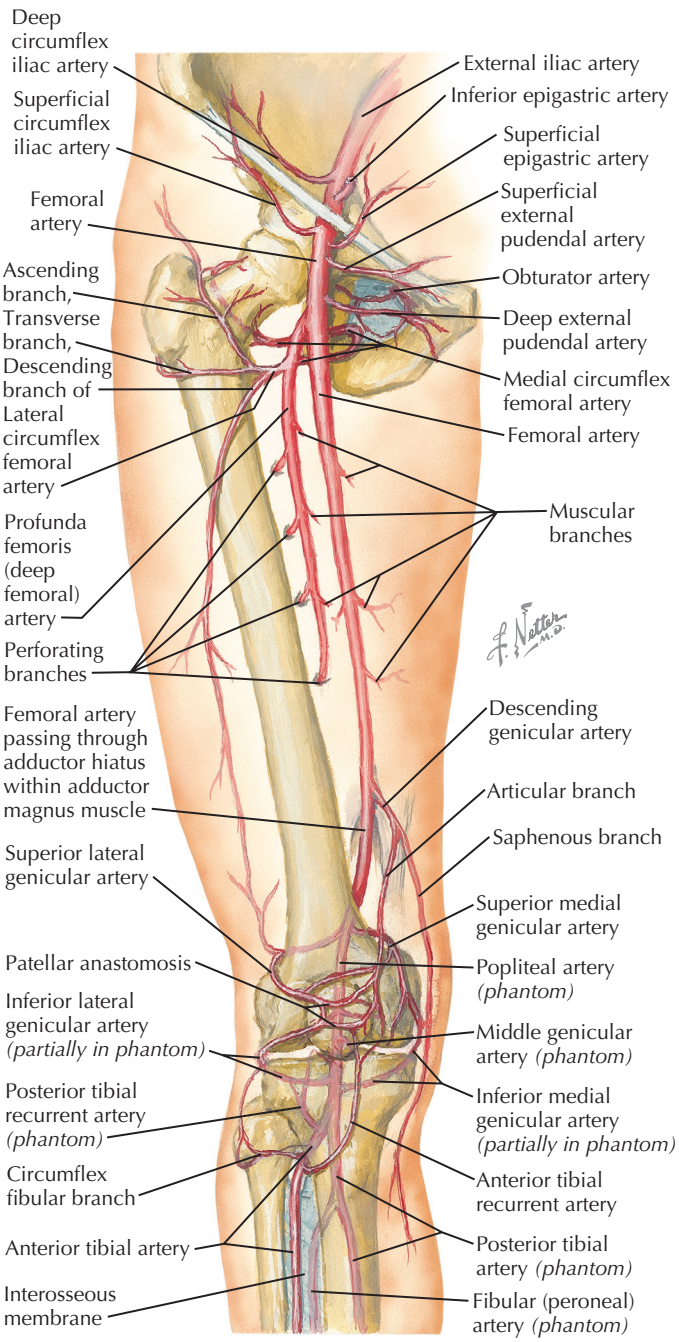



**Figure 54.5.** Arteries of the hip, pelvis, and thigh.

## History and Physical Examination

### History

- Acute onset of hip pain is more likely to be due to a specific pathology, which may be diagnosed through physical examination and imaging.
  - These injuries are typically easier to treat and carry a more favorable prognosis.
- Gradual onset of hip pain is likely to be due to (i) chronic disease or (ii) pain syndromes, which may be difficult to diagnose.
  - These injuries typically carry a worse prognosis and require more complex methods of treatment.

### Inspection

- Examine patient's gait and stance.
- Examine patient's posture in supine and seated positions, looking for internal or external rotation of the injured limb at rest, or a flexion contracture of the hip joint.
- Check for asymmetry in leg length, muscle atrophy, or pelvic obliquity.

### Range of Motion (ROM)

- When performing these measurements, it is important to assess if the range of motion (ROM) is limited or excessive; this will help differentiate between two different pathologies (impingement vs. dysplasia).
- Clinically, leg length can be measured from the ASIS to the medial malleolus.
- ROM should be measured bilaterally to compare the injured and noninjured sides. Hip flexion, extension, abduction, adduction, and internal and external rotation should be measured. Internal and external rotation should be assessed both with the patient supine, at 90 degrees of hip flexion, and prone with the hip at neutral. This last position will help define the patient's femoral version. Patients with excessive anteversion of the femur will have excessive internal rotation (IR) when they are prone and with the hip neutral.

### Palpation

- The patient should be asked to point to the single most painful location; this will provide helpful clues as to the potential diagnoses.
  - Patients with hip pain will normally do the "C sign," grabbing their hip in a C-shape with their hand.
  - Patients with posterior pelvic pain may also have associated groin pain, lateral thigh pain, or anterior thigh pain.
  - Posterior pelvic pain may be associated with labral tear, developmental dysplasia of the hip (DDH), and/or FAI.
- Palpation should begin away from the source of the patient's pain and progressively move closer to the painful location.
- The following anatomic sites should be palpated to assess for significant pain: lumbar vertebrae, sacroiliac (SI) joint, ischium, iliac crest, greater trochanter, trochanteric bursa, muscle bellies of the thigh and hamstrings, and pubic symphysis.

## SPECIFIC TESTS

- Before performing these tests, it is important to ask the patient to score the test. Tests are normally scored as follows:
  - + Patient feels pain but he/she has never felt this pain before.
  - ++ Patient has felt this pain before but it is not the pain that brings him/her to the office on the day of the visit.
  - +++ Patient states that the pain reproduced during the test is the pain that brings him/her to the office today.

**Impingement:**

- **FABER**: Stands for **F**lexion, **AB**duction, and **E**xternal **R**otation of the hip (Fig. 54.6A); pain during these movements represents a positive test indicating SI joint (if pain is located posteriorly) or hip (if pain is reproduced anteriorly in the groin) pathology.
- **FADIR:** Stands for **F**lexion, **AD**duction, and **I**nternal **R**otation of the hip (Fig. 54.6B); pain during these movements represents a positive test indicating either a femoral–acetabular impingement or an anterior labral tear.

**Iliopsoas:** The Thomas Test is used to test for a hip flexion contracture. The patient lies supine on the examination table and flexes the uninjured hip so that the knee is brought to the chest. If the contralateral leg remains on the table, there is no hip flexion contracture, whereas the leg will raise off of the table if a contracture is present. The bicycle test is also used to assess

**Figure 54.6. Physical Examination.** (A) Flexion, ABduction, External Rotation (FABER); (B) Flexion, ADduction, Internal Rotation (FADIR); (C) Bicycle test; (D) Posterior impingement test; (E) Adductor squeeze test; (F) Piriformis test.

for the presence of pain and snapping corresponding with iliopsoas tendinitis (Fig. 54.6C).

**Posterior Impingement:** The patient lies supine with the unaffected hip flexed as in the Thomas Test. The examiner places the affected limb in extension, external rotation, and slight abduction while applying an overpressure into hip extension (Fig. 54.6D). Pain at the posterior hip indicates posterior impingement of the labrum. Posterior impingement could be observed in patients with anteverted femurs or patients with large femoral head deformities such as SCFE or Perthes with posterior impingement.

**Adductor Squeeze Test:** Used to test the strength of the adductors

The patient lies supine on the examination table with both hips flexed to 45 degrees. The examiner places both fists between the knees and asks the patient to squeeze (adduct) both knees simultaneously (Fig. 54.6E). A noticeably lower force exerted by one of the knees indicates adductor muscle weakness. Pain can also be quantified. Adductor tendinosis is frequently seen in patients with hip FAI.

**Piriformis Test:** Used to test for piriformis syndrome

The patient lies on the side of the unaffected hip with both knees flexed and then attempts to abduct the top knee against resistance from the examiner and holds it for 30 seconds (Fig. 54.6F). A positive test is indicated by pain in the buttock or shooting pain/numbness radiating to the posterior thigh or lower leg.

**Pubalgia:** Patient is lying supine and is asked to perform a sit-up. Palpation is performed in the area of the pubis. Tenderness in this area corresponds with pubalgia or rectus anterior tendinitis. Pubalgia is frequently seen in patients with hip FAI.

## SPECIFIC INJURIES

### Proximal Hamstring Injuries

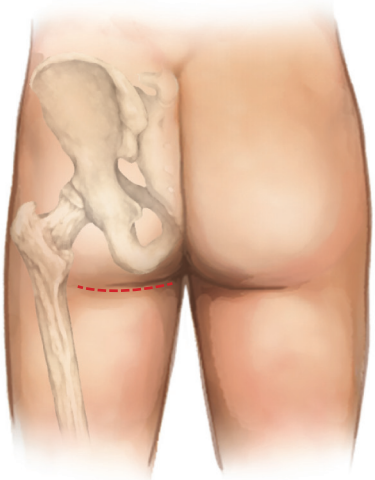
**Description:** Hamstring strain is the most common injury; this typically occurs during sprinting when there is a sudden stretch on the musculotendinous junction. In severe cases, complete avulsion may occur at the ischium. This is a very common injury seen in water skiers. Chronic tendinopathy is frequently seen in older patients as a degenerative process.

**Symptoms:** Sudden pain during exercising, particularly sprinting

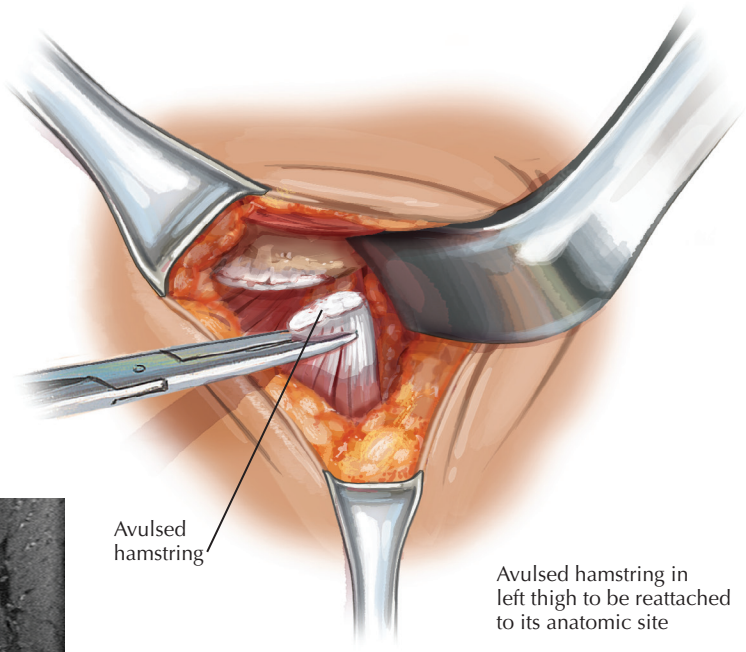
Pain may be along the posterior thigh up to the ischium. A bruise is normally evident in the buttock area. Certain patients may also report some numbness and tingling in the area corresponding to the sciatic nerve; this has been related to the presence of a hematoma that compresses the sciatic nerve.

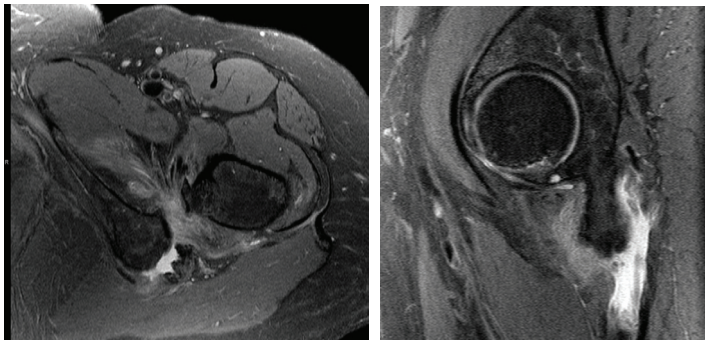
**Diagnosis:** History usually provides clues to the diagnosis. Magnetic resonance imaging (MRI) is necessary to confirm the diagnosis and define treatment (Fig. 54.7).

**Treatment:** For hamstring strains, supportive treatment (rest, ice, compression, and elevation [RICE]) is sufficient. Once the pain subsides, stretching and strengthening exercises should be performed. For complete avulsion injuries, it is important to define the degree of retraction. If the hamstring is retracted more than 3 cm, early surgical intervention (within 6 weeks) is associated with quicker return to sports and less morbidity.



Posterior view demonstrating gluteal line incision for left sided open hamstring repair



Axial *(left)* and coronal *(right)* MRI of the left hip showing complete avulsion of the proximal hamstring tendons

**Figure 54.7.** Proximal hamstring injuries.

Typically, an open surgery is performed (see Fig. 54.7); however, certain advanced hip arthroscopists may perform the procedure in a closed arthroscopic fashion. For patients with <3 cm of retraction, nonsurgical treatment with platelet rich plasma (PRP) can be offered initially.

**Prognosis:** Depends on extent of initial injury and patient's activity level

For hamstring strains, patients should be pain free with full ROM before return to play. For complete avulsion injuries, return to sports varies based on timing of surgical intervention and typically ranges 16–29 weeks following surgery.

## Rectus Femoris Injuries

**Description:** Similar to proximal hamstring injuries, strains of the rectus femoris (hip flexor strain) are more common than complete avulsions. The rectus femoris is the most commonly strained muscle in the quadriceps group. These injuries typically occur when attempting to kick a ball as in soccer or football, particularly when the patient's foot hits another player in the middle of the kicking motion.

**Symptoms:** Acute pain at the midline of the anterior thigh is most common. Pain or weakness may also be noted with resisted hip flexion or knee extension. A visible defect may be seen with complete avulsion of the rectus femoris tendon although these injuries are rare.

**Diagnosis:** Similar to hamstring strains, history and physical examination are usually sufficient for diagnosis, although MRI (Fig. 54.8) may be used to determine severity and confirm a complete avulsion of the rectus femoris from its origin on the AIIS.

**Treatment:** In cases of muscle strain, RICE and strengthening exercises are useful. With proximal rupture of the rectus femoris, nonsurgical management is an option, although surgical treatment with bone-anchoring sutures should be indicated in patients with loss of hip flexion strength and high-level athletes who wish to return to normal activity (see Fig. 54.8).

**Prognosis:** With conservative treatment, patients with rectus femoris strains do very well. Prognosis is also very good in patients who undergo surgical treatment for complete avulsion injuries, with return to sports at an average of 4 months following surgery.

## Gluteus Avulsion

**Description:** Avulsion of the gluteus could compromise the gluteus maximus, medius, or minimus; these injuries are more common in older patients. However, it can be seen after an abrupt trauma in young active patients. Gluteus medius avulsions may be chronic ("rotator cuff tear of the hip") or may occur iatrogenically during total hip arthroplasty (THA) through an anterolateral or transgluteus approach.

**Symptoms:** Lateral hip pain and weakness of hip abduction; patients who suffer gluteal avulsions during THA will typically also present with a limp.

**Diagnosis:** An MRI should always be indicated in those patients with chronic lateral pain. Several patients are inaccurately diagnosed with chronic bursitis. MRI confirms the presence of gluteal tears from the greater trochanter or within the tendon.

**Treatment:** Surgical treatment with transosseus sutures or suture anchors is necessary in case of gluteal tears with retraction. Repair is performed in a SpeeBridge fashion (double row)

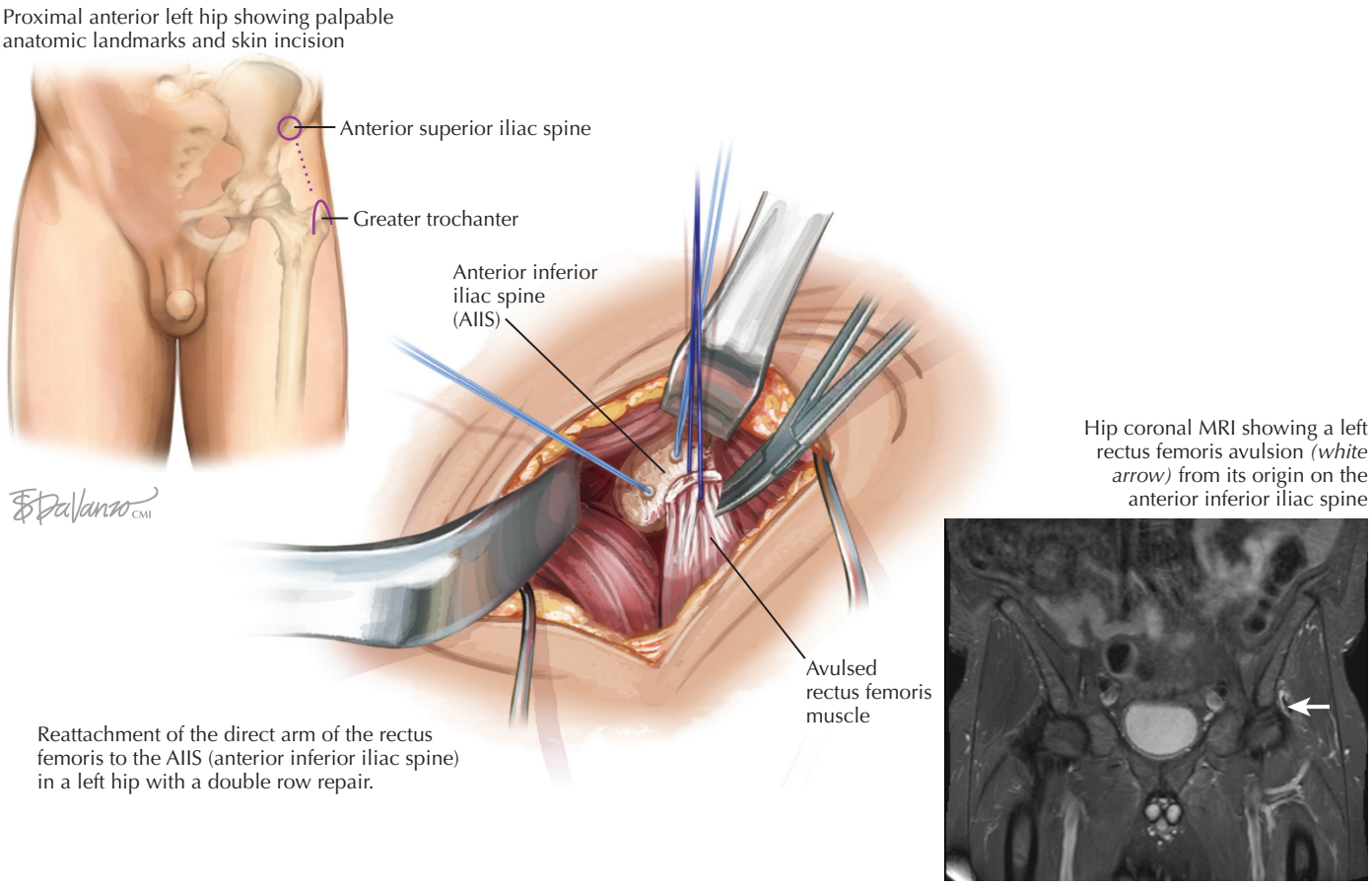



**Figure 54.8.** Rectus femoris injuries.

(Fig. 54.9). Use of an Achilles tendon allograft has been reported in patients with large chronic abductor tears. Early surgical management is preferred before the onset of muscle atrophy.

**Prognosis:** Surgical management is highly successful in relieving pain and weakness in patients with chronic tears. Patients with iatrogenic avulsions have less predictable improvement in strength after surgery.

## Femoroacetabular Impingement (FAI)

**Description:** Results from abnormal contact between the femoral head and the acetabulum; there are three subtypes of FAI: CAM, pincer, and mixed

- CAM: The femoral head is not round and therefore cannot rotate smoothly inside the acetabulum. The uneven surface of the femoral head results in an increased load on certain aspects of the acetabular cartilage during hip motion (zone of collision) (Fig. 54.10).
- Pincer: An acetabular abnormality whereby extra bone extends beyond the normal rim of the acetabulum, thereby impinging upon the femoral head as well as the labrum.
- Mixed: Patients may also have a combination of both CAM and pincer types of impingement (see Fig. 54.10).

**Symptoms:** Limited ROM, particularly in flexion and IR, with concomitant groin/hip pain

**Diagnosis:** Physical examination, such as the FADIR test, may provide clues to the diagnosis, although imaging is necessary to confirm FAI. Anteroposterior (AP) and true lateral views of the pelvis should be obtained. MRI or computed tomography (CT) may provide additional information on the femoral–acetabular incompatibility.

**Treatment:**

- Acetabular rim trimming: For patients with pincer or mixed-type deformities, arthroscopic removal of extra acetabular bone may be helpful. The labrum is normally hypoplastic in these patients and red secondary to inflammation. No more than 4–6 mm of the acetabular rim should be removed because removal beyond this results in increased loads in the hip joint (see Fig. 54.10). It is crucial to use CT before surgery to determine the amount of rim that should be trimmed. Excessive rim trimming can lead to a dysplastic hip.
- Labral repair: The femoral–acetabular mismatch in FAI results in frequent bone–bone contact, which can damage the labrum. Thus, labral repair should be performed at the same time as other procedures for FAI (see Fig. 54.10).
- Labral reconstruction: For irreparable labral tears, labral reconstruction may be necessary; this can be performed arthroscopically with autograft or allograft tissue to replace the labrum.
- Femoral osteochondroplasty: In patients with CAM-type lesions, the femoral head–neck junction may be flat or convex, whereas normally it is concave. To alleviate the resulting abnormal contact between the femoral head–neck junction and the acetabular cartilage, an open or arthroscopic procedure may be performed to resect some of the bone overgrowth (see Fig. 54.10).

**Prognosis:** A high proportion of patients are able to return to sports following surgery for FAI, with a majority of these returning to the same level of activity as before symptoms began.

## Hip Instability: Borderline Dysplasia and Developmental Dysplasia of the Hip (DDH)

**Description**: Typical anatomic changes in DDH include a misshapen femoral head, a shallow acetabulum with loss of anterolateral coverage, decreased acetabular lateral tilt, and excessive anteversion of the acetabulum and proximal femur. A combination of these bony abnormalities can result in anterior hip instability and early degenerative changes due to these abnormal hip joint forces. The center-edge angle (CEA) of Wiberg is the angle formed by a vertical line drawn from the center of the femoral head and a line from the center of the femoral head to the most lateral edge of the acetabulum. Values <20 degrees are considered abnormal (dysplastic). Values between 20 and 25 degrees are considered borderline dysplasia. The anterior center-edge angle (ACE) or Lequesne angle is measured on the false-profile view. Designed to assess anterior coverage of the femoral head, it can be calculated by measuring the angle between a vertical line through the center of the femoral head and a line connecting the center of the femoral head and the most anterior point of the acetabular sourcil. The measurement of the Tönnis angle can be determined by drawing three lines on the AP pelvic radiograph: (1) a horizontal line connecting the base of the acetabular teardrops (reference line); (2) a horizontal line parallel to line 1, running through the most inferior point of the sclerotic acetabular sourcil (point I); and (3) a line extending from point I to a point L at the lateral margin of the acetabular sourcil (the sclerotic weight-bearing portion of the acetabulum). The Tönnis angle is formed by the intersection of lines 2 and 3. Acetabula having a Tönnis angle between 0 and 10 degrees are considered normal, whereas those having an angle of >10 degrees or <0 degrees are considered to have increased and decreased inclination, respectively. Acetabula with increased Tönnis angles are subject to structural instability, whereas those with decreased Tönnis angles are at risk for pincer-type FAI.

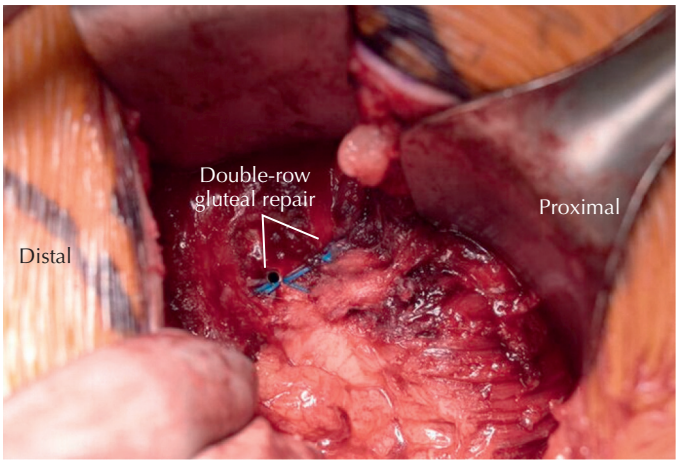
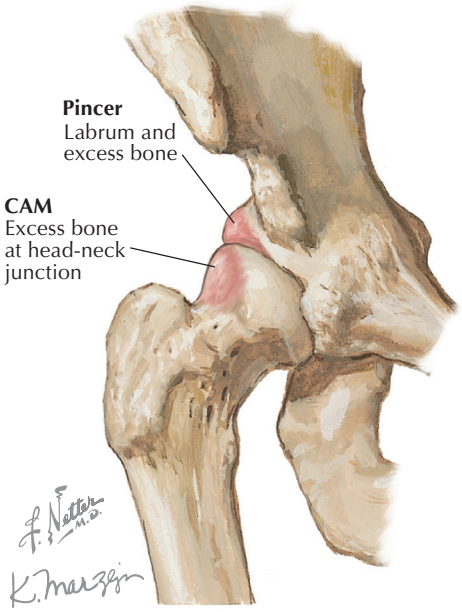



Figure 54.9. Knotless double-row repair of the gluteus medius tendon on a left hip.

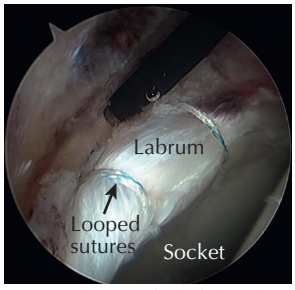
**Symptoms:** Dysplasia is more frequent in females. Patients normally present in the office complaining of hip pain. They are normally flexible and have preserved or excessive ROM,



Plain AP hip radiograph demonstrating bilateral CAM impingement in a male patient

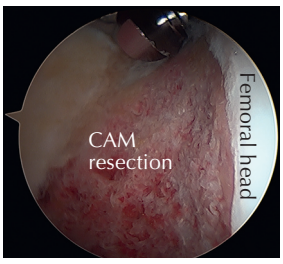
Plain AP hip radiograph with bilateral mixed FAI impingement (pincer-CAM)

Plain AP hip radiograph showing postoperative results of pincer and CAM resection in a left hip




Arthroscopic image of a left hip showing a repaired labrum with looped sutures in its superolateral portion

Dunn view of a left hip before *(left)* and after *(right)* CAM resection




Arthroscopic view of a right hip demonstrating a CAM resection

Figure 54.10. Femoroacetabular impingement.

particularly IR. Patients may also describe popping, secondary to iliopsoas irritation. Patients typically have pain while standing up, which differs from patients with FAI, who report pain during sitting.

**Diagnosis:** Physical examination, together with radiograph, MRI, and CT are necessary for an accurate diagnosis of dysplasia.

- Radiograph: AP pelvis, Dunn view, and false profile should be evaluated. The AP pelvis should be a true AP pelvis for accurate measurements of the CE angle and Tönnis angle. Dunn view will determine the presence of a concomitant FAI CAM lesion and the false profile is used to measure the ACE angle.
- CT: This is critical to evaluate the version of the socket, femoral version, and presence of a concomitant CAM lesion. Most of these patients will present with an anteverted socket (>20 degrees) and an excessively anteverted femur (>20 degrees). It is important to determine the presence of dysplasia in a tridimensional way. A hip can be anteriorly or laterally dysplastic. Lateral dysplasia includes those patients in whom the lateral center edge angle is less than 20 degrees (lateral undercoverage). Others may present with a normal CEA but with an excessively anteverted socket, which undercovers the femoral head anteriorly.

**Treatment:** Will depend on the age of the patient, status of the cartilage, and the presence of concomitant FAI CAM lesions

In patients with degenerative cartilage, surgery is contraindicated. Surgery is indicated in young and active patients with normal cartilage. Typically, a hip arthroscopy is indicated followed by a periacetabular osteotomy (PAO). Hip arthroscopy should be performed before PAO to treat intra-articular pathology, including labrum repair, ligamentum teres tears, and cartilage damage. In such cases, a PAO is indicated to correct the dysplastic socket. In general, surgery is performed to correct the anterior and lateral coverage. However, in certain situations, a reverse PAO is indicated. This is indicated for patients with an excessively retroverted socket and posterior instability.

## Perithrocanteric Space Disorders

### Gluteal Bursitis and Ischial Bursitis

**Description:** Also known as *ischiogluteal bursitis* and results from inflammation of the ischiogluteal bursa found between the gluteus maximus and the ischial tuberosity

**Symptoms:** Pain at the medial buttock; may be difficult to distinguish from hamstring strain

**Diagnosis:** Pain elicited by pressure over the ischial tuberosity with the patient in the lateral decubitus position; absence of pain with stretching distinguishes ischial bursitis from a hamstring injury

**Treatment:** Conservative treatment with anti-inflammatory medications and modification of training regimen; use of corticosteroid injections into the ischiogluteal bursa and ischiogluteal bursectomy are poorly studied.

**Prognosis:** Return to sports may be allowed based on relief of symptoms. In refractory cases, more extensive diagnostic tests such as MRI may be necessary to rule out other pathologies such as hamstring tendon avulsions.

### Trochanteric Bursitis

**Description:** Inflammation of the trochanteric bursa found just superficial to the greater trochanter

**Symptoms:** Pain in the buttock or lateral hip, exacerbated by lying on the affected side, going from a seated to a standing position, or running on banked surfaces such as roadsides

**Diagnosis:** Pain elicited by pressure at the lateral edge of the greater trochanter; occasionally, MRI is necessary to confirm the diagnosis and exclude the presence of a tear or tendinopathy of the glut

**Treatment:** Conservative treatment with anti-inflammatory medications, modification of training regimen, and corticosteroid injections into the trochanteric bursa; in refractory cases, arthroscopic bursectomy, iliotibial band release, or trochanteric reduction osteotomy can be considered.

**Prognosis:** Return to sports may be allowed based on relief of symptoms. Improvement in symptoms has been shown in a majority of patients undergoing various surgical treatments for trochanteric bursitis, although return to sports has not been well-defined in these patients.

### Piriformis Syndrome

**Description:** Results from entrapment of the sciatic nerve under the piriformis muscle at the sciatic notch

**Symptoms**: Pain and symptoms of sciatica with sitting as this compresses the piriformis muscle against the sciatic nerve

**Diagnosis**: Typically, a diagnosis of exclusion; SI joint dysfunction and lumbar disc herniation should be ruled out through pelvic radiographs and MRI of the lumbar spine, respectively.

**Treatment:** Massage of the piriformis muscle in addition to physical therapy with piriformis stretching exercises may improve symptoms in mild cases. Use of nonsteroidal anti-inflammatory medications may reduce inflammation at the site of nerve compression. Corticosteroid injections directly into the piriformis muscle may reduce muscle spasms and pain. In more severe cases, Botox injections in the piriformis muscle may be more effective in relaxing the muscle. In patients who do not respond to conservative treatment for ≥6 months, release of the distal tendon of the piriformis muscle may be performed.

**Prognosis:** Return to sports may be allowed based on relief of symptoms. Good outcomes have been shown in a majority of patients undergoing surgical release.

## SUMMARY

- History, physical examination, and imaging are the keys to accurate diagnosis of hip pathology.
- There have been significant advancements in arthroscopic techniques for surgical repair of hip pathology in recent years, which has resulted in an increase in the number of less invasive hip procedures.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 55 KNEE INJURIES

Eric C. McCarty • W. Michael Walsh • Christopher C. Madden

## PHYSICAL EXAMINATION

### Anatomy of the Knee

See Fig. 55.1.

### Observation and Measurement

#### Standing

**Alignment of lower extremities:** View the patient from the front, side, and back.
**Angular and rotational deformities:** Excessive valgus, varus, recurvatum, flexion contracture, and femoral or tibial torsion
**Foot alignment and mechanics:** Excessive cavus or pes planus; heels should invert and arches increase on toe rising
**Leg length inequality:** Best judged by pelvic levelness on standing
**Difference in size of legs:** Atrophy of one limb versus hypertrophy of opposite limb
**Popliteal masses:** May be seen better in the prone position

#### Sitting

**Patellar position:** With patient's knees flexed 90 degrees, check from the side to judge a high or low position. The anterior patellar surface normally faces the wall in front of the patient sitting with legs over the side of the examination table. View from the front to judge lateral posture. The patella should appear centered in the soft tissue outline of the knee.
**Osgood–Schlatter changes:** Enlarged and/or tender tibial tuberosity
**Vastus medialis obliquus/vastus lateralis (VMO/VL) relationship:** With the patient's knees held actively at 45 degrees of flexion (Fig. 55.2), the distal one-third of the vastus medialis should normally present as a substantial muscle from the adductor tubercle, inserting into upper the one-third to one-half of the medial patella. Dysplastic vastus medialis obliquus (VMO) appears hollow in this normal muscular location (see Fig. 55.2); also observe for apparent hypertrophy of vastus lateralis (VL).
**Patellar tracking:** Observe on active flexion and extension; watch for excessive displacement of the patella.

#### Lying

SUPINE

**Range of motion:** Both active and passive; compare the injured with the uninjured side
**Muscle bulk:** Thigh and calf; can measure circumferences
**Quadriceps (Q) angle:** With quadriceps contracted, measure the angle between the line from the anterior superior iliac spine to the midpoint of the patella and the line from the midpoint of the patella to the tibial tuberosity (see Fig. 55.2); normal in males is ≤10 degrees and in females is ≤15 degrees.
**Hamstring and heel cord tightness:** See Chapter 42, Musculoskeletal Injuries in Sports

PRONE

**Range of motion:** Lack of full knee flexion may show quadriceps tightness
**Popliteal masses:** Compare contours with those of the opposite knee

#### Walking/Running

**Mechanics of gait:** Stance and swing phase from side to side is even; look for limp, other asymmetry, excessive limb rotation, or limb malalignment.
**Patellofemoral tracking:** Observe the patella closely from the front view.

### Palpation

**Joint effusion:** With the patient's knee extended, milk fluid from the suprapatellar pouch, and palpate along medial and lateral sides of the patella (see Fig. 55.2). Try to distinguish intra-articular effusion that can be moved about from extra-articular swelling that feels more like thick soft tissue, which is not movable.
**Significant areas of tenderness:**
- Menisci: medial and lateral joint lines
- Ligament attachments: medial femoral epicondyle, adductor tubercle, lateral femoral epicondyle, proximal medial tibia
- Tendons: patellar tendon, quadriceps tendon, popliteus tendon, hamstrings
- Bursae: prepatellar, pes anserinus, tibial collateral ligament, deep infrapatellar
- Other: patellar facets, extensor retinaculum

**Crepitation: During range of motion**—from any rough joint surface (particularly patellofemoral joint), fractures, or soft tissue thickness; **patellofemoral compression**—longitudinal and/or transverse compression of the patella against the femur. Examine for crepitation or ask regarding any elicited pain.
**Muscle tone:** Overall turgor of muscle tissue; may be decreased early after injury, even if the bulk still measures normal

### Specific Tests

Perform all tests on an uninjured knee first to establish "normal" baseline for that patient (Table 55.1).

#### Ligaments

MEDIAL

**Valgus stress test at 30 and 0 degrees:** The patient is supine and relaxed, with the thigh supported on the table. The examiner applies valgus force at the foot while using other hand as a fulcrum along the lateral side of the joint. Watch and feel for medial joint line opening. Perform first with the knee flexed to 30 degrees, then with maximum possible extension or hyperextension (Fig. 55.3).
**Anterior drawer test with external rotation of tibia:** The patient is supine and relaxed, with the hip flexed to 45 degrees and knee to 90 degrees. Externally rotate the foot 30 degrees; then, pin the foot to the table with the examiner's thigh. Grasp the proximal tibia with both hands and pull toward the examiner. A positive test is excessive anterior rotation of the medial tibial condyle (see Fig. 55.3).

LATERAL

**Varus stress test at 30 and 0 degrees:** The patient is in the same position as for the abduction stress test. Reverse the hand

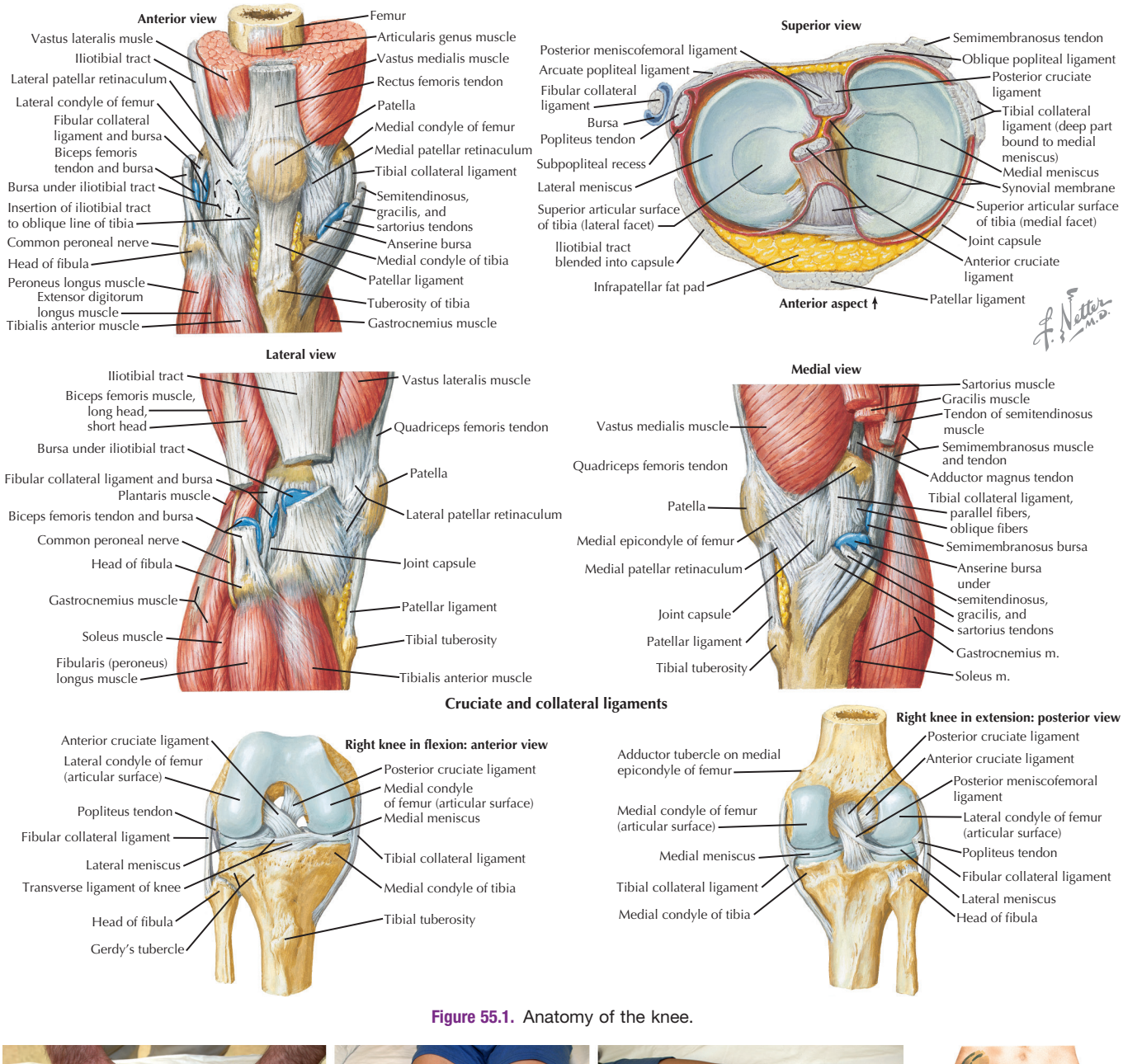
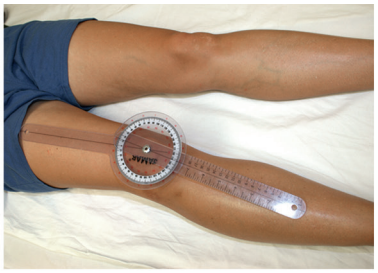


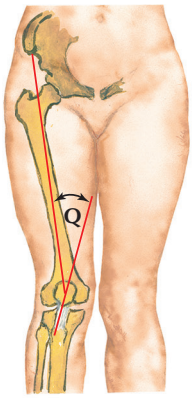

**Figure 55.1.** Anatomy of the knee.

**Effusion.** Fluid is milked from suprapatellar pouch with one hand and with other and the fluid is palpated on the sides of the patella.

**Vastus medialis obliquus (VMO).** Patient with marked VMO dysplasia. This is probably the most important predisposition to all extensor mechanism syndromes.



**Quadriceps (Q) angle measurement.** With quadriceps contracted, proximal arm of goniometer is directed toward anterior superior spone, pivot point of goniometer is directed toward anterior superior spone, pivot point of goniometer is placed over the center of patella, and distal arm of goniometer is placed on tibial tuberosity. Normal in males, up to 10 degrees; females, 15 degrees.




**Q angle** formed by intersection of lines from anterior superior iliac spine and from tibial tuberosity through midpoint of patella. Large Q angle predisposes to patellar subluxation.

**Figure 55.2.** Observations and measurement of the knee.

**TABLE 55.1 KEY PHYSICAL EXAMINATION TESTS AND INJURED STRUCTURES**

| Test | Injured Structure |
|---|---|
| Valgus stress test at 30 degrees and 0 degrees | 30 degrees: Medial collateral ligament<br>0 degrees: Posteromedial corner, medial collateral ligament, posterior cruciate ligament, possibly anterior cruciate ligament |
| Varus stress test at 30 degrees and 0 degrees | 30 degrees: Lateral collateral ligament<br>0 degrees: Posterolateral corner, lateral collateral ligament, posterior cruciate ligament |
| Lachman test | Anterior cruciate ligament |
| Anterior drawer test | Anterior cruciate ligament, but affected by other structures such as collaterals |
| Pivot shift test/jerk test | Anterior cruciate ligament |
| Posterior drawer test | Posterior cruciate ligament |
| Gravity or sag test | Posterior cruciate ligament |
| Posterolateral drawer test | Posterolateral corner structures |
| External rotation recurvatum test | Posterolateral corner structures |
| McMurray's test | Menisci |
| Apley's compression test | Menisci |
| Apprehension test | Medial patellofemoral ligament and retinaculum |
| Prone external rotation test (dial test) | Posterolateral corner structures |

position so that one hand applies varus stress while the other acts as a fulcrum along the medial side of the joint. Watch and feel for lateral joint line opening. Perform the test at 30 degrees of flexion and then at full possible extension or hyperextension (see Fig. 55.3).

**External rotation recurvatum test:** The patient is supine and relaxed. Lift the entire lower extremity by the first toe. Observe for excessive recurvatum and external rotation of the proximal tibia (tibial tuberosity) and apparent varus deformity of the knee; indicates posterolateral corner injury

**Posterolateral drawer test:** Same position as for the anterior drawer test with external rotation of the tibia; the examiner's hands pushes posteriorly on the proximal tibia; a positive test is excessive posterior rotation of the lateral tibial condyle (see Fig. 55.3).

**Prone external rotation test (Dial test):** The patient is prone with the knees together, and the feet are externally rotated at 30 degrees of knee flexion and then at 90 degrees. The external rotation of the foot relative to the thigh is compared with the contralateral side. The test is positive if there is >10 degrees of rotation of the affected side compared with that of the normal side. If asymmetry is present only at 30 degrees, then isolated posterolateral corner injury is likely. If asymmetry is present at both 30 and 90 degrees, combined injury to the posterior cruciate ligament (PCL) and posterolateral corner is present (see Fig. 55.3).

**Reverse pivot shift test:** Performed with the tibia in external rotation rather than internal rotation; with knee flexed 90 degrees, the lateral tibial condyle is subluxed posteriorly. With further knee extension, tibia reduces with detectable "clunk" (see later discussion of pivot shift test).

#### ANTERIOR CRUCIATE LIGAMENT (ACL)

**Lachman test:** The patient is supine and relaxed; the examiner grasps the distal femur with one hand, while other hand grasps the proximal tibia; knee flexed to approximately 15–20 degrees; apply anterior force to proximal tibia. Positive test is excessive anterior translation of the tibia beneath the femur and lack of a firm endpoint (see Fig. 55.3).

**Anterior drawer test in neutral rotation:** Same position as for anterior drawer with external rotation of the tibia except that foot and tibia are in neutral rotation; anterior pull is applied to the proximal tibia. Positive test is anterior translation of both tibial condyles from beneath the femur (see Fig. 55.3). *Note:* This test is influenced by structures other than anterior cruciate ligament (ACL). Do not rely on this test for diagnosis of ACL tear.

**Pivot shift test and jerk test:** The patient is supine and relaxed. Begin with knee fully extended (pivot shift test) or flexed to 90 degrees (jerk test); foot and tibia internally rotated; valgus applied at knee; knee progressively flexed (pivot shift test) or extended (jerk test). At approximately 30 degrees, watch and feel for anterior subluxation of lateral tibial condyle: tibia suddenly reduces with further flexion (pivot shift test) or extension (jerk test) (see Fig. 55.3).

#### POSTERIOR CRUCIATE LIGAMENT (PCL)

**Posterior drawer test:** Same position as for anterior drawer test in neutral rotation; posterior force is applied to proximal tibia. Positive test is straight posterior displacement of both tibial condyles (see Fig. 55.3). *Caution:* Ensure a neutral position as the starting point. Compare position of the tibia relative to the femur with normal knee. It is easy to start from a posteriorly displaced position and interpret reduction to neutral as a positive anterior drawer sign rather than starting at neutral and interpreting as a positive posterior drawer sign.

**Gravity or sag test:** The patient is supine and relaxed. Flex hips to 45 degrees and knees to 90 degrees with feet flat on table. With quadriceps relaxed, observe from the lateral side for posterior displacement of one tibial tuberosity compared to the other; then flex the hips to 90 degrees, support both legs by the ankles and feet, and observe again (see Fig. 55.3).

**Valgus or varus stress test at 0 degrees:** As described for abduction and adduction stress tests at 30 and 0 degrees; positive test in full extension in acute case is often due to PCL rupture in addition to injury to associated collateral ligaments (see Fig. 55.3).

### Menisci

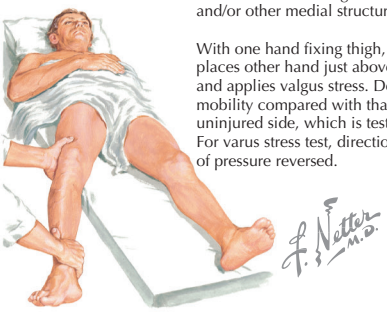
**Joint line tenderness:** Tenderness along the medial or lateral joint lines is among the most sensitive findings for a meniscal tear (see Fig. 55.3).

**McMurray's test:** The patient is supine and relaxed. Have patient flex knee maximally with external tibial rotation (medial meniscus) or internal tibial rotation (lateral meniscus). While maintaining rotation, patient brings the knee into full extension. Positive test is a painful pop occurring over the medial (medial meniscus) or lateral (lateral meniscus) joint line (see Fig. 55.3).

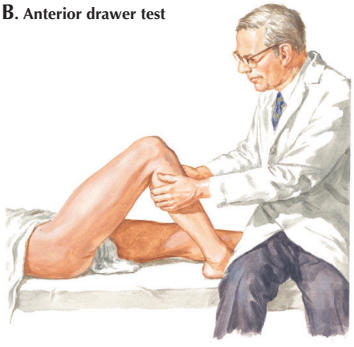
**Apley's compression test:** The patient is in joint line prone position. Knee is flexed to 90 degrees with external tibial rotation (medial meniscus) or internal tibial rotation (lateral meniscus). Apply axial compression to joint line tibia while joint line patient flexes and extends joint line knee. Positive test is a painful pop over joint line medial (medial meniscus) or lateral (lateral meniscus) joint line (see Fig. 55.3).

**A. Varus and valgus tests**
Patient supine on table, relaxed, leg over edge of table, flexed about 30° for medial ligament injury. When test is done at 0° and loose then there is typically an associated cruciale ligament injury and/or other medial structures.

With one hand fixing thigh, examiner places other hand just above ankle and applies valgus stress. Degree of mobility compared with that of uninjured side, which is tested first. For varus stress test, direction of pressure reversed.




**B. Anterior drawer test**



Test is performed with foot in neutral postition . . .

then with foot in progressive degrees of external rotation . . .

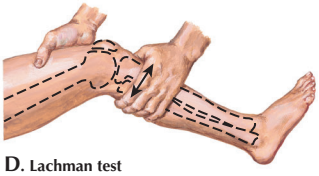
and with foot in progressive degrees of internal rotation

Patient supine on table, relaxed, with head on pillow; hip flexed 45°, knee flexed 90°; foot flat on table. Examiner sits partially on dorsum of patient's foot to stabilize it, places hands on each side of upper calf as shown. Anterior force applied to proximal tibia while index fingers ensure that hamstrings are relaxed. May also be done with external rotation and internal rotation of foot and tibia.

**C. Prone external rotation test (dial test)**

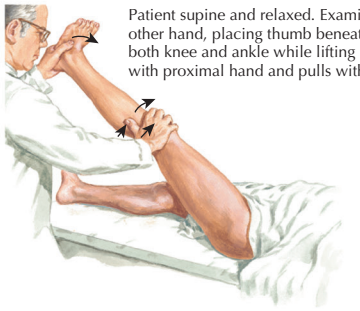
Dial test at 30°

Dial test at 90°



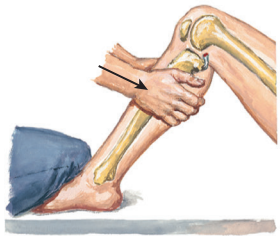
**D. Lachman test**
With patient's knee bent 20° to 30°, examiner's hands grasp limb over distal femur and proximal tibia. Tibia alternately pulled forward and pushed backward. Movement of 5 mm or more than that in normal limb indicates rupture of anterior cruciate ligament.

**E. Pivot shift test/Jerk test**

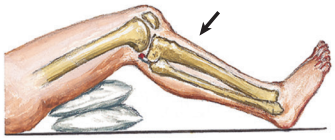


Patient supine and relaxed. Examiner lifts heel of foot to flex hip 45° keeping knee fully extended; grasps knee with other hand, placing thumb beneath head of fibula. Examiner applies strong internal rotation to tibia and fibula at both knee and ankle while lifting proximal fibula. Knee permitted to flex about 20°; examiner then pushes medially with proximal hand and pulls with distal hand to produce a valgus force at knee.

As internal rotation, valgus force, and forward displacement of lateral tibial condyle maintained, knee passively flexed. If anterior subluxation of tibia (anterolateral instability) present, sudden visible, audible, and palpable reduction occurs at about 20° to 40° flexion. Test positive if anterior cruciate ligament ruptured, especially if lateral capsular ligament also torn.



**F. Posterior drawer test.** Procedure same as for anterior drawer test, except that pressure on tibia is posterior instead of anterior.



**G. Sag test.** Posterior cruciate ligament rupture. Left knee shows positive gravity test with sagar left tibia.

**H. Joint line tenderness**

Both knees are palpated at the same time by thumbs. Tenderness of medial joint can be evaluated by comparing both sides.

Lateral joint line is palpated with knee in figure-of-four position.

**I. Apley's compression test**

Position for lateral meniscus with pressure on sole of foot, tibia is externally rotated while knee is flexed and extended.

For medial meniscus, foot is internally rotated while knee is flexed and extended.

**J. McMurray's test**

Position for lateral meniscus. With foot in internal rotation, knee is brought from full flexion to extension while fingers palpate lateral joint line.

Position for medial meniscus. With external tibial rotation, knee is brought from full extension while fingers palpate medial joint line.

**Figure 55.3.** Special tests.

### Patella

**Hypermobility/apprehension test:** The patient is supine and relaxed. The examiner sits on edge of the table with the patient's knee flexed approximately 30–45 degrees across the examiner's thigh. With the patient's quadriceps relaxed, the examiner uses both thumbs to forcefully displace the patella over the lateral femoral condyle. Positive test is increased lateral mobility of the patella compared to the opposite knee or other patients; more important is discomfort or extreme apprehension that the patella is going to dislocate because of lateral displacement.

**Plica tests:** The patient is supine and relaxed. With the tibia internally rotated, the examiner passively flexes and extends knee from 30 to 100 degrees of flexion. Examining the fingers placed along the medial patellofemoral joint may feel a click, possibly some tenderness, or even a pop of a pathologic plica.

## KNEE LIGAMENT INJURIES

### Medial Ligaments

**Description:** Injury to medial (tibial) collateral ligament and/or medial capsular ligament (Fig. 55.4)

**Mechanism of injury:** Valgus force applied to the knee with external tibial rotation; may be noncontact twist or a blow to lateral side of joint

**Presentation:** Initial pain on medial side of the knee; with complete tear, complaints of the knee giving way into valgus

**Examination:** Positive valgus stress test at a 30-degree flexion; compare with opposite knee. An injured medial collateral ligament (MCL) along with disrupted ACL or PCL will result in more gap occurring with a valgus stress test, particularly noticeable when the knee is tested in extension. Frequently, but not always, positive anterior drawer sign results with the tibia in external rotation. The medial tibial condyle rotates anteriorly.

**Imaging:** Abduction stress radiographs may be used to distinguish ligament injury from epiphyseal fracture in skeletally immature athletes. Fracture opens at the growth plate; ligament tear opens at the joint line; perform in 20–30 degrees of flexion

**Differential diagnosis:** In young patients, epiphyseal fracture of the distal femur or proximal tibia; patellar dislocation (may be associated with MCL tear); medial meniscus tear (may be associated with MCL tear)

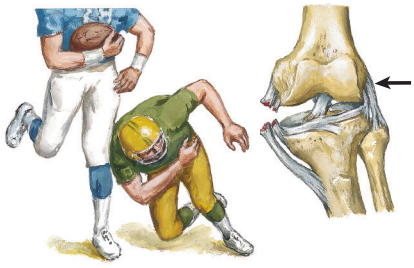
**Treatment:**

- **Grades I and II sprains:** Rest, ice, compression, and elevation (RICE), crutches, rehabilitation
- **Grade III sprain (complete ligament tear):** With other associated injuries, surgery may be considered (currently rare); if no surgery indicated, immobilization should be used for short period after acute injury; begin rehabilitation program as soon as possible; with only mild instability, rigid immobilization may not be necessary. RICE and functional rehabilitation may be adequate treatment.

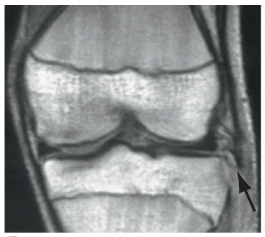
### Lateral Ligaments

**Description:** Sprain or tear of lateral (fibular) collateral ligament and/or lateral capsular ligament; may be associated injuries to popliteus tendon, iliotibial band, popliteofemoral ligament, and peroneal nerve

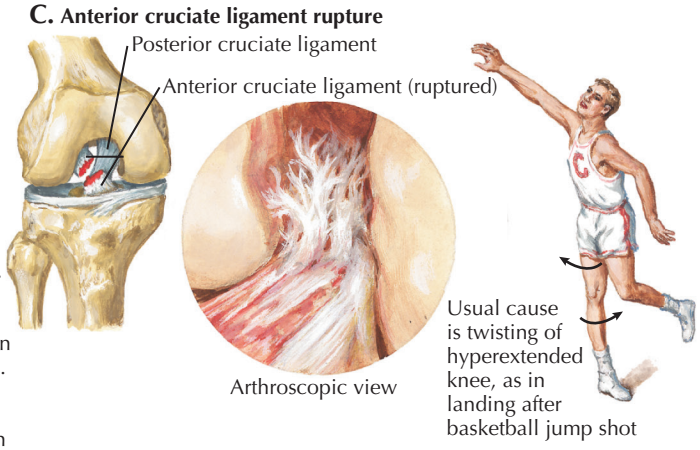
**Mechanism of injury:** Varus or twisting injury; may be contact or noncontact. Posterolateral ligaments often injured by a hyper-

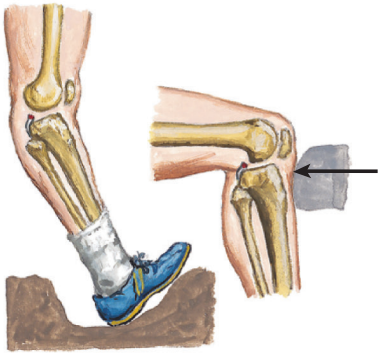


**A. Medial collateral ligament injury.** Usual cause is forceful impact on posterolateral aspect of knee with foot anchored, producing valgus stress on knee joint. Valgus stress may rupture tibial collateral and capsular ligaments.

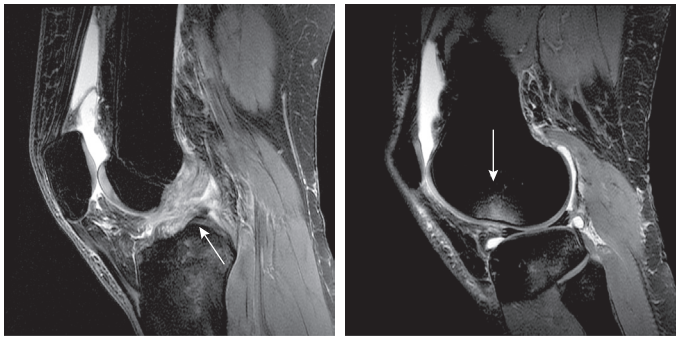


**B. Lateral ligament injury. Segond fracture of the knee.** Coronal proton density-weighted MRI demonstrates avulsion of the bony insertion of the iliotibial band *(arrow)*. Avulsions of the lateral collateral ligament complex have a close association with ACL injury. (From Adam A, Dixon A, Grainger R, Allison D. *Grainger & Allison's Diagnostic Radiology,* 5th ed. Philadelphia: Elsevier; 2008.)



**D. Posterior cruciate ligament injury.** Usual causes include hyperextension injury, as occurs from stepping into hole, and direct blow to flexed knee.



**Acute anterior cruciate ligament tear.** Fat-suppressed proton density–weighted image *(left)* shows irregular distorted fibers of torn ACL *(arrow)*. Hyperintense bone marrow edema *(right)* in lateral femoral condyle (*arrow*) is a secondary sign often seen with acute ACL injury. (Reused with permission from Witte D. Magnetic Resonance Imaging in Orthopaedics in *Campbells Orthopedics*. Elsevier; 2013).

**Figure 55.4.** Knee ligament injuries.

extension mechanism, frequently with a blow to the anteromedial tibia

**Presentation:** Pain is present over the lateral ligament complex. Knee may give way on twisting, cutting, or pivoting. In chronic cases, posterolateral corner injury gives a feeling of giving way into hyperextension when standing, walking, or running backward.

**Examination:** Compare with opposite knee; in acute case, may be increased varus stress test at 30 degrees of flexion and positive posterolateral drawer sign; chronic cases show a positive reverse pivot shift test and external rotation recurvatum test. External rotation recurvatum may also be apparent on standing, giving increased varus appearance to the knee.

**Imaging:** Lateral capsular sign shows avulsion of the midportion of the lateral capsular ligament with a small fragment of proximal lateral tibia. Associated with a high incidence of anterior cruciate tear and indicates anterolateral instability (see Fig. 55.4). Arcuate sign shows avulsion of proximal fibula with the posterolateral ligament complex; indicates posterolateral instability

**Differential diagnosis:** Chronic posterolateral injury may be confused with medial compartment arthritis because of progressive varus appearance; difficult to differentiate from posterior cruciate injury. Acute lateral ligament injury may be confused with lateral meniscus tear. Injury to the middle third of the lateral capsular ligament, as shown by lateral capsular sign on radiograph, usually associated with ACL injury.

**Treatment:**

- **Grade I and II sprains:** RICE, crutches, rehabilitation
- **Grade III sprain (complete ligament tear):** Surgical repair is usually preferable if injury involves more than just lateral (fibular) collateral ligament. Immobilization is not really useful by itself. Mild instability may be treated by RICE and functional rehabilitation.

### Anterior Cruciate Ligament (ACL)

**Description:** Tear of part or all of two major bundles (posterolateral and anteromedial) of ACL; may be associated with tears of middle one-third of lateral capsular ligament. ACL is torn from femur or tibia, or torn in its midportion; may avulse tibial spine in young patients (see Fig. 55.4)

**Mechanism of injury:** Hyperextension, varus/internal rotation, and extremes of valgus and external rotation are possible causes.

**Presentation:** Usually a loud pop occurs; may be followed by autonomic symptoms of dizziness, sweating, faintness, and slight nausea. A large swelling usually occurs within first 2 hours after an acute injury (hemarthrosis). Conversely, most acute hemarthroses (85%) are anterior cruciate tears; in chronic cases, complaints of giving way on twisting, pivoting, and cutting

**Examination:** Acute, large hemarthrosis, positive Lachman test; chronic, positive Lachman test, positive pivot shift test or jerk test; perhaps a positive anterior drawer sign but not reliable; do not rely on the anterior drawer sign

**Imaging:** Lateral capsular sign; avulsion of the tibial spine may be seen in young patients; magnetic resonance imaging (MRI) useful in acute injury to confirm diagnosis and evaluate for injuries to other structures; reported accuracy rates as high as 95% in detecting ACL tears (see Fig. 55.4)

**Differential diagnosis:** Acute, differentiate from other causes of hemarthrosis (e.g., osteochondral fracture, peripheral meniscus tear, and patellar dislocation); chronic, differentiate from other types of ligamentous laxity and/or meniscal tears

**Treatment:**

- Acute
  - Various methods delineate degree of damage and associated injuries
  - Knee may be treated symptomatically, followed by repeated evaluations over initial 2–3 weeks following injury.
  - Most active patients engaged in agility sports require surgical reconstruction.
  - Reconstruction is now usually delayed at least 3 weeks after injury to allow decrease in swelling and increase in range of motion.
  - For mild laxity with a firm endpoint (partial ACL injury) and no other associated injury, may treat with PRICES, functional rehabilitation, and protective bracing
  - Apparent partial injuries often progress to more obvious complete tears.
- Chronic
  - May attempt functional stabilization through rehabilitation, bracing, lifestyle modification; often requires surgical reconstruction

### Posterior Cruciate Ligament (PCL)

**Description:** Tear of part or all of two major bundles of the PCL (posteromedial and anterolateral)

**Mechanism of injury:** Valgus/varus in full extension; in rare cases, severe twist; direct blow to the anterior proximal tibia, as in fall on artificial turf or other hard playing surface

**Presentation:** Usually less swelling than with ACL; otherwise, in acute stage, nothing particularly distinguishing; chronically, feeling of femur sliding anteriorly off tibia, particularly when rapidly decelerating or descending slopes or stairs

**Examination:** Acute, if produced by varus or valgus mechanism, may find abduction or adduction stress test positive in full extension; if produced by blow to anterior tibia, posterior drawer sign may be positive; chronic, rely on posterior drawer sign and gravity test (see Fig. 55.4)

**Imaging:** Cross-table lateral view radiographs may show sag of tibia compared to opposite side; may accentuate by doing posterior drawer sign while taking cross-table lateral view; may see bony avulsion with tibial attachment of the PCL; MRI shows posterior cruciate well and may help confirm diagnosis and evaluate for other injuries (see Fig. 55.4).

**Differential diagnosis:** Most difficult is distinguishing posterior cruciate injury from posterolateral corner injury; posterior drawer sign and posterolateral drawer sign may appear the same. Both injuries may exist in same knee.

**Treatment: Acute,** most important to delineate degree of injury; may require examination under anesthesia and arthroscopy; for mild laxity (isolated PCL tear), may treat with PRICES, functional rehabilitation, protective bracing; for moderate or severe laxity, surgical repair/reconstruction is usually required

**Chronic,** may attempt functional stabilization through rehabilitation and bracing; often requires surgical reconstruction if instability is more than mild

## MENISCAL INJURIES

### Medial Meniscus

**Description:** Disruption of medial semilunar cartilage of the knee; may be from single traumatic episode, degenerative processes, or a combination; tears take different forms, such as radial, longitudinal, or horizontal. Most important surgical factor is whether the tear is peripheral in the vascular zone or more central in the nonvascular zone; more common than lateral meniscus tears because medial meniscus is less mobile

**Mechanism of injury:** Twisting or squatting; may be in association with ligament injuries due to any of their precipitating mechanisms

**Presentation:** Usually mild swelling and joint line pain; in an acute setting, important to know whether knee lacked full extension

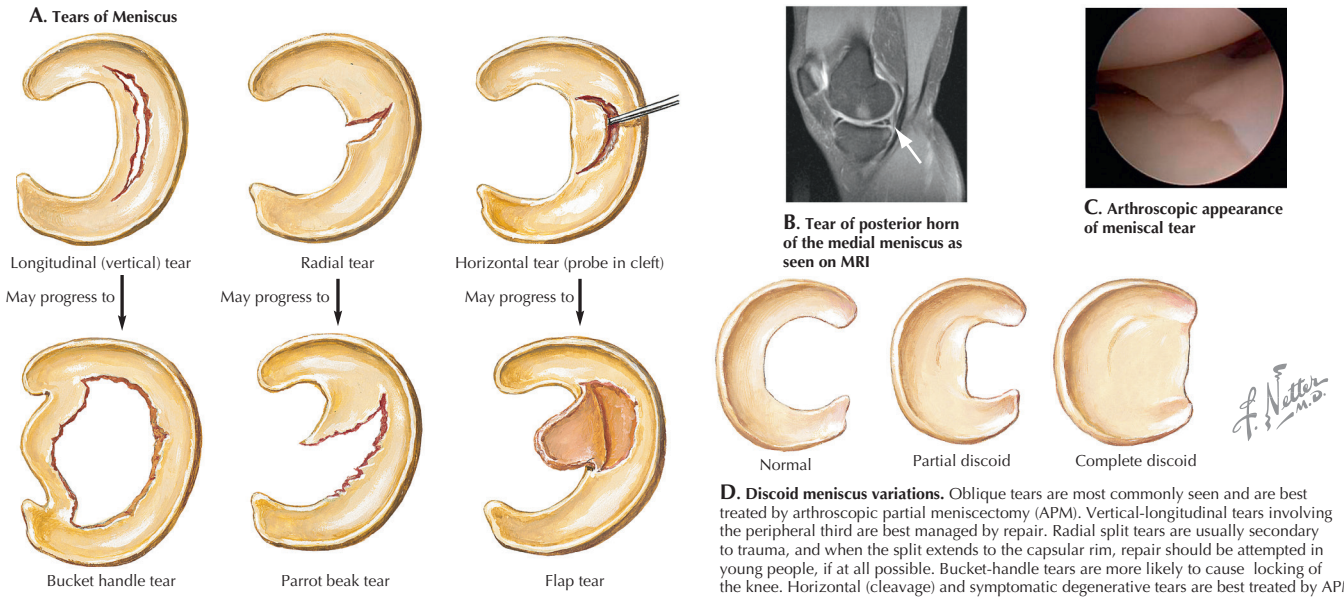



**Figure 55.5.** Meniscal injuries. (MRI and arthroscopy images from Hart J, Miller M. Netter's *Musculoskeletal Flash Cards*. Philadelphia: Elsevier; 2007.)

from time of injury (locked knee from displaced fragment) or knee lacked full extension next day (pseudolocking from hamstring spasm). In a chronic setting, recurrent locking is typical. Otherwise, symptoms may include slipping or catching over the joint line.

**Examination:** Positive McMurray's and Apley's tests; results may vary considerably from one examination session to the next. Joint line tenderness and mild effusion may be present. Chronically, quadriceps atrophy is common. With peripheral meniscus detachment and positive anterior drawer test, loud "clunk" may be elicited as the meniscus displaces during the anterior drawer test.

**Imaging:** Plain radiographs are usually normal, unless meniscal tear has been present for a significant amount of time. Thereafter, they may show joint line spurring and/or narrowing. MRIs have now supplanted arthrograms for diagnosis of meniscal injury. For medial meniscus, MRI has a sensitivity as high as 94% (Fig. 55.5).

**Differential diagnosis:** Ligamentous injury (causing pain in the same area), patellar problems (anteromedial joint pain that is confused with pain from medial meniscus injury), pathologic synovial plica (similar pain, swelling, catching, and popping), loose bodies may cause locking, medial compartment arthritis (medial joint pain similar to that from torn meniscus)

**Treatment:** Suspected meniscus tear with no ligamentous instability may be managed initially through symptomatic treatment and functional rehabilitation. If no improvement, or if time constraints do not allow initial conservative treatment, diagnostic arthroscopy is pathologically the most certain way of diagnosing and treating meniscal injury. MRI may help decide whether to proceed with surgical treatment or continue with nonsurgical care. Most meniscal tears still require arthroscopic partial meniscectomy. Meniscectomy increases the risk of development of future arthritic changes in the knee. Vertical tears in the peripheral vascular zone are now routinely treated by meniscal repair rather than removal of the meniscus.

### Lateral Meniscus

**Description:** Disruption of lateral semilunar cartilage of the knee; may be from single traumatic episode, degenerative processes, or combination; with lateral meniscus injury, may also encounter injuries of congenital discoid meniscus (see Fig. 55.5). Tears take different forms, such as radial, longitudinal, or horizontal (see Fig. 55.5). Most important surgical factor is whether the tear is peripheral in the vascular zone or more central in the nonvascular zone. Lateral meniscus tears are less common than medial meniscus tears because the lateral meniscus is more mobile (see Fig. 55.5).

**Mechanism of injury:** Same as for medial meniscus injury

**Presentation:** Same as for medial meniscus injury, although often more pain and fewer mechanical symptoms than with medial meniscus tears; patients may give history of a cystic lesion directly over the lateral joint line.

**Examination:** Much the same as for medial meniscus injury; may palpate localized puffiness or distinct cystic lesion over lateral joint line

**Imaging:** Same findings as for medial meniscus injury; in children with congenital discoid lateral meniscus, widening of the lateral joint space may be seen. In contrast to medial meniscus, MRI has somewhat less sensitivity (approximately 78%) in detecting lateral meniscus tears.

**Differential diagnosis:** Lateral ligamentous injury, loose bodies, degenerative arthritis of lateral compartment; popliteus tendinitis; iliotibial band friction syndrome

**Treatment:** Same as for medial meniscus injury; special considerations in youngsters with lateral discoid meniscus include whether to remove or repair and how much meniscus to remove

## EXTENSOR MECHANISM PROBLEMS

### Instability Syndromes

#### Dislocation

**Description:** Complete, usually lateral displacement of the patella from femoral trochlea that persists until reduced, usually by extending knee

**Mechanism of injury:** Valgus and/or twisting with strong quadriceps contraction

**Predisposing factors:** All the stigmata indicating congenital extensor mechanism malalignment, such as VMO dysplasia, VL hypertrophy, high and lateral patellar posture, increased Q-angle, and bony deformity; usually more easily seen in acute cases on the opposite uninjured side

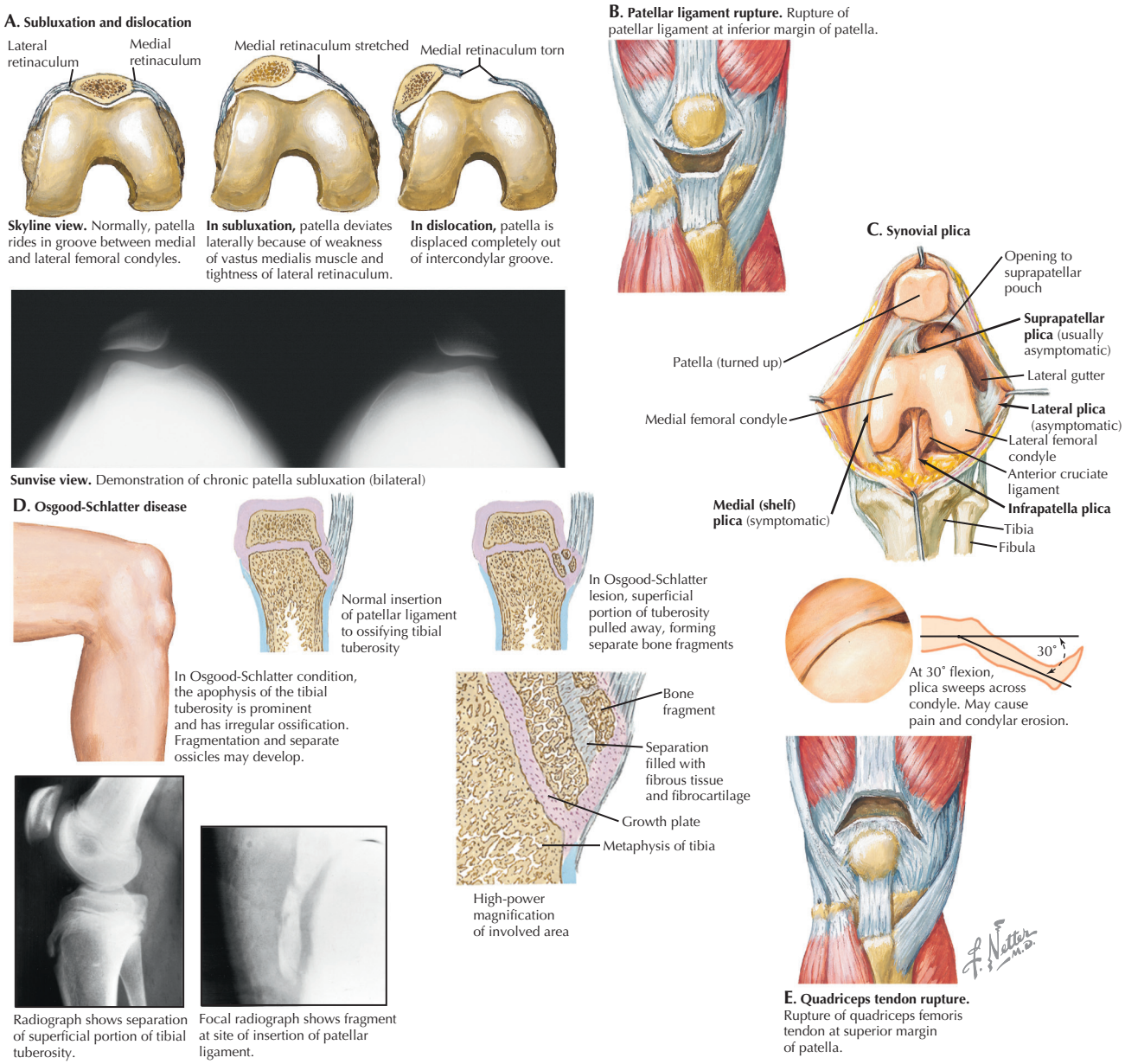



**Figure 55.6.** Extensor mechanism problems. (Sunrise view from Miller M, Cole B. *Textbook of Arthroscopy*. Philadelphia: Elsevier; 2004.)

**Presentation:** May or may not be previous symptoms of instability or patellofemoral pain; feeling of patellar dislocation when injury occurred; report of lying on ground with knee flexed; report of "something coming out" medially, which usually represents uncovered medial femoral condyle rather than the patella going medially; report of "something going back into place" when the knee is extended. Swelling occurs within the initial 2 hours.

**Examination:** Depends on whether the patella is still dislocated or has been reduced; predisposing physical findings seen on the opposite knee; if the patella is still dislocated, will be located over lateral femoral condyle with prominence of uncovered medial femoral condyle (Fig. 55.6); if the patella has been reduced, there may be large hemarthrosis with hypermobility and marked apprehension on hypermobility testing; may also find associated medial ligamentous instability

**Imaging:** Unusual to find the patella still dislocated on radiograph because positioning on the radiograph table usually reduces dislocation; infrapatellar view may show an avulsion of the medial edge of the patella. Large osteochondral fracture may be visible; important to take infrapatellar view with knee flexed only 30–45 degrees, rather than a traditional "sunrise" or "skyline" view with the knee flexed beyond 90 degrees. Patella alta can be objectively measured on lateral view. Lesions of medial supporting structures often visualized on MRI.

**Differential diagnosis:** In acute cases, differentiate from ligamentous tears; in chronic recurrent cases, distinguish from meniscus disorders

**Treatment:** If patella dislocated, knee extension and gentle pressure along the lateral patellar edge usually reduces it easily and without anesthesia. Aspiration may be indicated for comfort or to search for fat in blood secondary to osteochondral fracture. Perceptions about rigid immobilization are changing, even with first-time dislocation, because of harmful effects of immobilization on the knee joint. Immobilize first-time dislocation only as needed for symptoms, followed by an extensive rehabilitation

program and functional patellar bracing. Obvious disruption of VMO insertion into the medial patellar edge or rupture of the medial patellofemoral ligament from the adductor tubercle provides best outcomes with early surgical repair. Treat recurrent dislocation symptomatically with crutches, followed by functional rehabilitation and bracing. Consider surgical realignment of extensor mechanism if there is residual functional disability despite extensive conservative treatment. Surgical realignment is not always successful and should be the last resort.

### Subluxation

**Description:** Transient partial displacement of the patella from the femoral trochlea; may occur acutely, as in patellar dislocation, or may be intermittent. There is spontaneous reduction of displacement.
**Mechanism of injury:** Same as for patellar dislocation; may occur with less severe force or in normal everyday activity
**Predisposing factors:** Same as for patellar dislocation
**Presentation:** Patient may or may not have a history of complete dislocation or patellofemoral pain. Feeling of slipping when cutting, twisting, or pivoting; mild recurrent swelling
**Examination:** Predisposing physical findings seen in both knees but may be more obvious on asymptomatic side, particularly if there has been an acute injury on the symptomatic side; mild effusion; positive hypermobility and apprehension test
**Imaging:** Infrapatellar radiograph view must be performed with appropriate technique and knee flexed only 30–45 degrees; may show lateral tilt and/or lateral subluxation or be normal in appearance. Patellofemoral indices (Merchant, Laurin, and Brattstrom) show a tendency toward patellofemoral problems but do not give specific diagnosis (see Fig. 55.6).
**Differential diagnosis:** Chronic knee ligament instability, causing giving way of the knee
**Treatment:** For acute subluxation, use temporary symptomatic immobilization, followed by functional rehabilitation and bracing of the patella; if no acute episode, treat with functional rehabilitation, bracing, and NSAIDs. Patients disabled by subluxation may require arthroscopic lateral release or open extensor mechanism reconstruction. Surgical treatment has less than perfect results and should be considered as the last resort (see Fig. 55.6).

## Painful Syndromes

### Patellofemoral Pain Syndrome

**Description:** Various syndromes characterized by anterior knee pain as major symptom; an imprecise term wherein pain is not explained by a more readily definable cause; often called "chondromalacia patella," a term that should be reserved for articular cartilage damage actually observed
**Mechanism of injury:** May result from extensor mechanism malalignment, with or without an instability syndrome; may occur as overuse injury with extreme and/or repetitive loading of patellofemoral joint (e.g., knee flexion, running, and jumping)
**Predisposing factors:** Same as for instability syndromes
**Presentation:** Anterior knee pain, often worse with sitting in tight space with knee flexed and on descending stairs or slopes; mild swelling (may be bilateral); may be snapping and popping around patella
**Examination:** Findings predisposing to extensor mechanism problems in both legs; pain on patellofemoral compression test; crepitation about patella on range of motion; tenderness to palpation around patella. Mild effusion may be present. Foot malalignment or leg length inequality may aggravate the symptoms.
**Imaging:** Same as for subluxation; may be normal
**Differential diagnosis:** In preadolescents and young adolescents, consider referred pain from hip disorder (e.g., Legg–Calvé–Perthes disease and slipped capital femoral epiphysis), osteochondritis dissecans of femur or patella, bone tumor—particularly in case of unilateral symptoms. In older patients, osteoarthritis or some other inflammatory joint disease.
**Treatment:** Functional rehabilitation program, NSAIDs, functional bracing of the patella, orthotics for foot malalignment; if other treatments are unsuccessful, surgical treatment may be considered—either lateral release or extensor mechanism reconstruction. Because results are unpredictable, surgery should be considered as the last resort. Always look for other more specific causes of anterior knee pain.

### Patellar Tendinitis ("Jumper's Knee")

**Description:** Inflammation of the patellar tendon, usually at its attachment to the inferior pole of the patella. Rupture of the patellar tendon may occur with or without a history of tendinopathy (see Fig. 55.6).
**Mechanism of injury:** Usually excessive jumping or bounding activity or other high patellofemoral stress activity; less commonly from running
**Predisposing factors:** Same as for other extensor mechanism disorders; possibly ankle dorsiflexor muscle weakness, perhaps secondary to ankle injury
**Presentation:** Activity, such as jumping sport, typically associated with this problem; complaint of infrapatellar pain, originally after exercise, later during exercise and while at rest; rupture occurs with forceful knee flexion against resistance
**Examination:** Tenderness at inferior pole of the patella; less commonly, tenderness over body of the patellar tendon. Other findings of extensor mechanism malalignment; weakness of ankle dorsiflexors; hamstring, heel cord, and/or quadriceps muscle tightness; patellar tendon incongruity and significant knee extension weakness noted
**Imaging:** Radiographs occasionally show irregularity at inferior pole of the patella; may show extensor mechanism malalignment, including patella alta (particularly with rupture). MRI may demonstrate degenerative changes in tendon, which are often read as a partial tear of the patellar tendon by the radiologist but are, in fact, representative of changes in the tendon consistent with patellar tendinosis (see Fig. 55.6).
**Differential diagnosis:** Usually firm diagnosis not difficult with this entity; may consider some other soft tissue lesion of the patellar tendon or fat pad, such as tumor; otherwise, could be any of other causes of patellofemoral pain
**Treatment:** Rehabilitative exercise program, concentrating on hamstring, heel cord, and quadriceps flexibility, as well as quadriceps strength; eccentric strengthening exercises for ankle dorsiflexors are important; anti-inflammatory medication; ultrasound, using hydrocortisone phonophoresis; questionable benefit from infrapatellar strap; more invasive but relatively safe measures used to treat recalcitrant patellar tendinosis include prolotherapy (injection at multiple points in the tendon with "sugar water") and injection of platelet-rich plasma gel. Surgical treatment should be the last resort because of unpredictable results. Immediate surgical repair is indicated with rupture.

### Synovial Plica

**Description:** Structurally, remnant of embryological walls that divide knee into medial, lateral, and suprapatellar pouches; appears as a fold of synovium attached to the joint periphery and to the underside of the quadriceps tendon (suprapatellar plica); may also present as a free edge along medial patellofemoral joint (medial plica) or may be in both locations; rarely seen in other configurations; edge protruding into joint may be of various sizes (see Fig. 55.6)
**Mechanism of injury:** Overuse with repetitive flexion and extension (e.g., running); direct blow to medial patellofemoral joint (e.g., falling on turf or dashboard injury)

**Predisposing factors:** Congenital presence of plica; other extensor mechanism malalignment predispositions may increase the likelihood of symptoms because of plica
**Presentation:** Complaints of anterior knee pain, pain over suprapatellar or medial peripatellar regions with long periods of knee flexion (particularly when accompanied by distinct snap or pop when knee is extended) and painful catching episodes over the medial patellofemoral joint
**Examination:** Often difficult to palpate plica; best performed with passive flexion and extension with tibia held internally rotated. Fingers should lie over the medial patellofemoral joint; may see other extensor mechanism malalignment stigmata; heel cord tightness and hamstring tightness aggravate significantly.
**Imaging:** Not helpful
**Differential diagnosis:** Other painful patellofemoral conditions; possibly medial meniscus injury or loose body; patients often dismissed as "neurotic" because of lack of findings in the face of significant symptoms.
**Treatment:** If inflammatory process is not reversible and plica is fibrotic, persistent symptoms require arthroscopic removal of plica; promises good relief of symptoms and good future functioning. If the inflammatory process in the synovium is still reversible:
- The condition may improve with hamstring stretching, heel cord stretching, or VMO exercises (if VMO is dysplastic)
- NSAIDs, ice, activity modification
- Simple external patellar support may help
- Phonophoresis and/or a corticosteroid injection to plica area may also be beneficial.

### Osgood–Schlatter Disease

**Description:** Painful enlargement of the tibial tuberosity at the patellar tendon insertion. Rather than being a disease, the condition is caused by mechanical stress and excessive tension on growing tibial tuberosity apophysis; occurs in preadolescence and early adolescence, usually during a rapid growth period (see Fig. 55.6).
**Mechanism of injury:** Overuse in normal childhood activities, including sports; rarely, acute onset of popping and pain over tibial tuberosity
**Predisposing factors:** Patella alta, other evidence of extensor mechanism malalignment and altered extensor mechanics; tight hamstrings, heel cords, and quadriceps muscles predispose to symptoms
**Presentation:** Complaints of painful enlargement of tibial tuberosity
**Examination:** Enlarged, tender tibial tuberosity; stigmata of extensor mechanism malalignment, particularly patella alta; tight hamstrings, heel cords, and quadriceps muscles
**Imaging:** Enlarged tibial tuberosity, irregularity of tibial tuberosity, loose ossicle separated from the tuberosity, and patella alta shown in radiographs
**Differential diagnosis:** Other forms of patellar tendinitis; in acute episodes, avulsion fracture of tibial tuberosity; tumorous processes of tibial tuberosity
**Treatment:** Hamstring stretching, heel cord stretching, and quadriceps stretching exercises; VMO-strengthening exercises; activity modification as necessitated by symptoms; simple modalities; local padding

### Quadriceps Tendinitis (including VL Tendinitis and VMO Tendinitis) and Rupture

**Description:** Inflammation of the quadriceps tendon at its insertion into superior edge of the patella; may involve only VL insertion into superolateral pole of the patella or VMO insertion into superomedial pole of the patella. Rupture of the quadriceps mechanism may occur with or without a history of tendonitis following forceful knee flexion against resistance (see Fig. 55.6).
**Mechanism of injury:** Same as for patellar tendinitis
**Predisposing factors:** Extensor mechanism malalignment
**Presentation:** Complaints of suprapatellar pain
**Examination:** Tenderness at superior pole of the patella; may be over central rectus femoris insertion, superolateral VL insertion, or superomedial VMO insertion. Other findings of extensor mechanism malalignment; hamstring, heel cord, and quadriceps muscle tightness; quadriceps tendon defect and inability to extend knee present with rupture
**Imaging:** Usually there are no radiographic findings with tendonitis but may observe patella baja with rupture
**Differential diagnosis:** Suprapatellar pain from synovial plica, bone tumor of distal femur
**Treatment:** Same as for patellar tendonitis; immediate surgical repair for rupture

## MISCELLANEOUS KNEE CONDITIONS

### Bursitis

**Description:** Inflammation of any of various bursae around knee, evidenced by swelling and/or pain; typically prepatellar bursa, pes anserinus bursa, tibial collateral ligament bursa, and deep infrapatellar bursa (Fig. 55.7)
**Mechanism of injury:** Usually overuse; may be due to direct blow with bleeding into bursa
**Predisposing factors:** For pes anserinus bursitis, tight hamstrings seem to predispose
**Presentation:** Complaints of swelling (if prepatellar bursa), pain in the prepatellar region (for prepatellar bursitis), pain in the distal patellar tendon region (for deep infrapatellar bursitis), pain in the proximal medial tibia (for pes anserinus bursitis), or pain over the medial joint line (for tibial collateral ligament bursitis)
**Examination:** For prepatellar bursa, check for localized swelling and tenderness; for others, tenderness over described areas
**Imaging:** Not helpful for diagnosis
**Differential diagnosis:** For deep infrapatellar bursitis, other causes of patellar tendon pain; for tibial collateral ligament bursitis, medial meniscus tear; for prepatellar bursitis, usually no differential; for pes anserinus bursitis, pain from pes anserinus tendons, tumors, other causes of proximal medial tibial pain
**Treatment:**
- Acute prepatellar bursitis: ice, compression, possible aspiration, and padding
- Chronic prepatellar bursitis: NSAIDs, compression, hamstring stretching, ultrasound, possible aspiration, and corticosteroid injections
- Pes anserinus bursitis: hamstring stretching, ultrasound, NSAIDs, and corticosteroid injections
- Tibial collateral ligament bursitis: injection, both as diagnostic test and as treatment
- Deep infrapatellar bursitis: hamstring stretching, possible injection behind the patellar tendon

### Other Tendonitis

**Description:** Inflammation of any other tendinous structures about knee, typically semimembranosus, popliteus, or biceps femoris tendons; inflammation of the gastrocnemius tendon is rare.
**Mechanism of injury:** Usually overuse; much less commonly, single episode of strain. Popliteus tendinitis is usually a running injury
**Predisposing factors:** For semimembranosus, pes anserinus, or biceps femoris tendinitis, and hamstring tightness predisposes

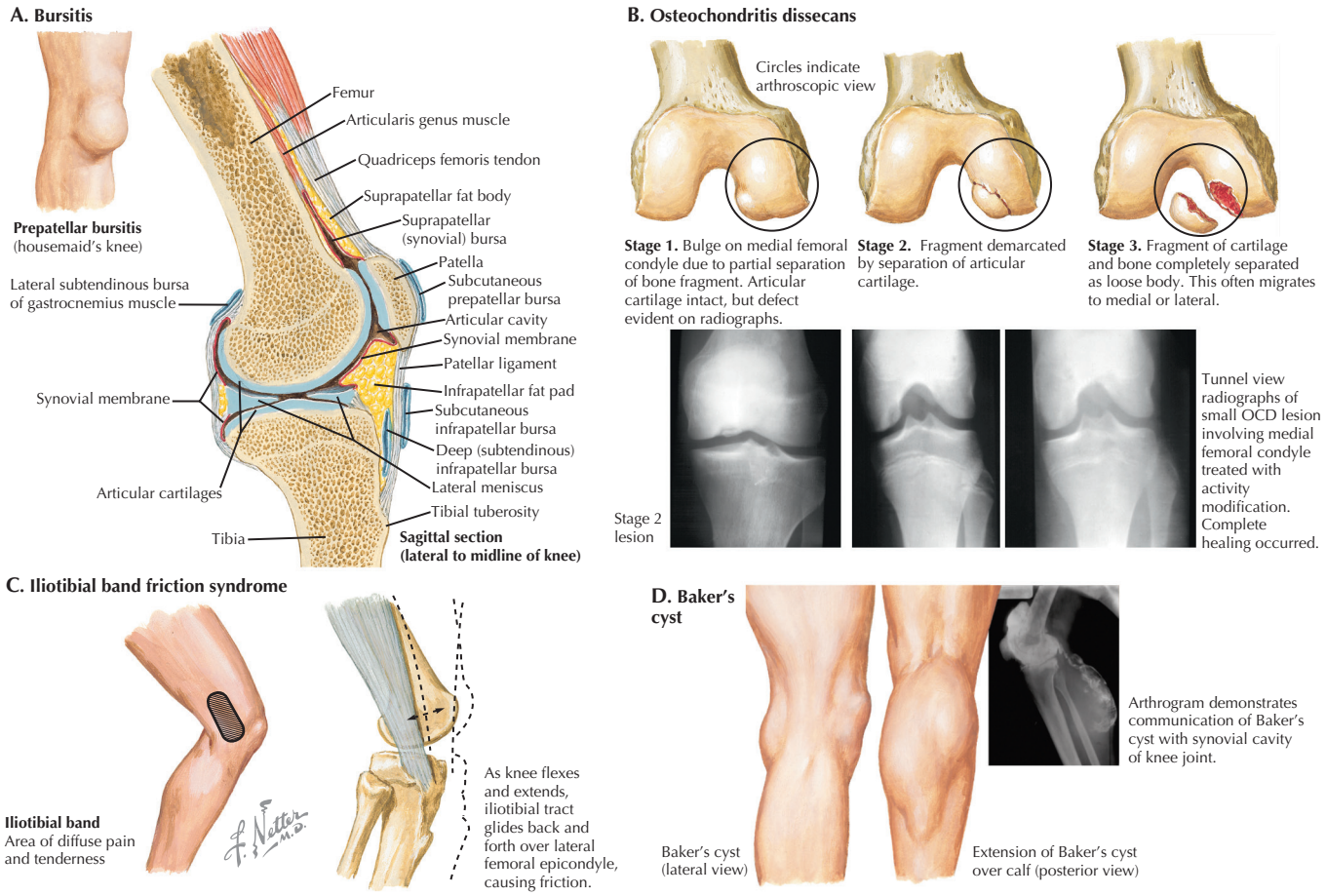



**Figure 55.7.** Miscellaneous knee conditions.

**Presentation:** Complaints of pain over appropriate tendon area; for popliteus tendinitis, lateral knee pain, particularly while running downhill
**Examination:** Tenderness over appropriate tendon; tight hamstrings; for popliteus tendinitis, painful resisted internal rotation of tibia with knee flexed; for all, initially pain on stretching tendon, later pain on active contraction of tendon
**Imaging:** Usually not helpful
**Differential diagnosis:** Hamstring tendinitis occasionally to be differentiated from sciatica; semimembranosus tendinitis may be confused with medial meniscus disorders; for popliteus tendinitis, lateral meniscus injury, and iliotibial band friction syndrome
**Treatment:** Usual anti-inflammatory methods; hamstring stretching exercises; patient may require corticosteroid injections. Immobilization is inappropriate except in the acute phase because of adverse effects on collagen tissue and muscle atrophy

## Neuromas

**Description:** Nonneoplastic enlargement of nerve, usually from direct trauma; typically involves various portions of saphenous nerve around knee
**Mechanism of injury:** Direct blow, previous surgery
**Predisposing factors:** Previous surgical incisions
**Presentation:** Pain, particularly nerve-like quality (i.e., paresthesias, burning, and other alterations of sensation)
**Examination:** Tenderness over neuroma, positive Tinel's sign, objective changes in sensation in appropriate distribution
**Imaging:** Not helpful
**Differential diagnosis:** More central sources of nerve compression
**Treatment:** Injection, surgical excision

## Loose Bodies ("Joint Mouse," Chondral Fracture, Osteochondral Fracture, Osteochondritis Dissecans)

**Description:** Cartilaginous or osteocartilaginous fragments usually free-floating within knee joint (though may be attached to synovium more or less firmly) (see Fig. 55.7)
**Mechanism of injury:** Dislocation of the patella (see Instability Syndromes), other trauma to joint surface; may be result of preexisting osteochondritis dissecans; rarely due to synovial osteochondromatosis
**Predisposing factors:** Predisposition to patellar dislocation, preexisting osteochondritis dissecans
**Presentation:** Consistent with previous patellar instability, twisting or direct blow injury, locking episodes, subcutaneous mass that comes and goes and may be felt in various locations around the knee
**Examination:** Patellar findings; may feel movable mass, usually around patellofemoral joint, although it may be at the anteromedial or anterolateral joint line
**Imaging:** In radiographs, purely cartilaginous fragments are not visible; tunnel view may reveal small osteochondritis dessicans (OCD) lesion on lateral aspect of medical femoral condyle; very small bony loose bodies may be obscured; most loose bodies of significance containing bone are visible; source (e.g., osteochondritis dissecans) may be seen. MRI, tomogram, computed

tomography (CT), or arthrogram may help in delineation (see Fig. 55.7).

**Differential diagnosis:** Meniscal tears as source of locking

**Treatment:** Symptomatic loose bodies require surgical removal, usually arthroscopically; a few loose bodies may require replacement and internal fixation. Patellofemoral instability may require treatment. Osteochondritis dissecans may require other treatment. Large chondral or osteochondral fractures may require surgical debridement of joint surface as well as prolonged and protected weight bearing.

### Cysts (Popliteal Cyst, Popliteal Ganglion, Baker's Cyst, Meniscus Cyst)

**Description:** Fluid-filled lesion about the knee arising usually as extension of synovial space, either into normal bursal structure or into soft tissue surrounding knee (see Fig. 55.7)

**Mechanism of injury:** Normally, no specific injury is involved

**Predisposing factors:** None

**Presentation:** Localized swelling in popliteal space or over meniscus

**Examination:** Cystic swelling in medial popliteal space or over mid–joint line, usually lateral joint line

**Imaging:** Plain films are no help; MRI is very helpful in delineating cysts (see Fig. 55.7)

**Differential diagnosis:** Other tumorous lesions about knee

**Treatment:** Aspiration and injection with corticosteroids not very likely to give permanent cure; surgical excision is usually curative. Presence of cyst is usually secondary to another process in knee that leads to excessive synovial fluid, thus causing a cyst; most likely meniscal tear causing popliteal cyst, or lateral meniscus tear causing lateral meniscus cyst. Underlying disorders must be treated.

### Iliotibial Band Friction Syndrome

**Description:** Chronic inflammatory process involving soft tissues adjacent to lateral femoral epicondyle; presumably caused by chronic "friction" of iliotibial band rubbing over bony prominence of this area (see Fig. 55.7)

**Mechanism of injury:** Overuse; most cases caused by running

**Predisposing factors:** Varus alignment of the knee; running on sloped surfaces

**Presentation:** Lateral knee pain on activity, occasional popping

**Examination:** Tenderness over lateral femoral epicondyle, tight iliotibial band, and absence of intra-articular findings

**Imaging:** Not helpful

**Differential diagnosis:** Other causes of lateral knee pain, particularly popliteus tendinitis; lateral meniscus disorders, lateral patellofemoral joint sources such as VL tendinitis

**Treatment:** Iliotibial band stretching exercises, anti-inflammatory treatment, ultrasound to lateral femoral epicondyle, and corticosteroid injections; rarely, surgery to release area of tightness

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 56 ANKLE AND LEG INJURIES

Christopher Kim • Annunziato Amendola • John E. Femino

## GENERAL PRINCIPLES

- Leg and ankle injuries often occur concomitantly. Evaluation of one must include the other (Fig. 56.1).
- A neurovascular examination is essential as well as making note of the amount of swelling.
- Injuries that require immediate treatment include open fractures, dislocations, neurovascularly compromised extremities, and acute compartment syndrome.
- Ankle sprains are the most common injury of the ankle.
- Chronic pain and disability may result from missed injuries, delayed treatment, or inadequate rehabilitation.

### History

#### Acute Injuries

- Collect the detailed pain history, including onset, quality, severity, location, radiating and associated symptoms, and exacerbating and relieving factors.
- Mechanism of injury will give clues as to the type of injury
- Determine whether there is a history of prior injury or surgery. Prodromal symptoms may suggest stress fracture.
- In open fractures, determine the timing of injury and environment in which the injury occurred to help guide antibiotic use.

#### Chronic Injuries

- Obtain the detailed history of training activities, including frequency, duration, and intensity.
- Collect a detailed pain history.
- Type of shoe worn and use of orthotics should be considered.

### Physical Examination

#### Inspection

- Evaluate the skin for lacerations and bleeding. Bleeding with fat globules suggests an open fracture.
- Evaluate for a deformity that may indicate a displaced fracture or dislocation.
- Note excessive swelling or skin tenting because these may lead to open wounds.

#### Palpation

- Palpation should be systematic, anatomically based, and end with the most painful area.
- Palpation of pulses may be limited because of the location of pain: the posterior tibial artery is palpated behind the medial malleolus, the anterior tibial artery is palpated beneath the extensor hallucis longus or over the first intercuneiform joint at the dorsalis pedis, and peroneal artery pulse is found anteriorly over the syndesmosis (Fig. 56.2A and Fig. 56.2C).
- Neurologic examination should include the deep and superficial peroneal, saphenous, sural, and posterior tibial nerves.
- Palpation of bony prominences should include the tibial crest from the knee to the ankle (see Fig. 56.2B). Check the joints above and below the site of injury. Palpate the medial and lateral malleoli, beginning at the distal tips and moving proximally. Palpate the forefoot and midfoot.
- Palpation of leg compartments should reveal supple compartments; firm compartments may indicate compartment syndrome.
- Palpate the lateral ligaments. Tenderness of the syndesmosis is palpated anteriorly between the tibia and fibula just above the ankle joint line and extending proximally several centimeters. Palpate the medial ankle ligaments, including the superficial and deep deltoid ligaments. The anterior band of the deltoid between the medial malleolus and navicular is superficial and can be directly palpated (see Fig. 56.2D).
- Palpation of the tendons should include the Achilles, posterior tibial tendon, flexor hallucis longus (FHL), and the peroneals. Anterior tibial tendon and digital extensors can be individually palpated but are less frequently injured (see Fig. 56.2C).
- The ankle joint is directly palpated medial to the anterior tibialis over the anteromedial recess and lateral to the peroneus tertius over the anterolateral recess; joint effusion can be detected in these two areas.
- Palpation of the ankle posteriorly on either side of the Achilles tendon may reveal tenderness caused by a fracture or injury of the trigonal process of the talus, os trigonum, FHL tendonitis, or posterior ankle impingement.

#### Range of Motion

- Ankle range of motion is assessed for dorsiflexion and plantarflexion with the knee flexed and extended.
- Limitation of dorsiflexion with the knee extended may indicate contracture of the gastrocnemius.
- Subtalar joint motion can be isolated by stabilizing the ankle joint with one hand on the lateral side of the ankle and calcaneus. The subtalar joint is then inverted and everted, while the tibiotalar joint is fixed.

#### Muscle Testing

- Resisted ankle and toe dorsiflexion and plantarflexion should be tested. Tension in the tendons can be assessed at the same time.
- Weak plantarflexion and loss of resistance to passive ankle dorsiflexion are hallmarks of Achilles tendon rupture. However, patients may demonstrate minor strength by using toe flexor tendons, posterior tibial tendon, or an intact plantaris muscle.
- Resisted inversion with the foot plantar-flexed and everted isolates the posterior tibialis muscle.
- Resisted eversion tests the peroneals, and detection of instability is enhanced with the ankle in dorsiflexion as well; peroneal tendons can be palpated with the opposite hand from behind the ankle.

#### Special Tests

**Anterior drawer test:** Best performed seated with the knee flexed to relax the gastrocnemius muscle; the ankle is plantar-flexed, and the examiner stabilizes the tibia with one hand, while the other hand is placed along the lateral side of the foot with the fingers wrapped around the heel. The ankle is then shucked back and forth gently with emphasis on detecting an anterior translation (Fig. 56.3A). This motion should be combined with internal rotation of the talus because true lateral ankle instability is a rotatory phenomenon. The examination is repeated on the contralateral ankle for comparison.

**Talar tilt test:** With the ankle in less plantarflexion, a varus force is applied to the hindfoot. Internal rotation can enhance the test, which is supposed to detect insufficiency of the calcaneal fibular ligament (CFL) (Fig. 56.3B).

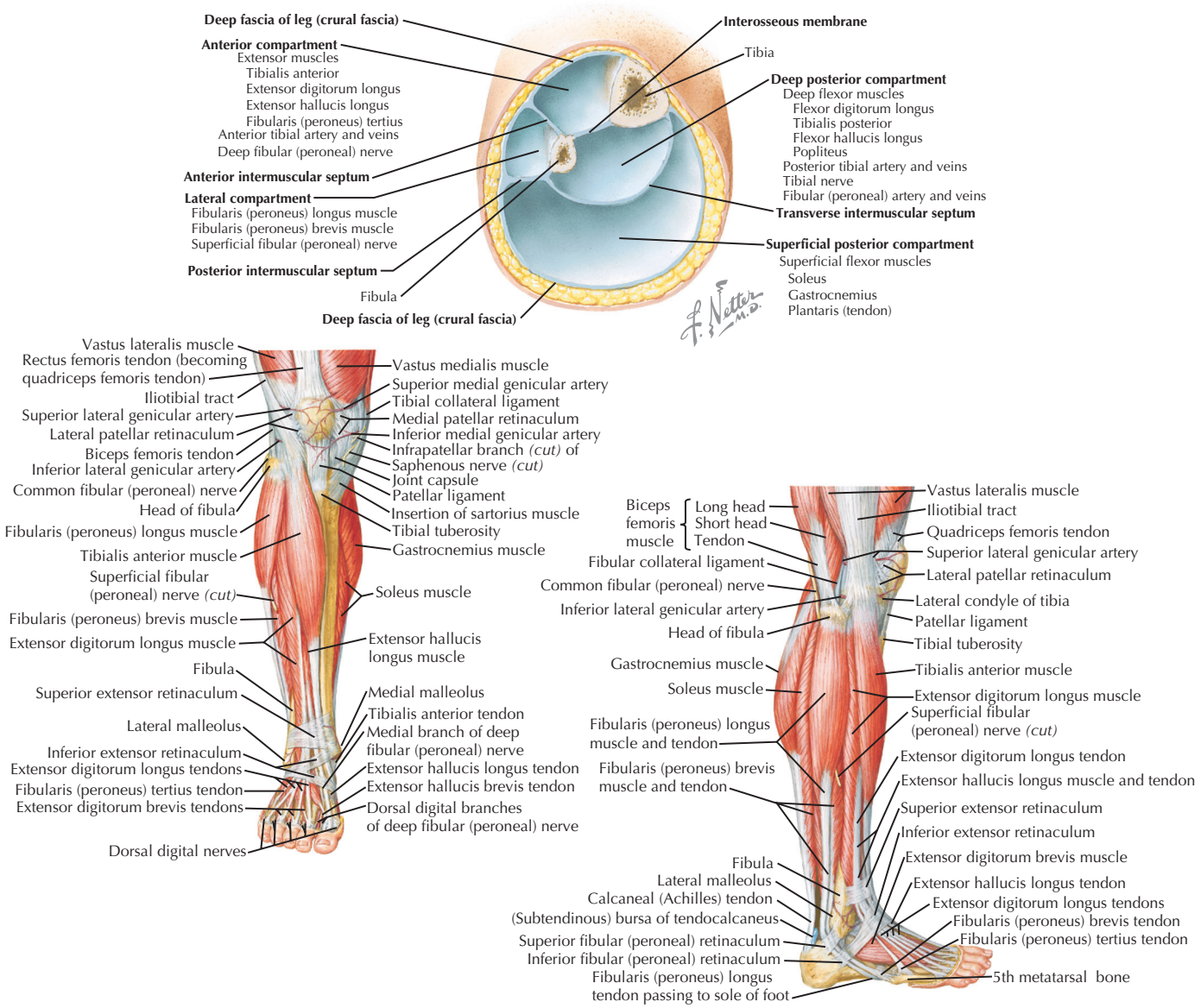



**Figure 56.1.** Compartments of the leg and ankle.

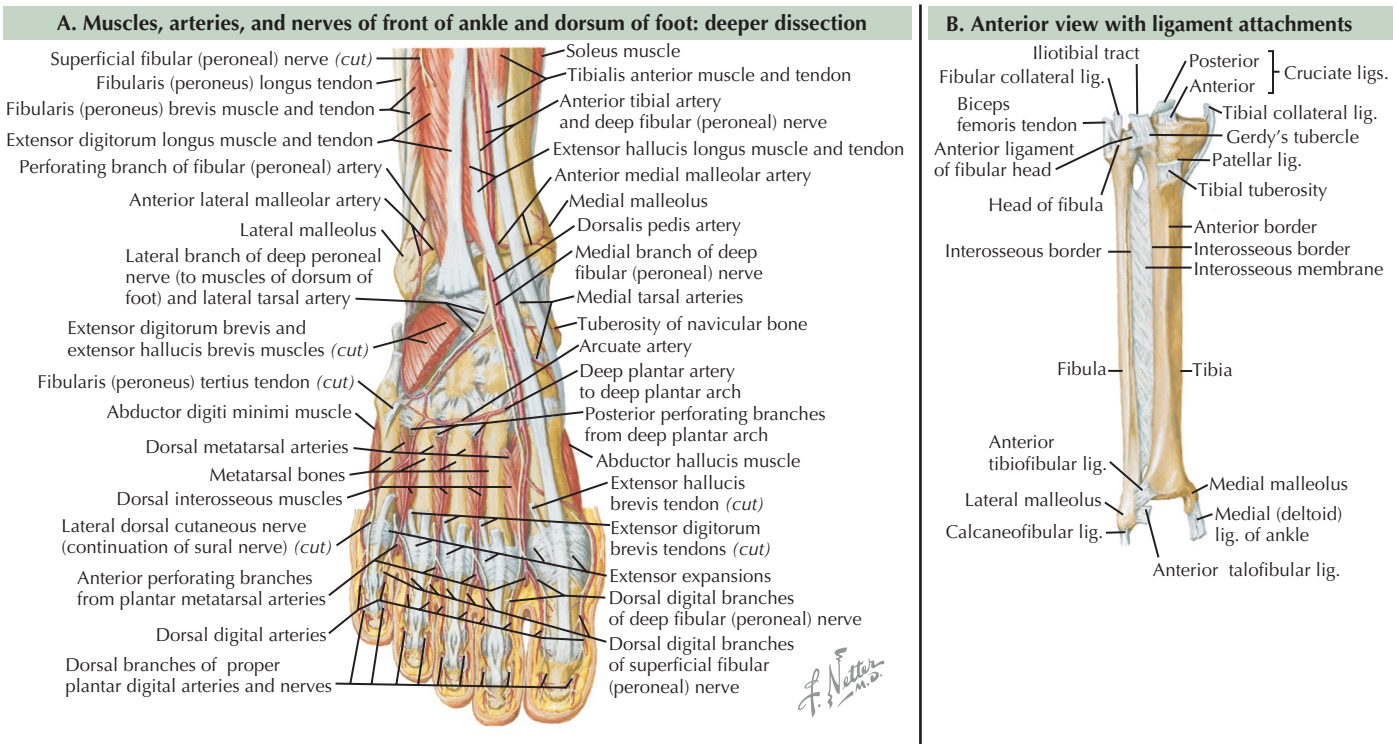
**External rotation stress test:** Can reveal syndesmotic injury; stabilize the tibia and externally rotate the foot and ankle. This is performed with the knee flexed to 90 degrees and the heel locked in neutral. Pain and apprehension may indicate syndesmotic injury. If controlling tibial rotation is difficult, have the patient stand on the involved leg and rotate externally on the foot; this places a more realistic force across the syndesmosis.

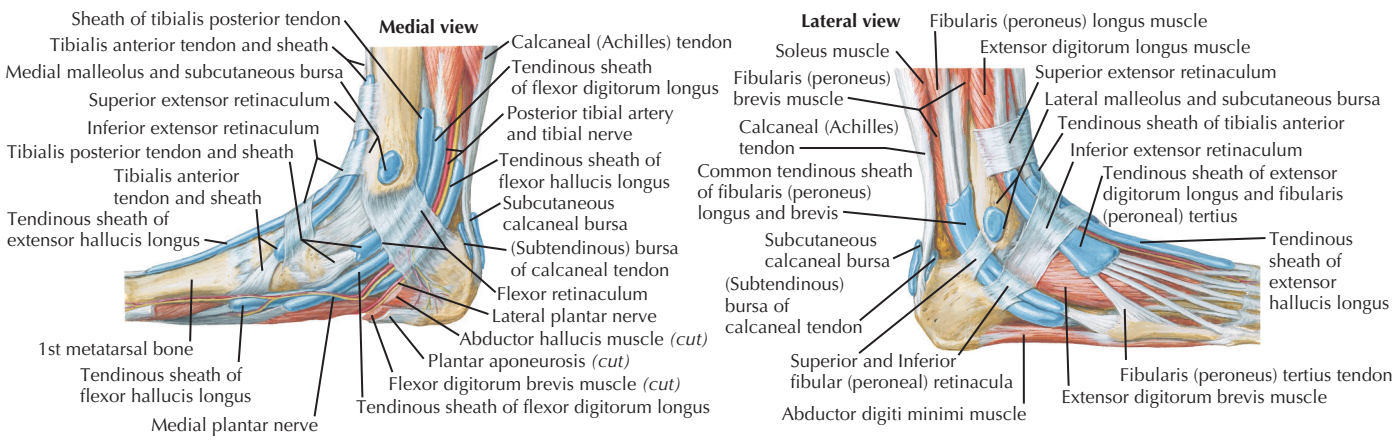
**Impingement testing:** Performed in four locations with the joint in an "open" position and placing mild digital pressure at the point of impingement; the starting position is plantarflexion for anterior impingement and dorsiflexion for posterior impingement. With pressure held, the ankle is then passively moved into a "closed" position, which can then entrap the redundant tissue, reproducing the soft tissue impingement and pain. For bony impingement, the talar osteophyte can often be palpated while moving the ankle in dorsiflexion or plantarflexion with the examiner's finger over the anteromedial or lateral recess of the ankle. This same maneuver can be performed for the subtalar joint with the foot in an inverted, plantarflexed position and moved into a dorsiflexed and everted position.

**Compartment pressure measurement:** Most common measuring devices are solid-state battery powered units that use a side-slit catheter (Fig. 56.3C). Compartment syndrome is a clinical diagnosis. Compartment pressure measurements should be used as adjuncts to a thorough clinical assessment. Measuring compartment pressures are particularly useful in obtunded or unconscious patients in whom an accurate clinical assessment is not possible. An absolute pressure >30 mmHg or a difference between the compartment pressure and a diastolic blood pressure <30 mmHg are very concerning. Hallmarks of compartment syndrome include pain out of proportion to the presenting injury, with particularly excruciating pain with passive stretching of the muscles in the affected compartment. Other features include firm compartments, pallor, paresthesia, paralysis, and loss of pulses. A diagnosis of compartment syndrome warrants emergent surgical release in the form of fasciotomies.

## Radiological Tests

**Plain radiographs:** Should include area of injury, and include the joint above and joint below; a minimum of 2 views should



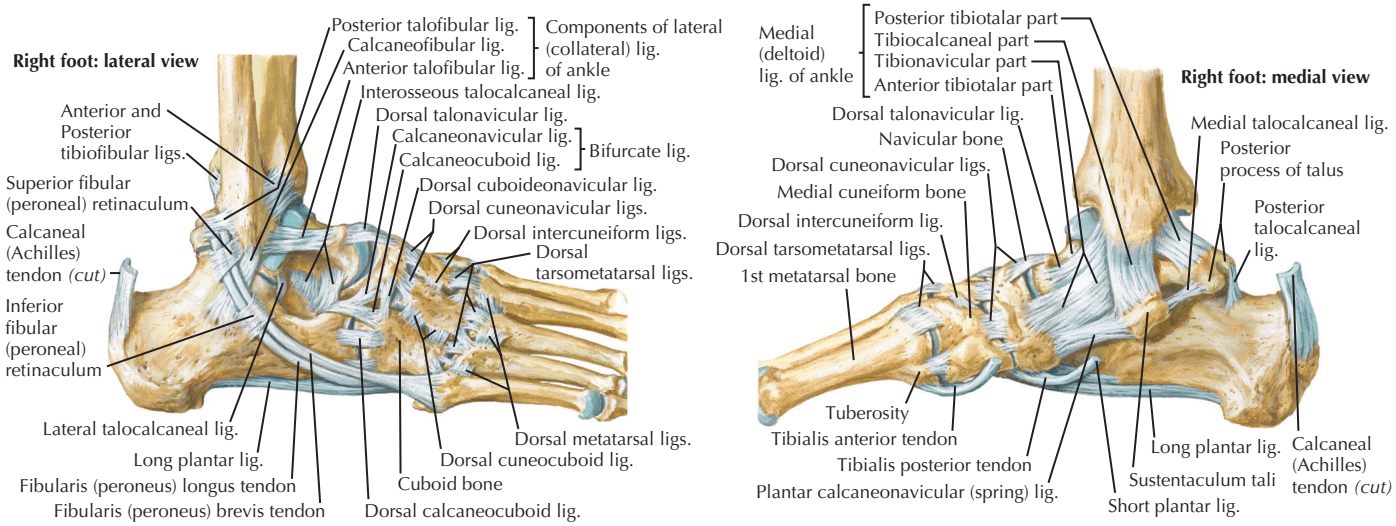




**Figure 56.2.** Anatomy of the leg and ankle.

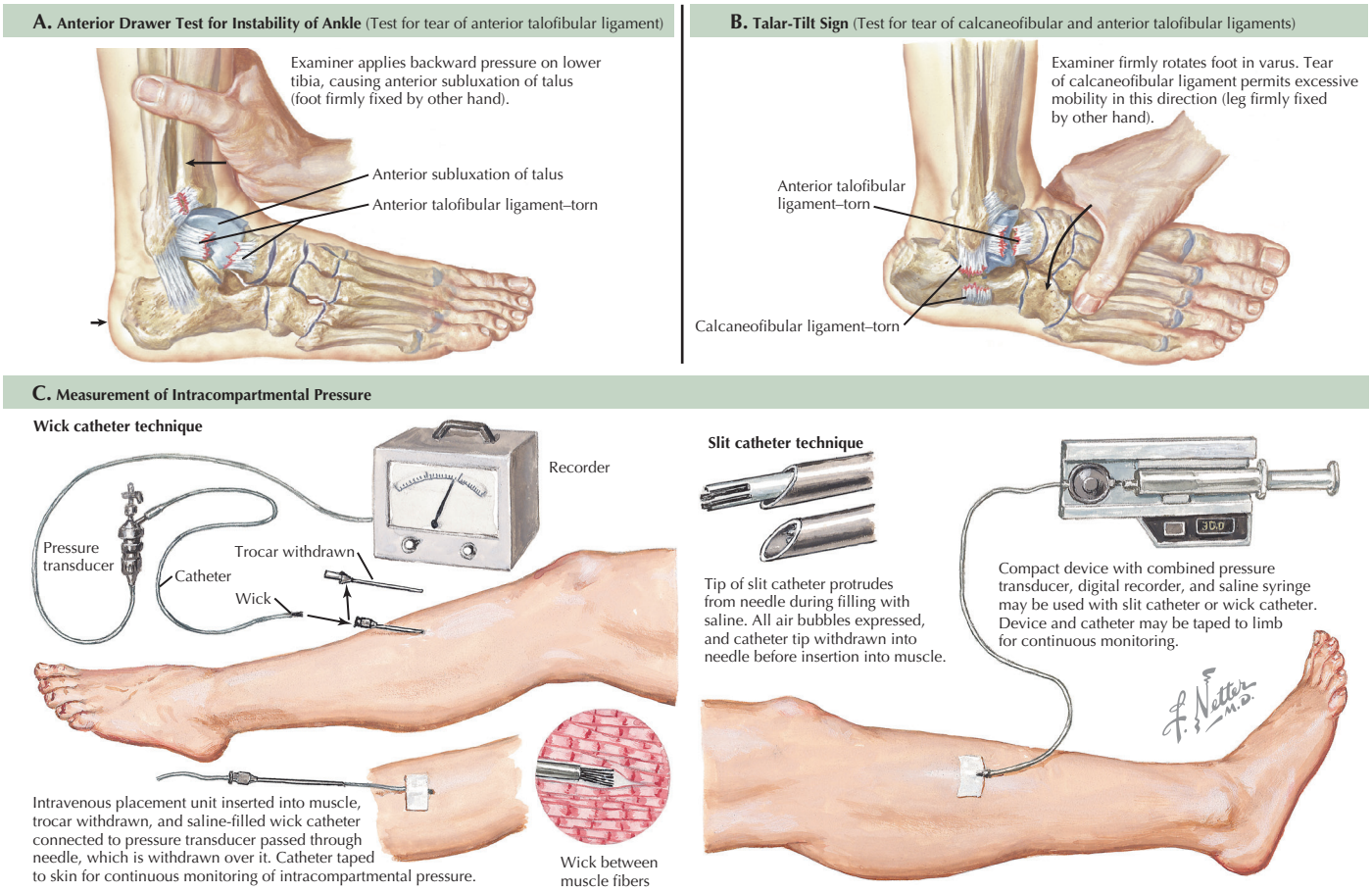



**Figure 56.3.** Special tests.

be performed for each area. Standard views for feet include anteroposterior (AP), lateral, and oblique. Standard views for the ankle include AP, lateral, and mortise (30-degree oblique) views. Radiographs are often performed while patient is bearing weight.

**Stress radiographs:** Helpful to document instability about the ankle, which may often guide treatment decisions; best compared to a normal contralateral part

- Anterior drawer test will show anterior translation of the talus relative to the tibia.
- Talar tilt test will show varus tilting of the talus within the mortise.
- External rotation stress test will show syndesmotic or medial ankle widening.

**Nuclear medicine scans:** Helpful to pinpoint bone pathology that is not seen on radiographs; positive findings often nonspecific, and several experts will prefer MRI before this modality

**Computed tomography (CT) scans:** Often used in the foot and ankle to confirm and better delineate fractures; excellent in showing bony details in 3 dimensions

**Magnetic resonance imaging (MRI):** Excellent visualization of soft tissue structures and bone marrow, making it very good at detecting early stress fractures.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Acute Fractures

#### Tibia Fractures

**Description:** Tibia is often divided into thirds and fractures are described as proximal, middle, or distal. Numerous fracture patterns are seen, including spiral, oblique, transverse, and comminuted fractures. Open fractures are common because there is little soft tissue surrounding the bone in this area (Fig. 56.4A).

**Presentation:** Patients present with pain and deformity and are unable to bear weight. Swelling is common, and it is important to check for open wounds that may suggest open fracture.

**Treatment:** Displaced fractures require provisional reduction and immobilization. Open fractures require immediate irrigation and debridement. Fractures associated with neurovascular compromise require immediate reduction, with urgent surgical treatment if pulses do not return with provisional reduction. Undisplaced or minimally displaced fractures may be treated with cast immobilization. Fractures that remain displaced or with inappropriate alignment require open reduction and internal fixation; should always monitor for compartment syndrome in tibia fractures

#### Fibular Shaft Fractures

**Description:** Often associated with tibia fractures; with weight bearing, the fibula will take up to 20% and the tibia up to 80% of the load; fibular shaft fractures may indicate syndesmotic injury

**Presentation:** Fractures often occur with twisting or a direct blow to the lower leg. Twisting injuries will produce spiral fractures, while direct blows will cause transverse or comminuted fractures.

**Treatment:** A majority of fibular shaft fractures are nonsurgically treated. If associated with other injuries, such as tibial shaft fractures, treatment of the other injury will often be sufficient; must be suspicious and check for associated injuries, such as syndesmotic or ankle injuries

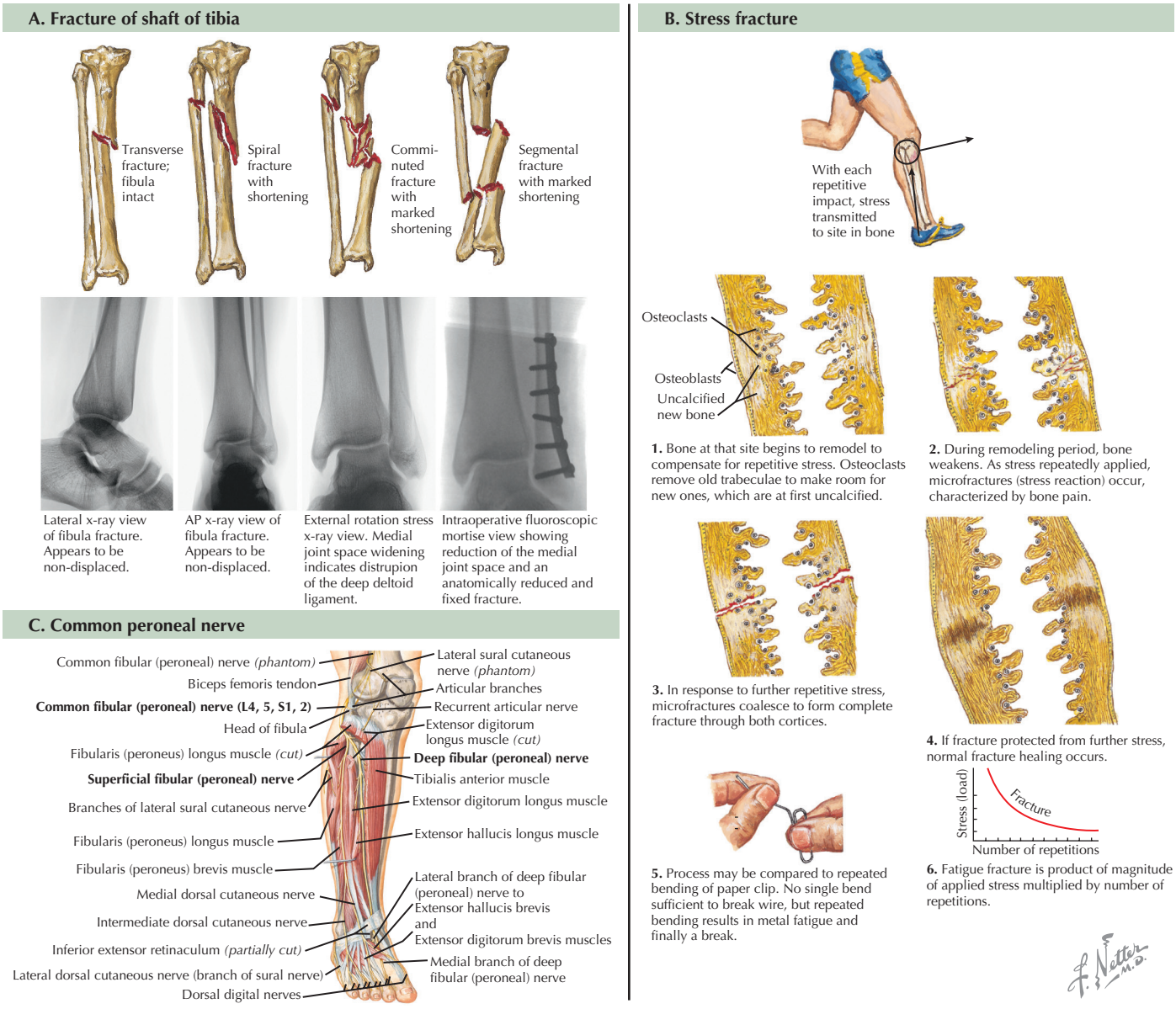



**Figure 56.4.** Conditions of the leg.

## High Fibula Fracture With Syndesmosis Injury

**Description:** Difficult injuries with often significant instability; usually involves injury to the medial ankle such as medial malleolus fracture or deltoid ligament rupture

**Examination:** Crucial to examine the ankle and syndesmosis in the presence of a high fibula fracture; common peroneal nerve traverses at level of proximal fibular neck and should be examined if injury is close to this region

**Imaging:** In the presence of a high fibula fracture, it is very important to also image the ankle to rule out associated injuries. Radiographs may demonstrate subtle widening of the mortise or a medial malleolus fracture. Stress views may be required to rule out syndesmotic injury.

## Ankle Fractures

**Description:** Any fracture that directly enters the ankle or is associated with a ligamentous injury that compromises the ankle mortise

**Treatment:** Dislocated or subluxated ankles require immediate reduction and immobilization. Open fractures require immediate irrigation and debridement. Fractures or dislocations with neurovascular compromise require immediate reduction. Completely undisplaced fractures may be treated nonsurgically using a cast. Syndesmotic injury, disruption of the mortise, or displaced fractures require surgical fixation. Stress-view radiographs may be necessary to rule out mortise instability. Syndesmotic injuries usually require prolonged nonweight-bearing periods after surgery.

# Chronic Exercise-Induced Leg Pain

## Tibial Stress Fractures

**Description:** Commonly seen in athletes who perform repetitive running and jumping such as track athletes (Fig. 56.4B)

**Presentation:** May present with low-level aching pain with early stress fractures, or acute intense pain if fracture progresses; athletes often have period of prodromal symptoms

**Examination:** Tenderness and pain at location of stress fracture

**Imaging:** Begin with radiographs. If not visualized on radiograph and suspicion remains high, an MRI may show bony edema

in early stress reactions or a fracture line in early stress fractures.

**Treatment:** Early stress reactions or fractures can be treated nonsurgically with rest and immobilization. Patients may return to activities as symptoms allow. Repeat imaging may be necessary to assess progress of healing. Stress fractures that have progressed to fully displaced fractures may require surgical fixation. Patient factors should be optimized to promote early healing, such as adequate sugar control in diabetes or smoking cessation.

### Medial Tibial Stress Syndrome

**Description:** Common cause of exercise-induced leg pain in athletes; seen in sports wherein repetitive running and jumping are required; considered a periostitis caused by traction of posterior leg muscles

**Presentation:** Pain along the posteromedial border of the mid tibia, which worsens with activity; pain improves with rest but often does not completely resolve

**Imaging:** MRI is the best confirmatory test. Characteristic MRI finding is linear longitudinal edema of the periosteum; this can clearly differentiate medial tibial stress syndrome from a tibial stress fracture, which would show marrow edema and a transverse line of signal change within the bone.

**Treatment:** Initial treatment is rest and evaluation of training methods. If no response, surgery may be considered. Results have been mixed. Most common surgical procedure is fasciotomy adjacent to the area of pain, but this should be reserved for the most recalcitrant cases.

### Chronic Exertional Compartment Syndrome (CECS)

**Description:** Activity-related increase in lower leg intracompartmental pressures leading to ischemic-like pain; symptoms slowly improve with rest but may remain quite bothersome to patients

**Examination:** Compartment pressure measurements before and after exercise can be used for diagnosis. A pre-exercise resting pressure >15 mmHg, a 1-minute postexercise pressure >30 mmHg, and a 5-minute postexercise pressure >20 mmHg have been suggested as diagnostic thresholds.

**Treatment:** Nonsurgical treatment comprises activity modifications. Fasciotomy is the surgical treatment of choice, which can be performed using open, mini-open, or endoscopic techniques. Surgical risks include damage to the peroneal nerves. Incomplete release is a common cause for persistent symptoms after surgery.

### Peroneal Nerve Entrapment

**Description:** Common peroneal nerve is vulnerable at the lateral knee as it passes around the fibular neck and divides at the intramuscular septum between the anterior and lateral compartments (Fig. 56.4C). Superficial peroneal nerve is most vulnerable at the fascial exit, but it may pass through a fibrous tunnel before exiting to the subcutaneous tissues. The nerve may become tethered or entrapped within this tunnel.

**Presentation:** Symptoms may be vague and poorly defined. Patients may complain of sensory changes, particularly at the dorsum of the foot. They may have weakness in the muscles of the anterior or lateral compartments.

**Examination:** May have tenderness at site of entrapment; symptoms may be reproduced with direct pressure over area of entrapment. Tinel's sign may be positive, particularly after running when symptoms are usually present. In severe cases of common peroneal nerve entrapment, transient foot drop and weakness of the anterior and lateral compartments may be presenting complaints. Nerve conduction and electrodiagnostic studies may help to delineate the location of entrapment.

**Treatment:** Nonsurgical treatment comprises rest and activity modifications. Physical therapy with desensitization therapy may help. Surgical treatment involves nerve release at the areas of entrapment or tethering.

## Tendon Problems Around the Ankle

### Achilles Tendon Rupture

**Description:** Frequently seen in athletes, particularly in males in the 4th decade; usually occurs in a zone approximately 6 cm above the calcaneal insertion, which is considered a hypovascular region of the tendon. Rupture of the tendon probably requires 50% of the tendon to be degenerative.

**Presentation:** Patients may admit to prodromal symptoms before the rupture; usually occurs during push-off during running or a hard lateral movement. Patients may hear a "pop," often described as a sensation of getting hit in the back, before realizing that they are unable to push off with the ankle and that the pain is localized to the heel.

**Examination:** Most reliable finding is loss of resistance to passive dorsiflexion or loss of active plantarflexion. There may be a palpable gap. Bruising is present. Thompson's test is often positive.

**Imaging:** MRI is helpful in preoperative planning to determine the extent of tendon degeneration and the level of rupture.

**Treatment:** Both surgical and nonsurgical treatments can be considered. Nonsurgical treatment initially begins with immobilization in plantarflexion, with progressive dorsiflexion as healing progresses. Early functional rehabilitation has produced excellent results, with re-rupture rates similar to those with surgical treatment. Surgical treatment primarily consists of repairing the ruptured ends of the tendon using sutures. Re-rupture rates are low with surgical repair. Disadvantages of surgical management include wound healing problems, infection, and sural nerve injury. Minimally invasive percutaneous repair tries to avoid these complications and has been increasing in popularity. Early functional rehabilitation has also been achieved after surgical management.

### Achilles Tendinopathy (Noninsertional)

**Description:** Tendinitis is a thickening and inflammation of the peritendinous tissue and is associated with acute pain; tendinosis is a degenerative condition that occurs within the tendon substance with mucoid degeneration, chondroid metaplasia, and fatty degeneration.

**Examination:** Collect the history of exercise and warm-up activity. Physical examination findings typically show thickening of the involved area with tenderness and varying degrees of local inflammation. Pain is enhanced with dorsiflexion and placing tension on the tendon (Fig. 56.5A). Biomechanical evaluation for hypermobile pes planus is important because corrective orthoses and shoe wear to control motion may be beneficial. MRI is the imaging modality of choice to assess inflammation or the extent of tendon degeneration.

**Treatment:** An improved stretching program, night splints, nonsteroidal anti-inflammatory drugs (NSAIDs), ice, rest, and even immobilization for a period of 2–4 weeks can be helpful. Surgical treatment involves debridement of inflammatory and degenerative tissue.

### Posterior Tibial Tendonitis and Tears

**Description:** Uncommon in athletes under 30 years of age; unlike adult-acquired flat foot deformity, numerous cases of acute tendonitis do not involve a flat foot deformity. However, such injuries may indicate the earliest point in the development of this problem. A hypovascular zone of the tendon, behind and below the medial malleolus, is thought to be most susceptible to injury (Fig. 56.5B).

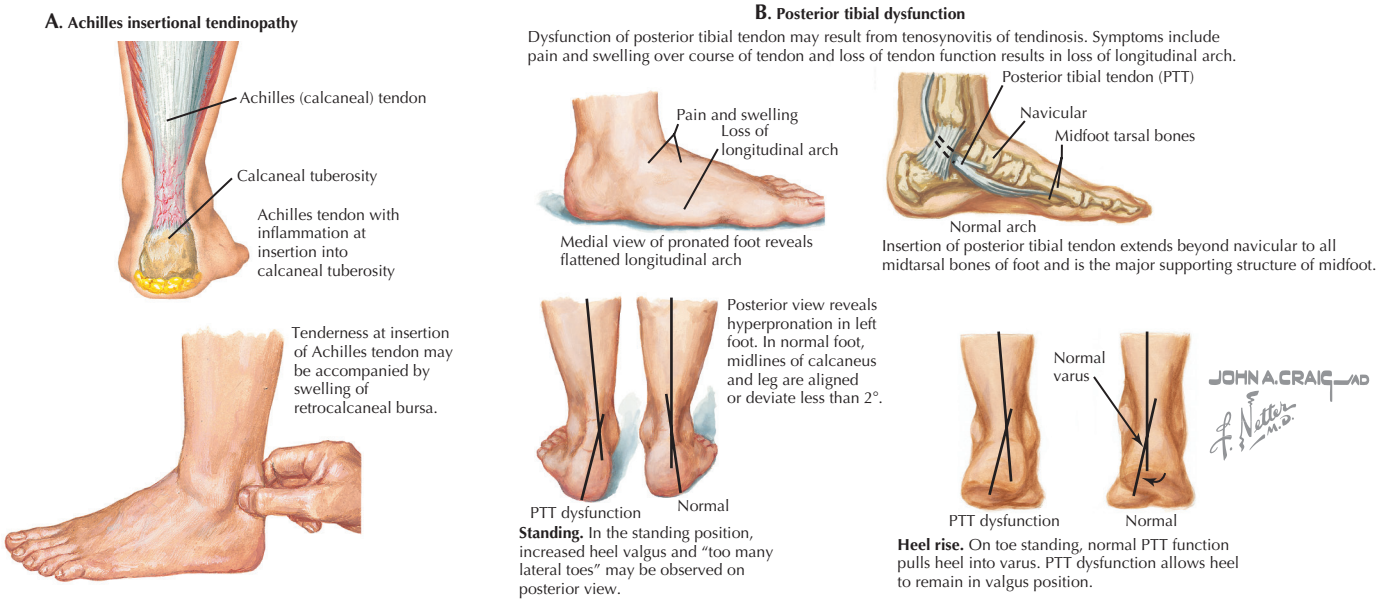



**Figure 56.5.** Tendon problems around the ankle.

**Presentation:** Focal pain with activity on the medial side of the ankle; may be particularly painful during push-off

**Examination:** Weakness, pain against resistance, and tenderness along the course of the tendon are strong diagnostic findings. Focal pain near insertion of the tendon, particularly with bony prominence, may indicate accessory navicular bone.

**Imaging:** Standing radiographs to determine alignment of foot and ankle; will also show accessory navicular. MRI will show status of the tendon and peritendinous tissue.

**Treatment:** Tendonitis can be treated with rest. An orthosis to unload the posterior tibial tendon may be helpful. When pain is too severe, cast immobilization for a period of 2–4 weeks may be necessary. Steroid injections may lead to tendon rupture and should be avoided. Physical therapy can be very helpful. Orthotics to support medial column can also help.

### Flexor Hallucis Longus (FHL) Tendonitis

**Description:** Seen in many athletes, and classically in ballet dancers

**Presentation:** Symptoms may be vague, and located medially or behind the ankle. Pain is usually activity related and may be severe enough to limit further participation in the competition.

**Examination:** Findings may include focal pain over the FHL, including behind the talus or medially between the Achilles and neurovascular bundle. Tenderness is increased with passive dorsiflexion of the hallux as the tendon is brought under tension. Motion of the hallux metatarsophalangeal joint may be decreased with ankle dorsiflexion compared with when the ankle is in plantarflexion.

**Imaging:** Radiographs may reveal a large trigonal process of the posterior talus or an os trigonum. MRI may show edema and fluid around the tendon behind the talus.

**Treatment:** Stretching, rest, night splints, and NSAIDs can lead to resolution of symptoms. Appropriate shoe wear is essential. A period of immobilization may be necessary to decrease any acute inflammation. Surgical release of the tendon sheath and tenolysis may be necessary if the patient fails nonoperative treatments. A symptomatic os trigonum should be considered for excision. Both open and posterior arthroscopic techniques have been described, and the choice of procedure depends on the experience of the surgeon and location of the pathology.

### Peroneal Tendon Problems

**Description:** Include acute and chronic dislocations and tears; often occur with inversion injuries or other trauma such as calcaneal fractures

**Presentation:** Typically, a chronic problem that develops in the setting of other injuries may present as an acute injury. Patients will complain of pain to the lateral ankle as the tendon subluxates or dislocates. They are often able to voluntarily dislocate the tendon.

**Examination:** Cavovarus foot posture may be a risk factor for developing peroneal tendon pathology. Pain in the lateral retromalleolar region that is reproduced with palpation and resisted eversion is typical of peroneal tendon pathology. Testing for transient dislocation or subluxation is essential in evaluation of the integrity of the superficial peroneal retinaculum. This can be performed with the ankle starting in dorsiflexion and inversion, and application of resistance as the patient tries to evert the foot. Often the dislocating tendon can be visualized and felt.

**Imaging:** MRI is the modality of choice for assessing pathology of peroneal tendons. Ultrasound is also helpful because it can be adjusted to the obliquity of the tendons. It is also dynamic and allows direct visualization of the dislocating tendon.

**Treatment:** Rest and brief immobilization may initially help to diminish pain. Orthoses with a lateral forefoot post can help offload the peroneals in a cavus foot. Gross instability or fixed dislocation warrants surgical consideration. Surgery for recalcitrant pain is performed to debride and repair longitudinal tears and, in certain cases, debulk the tendons if tendinosis has led to gross thickening.

## Ankle Joint Problems

### Osteochondral Lesions of the Talus

**Description:** Seen in several ankle injuries, but particularly inversion injuries; medial talar lesions typically have no history of trauma. Lateral talar lesions are typically associated with inversion injuries.

**Presentation:** Present with pain and swelling of the ankle joint; may have mechanical symptoms of clicking, catching, and locking

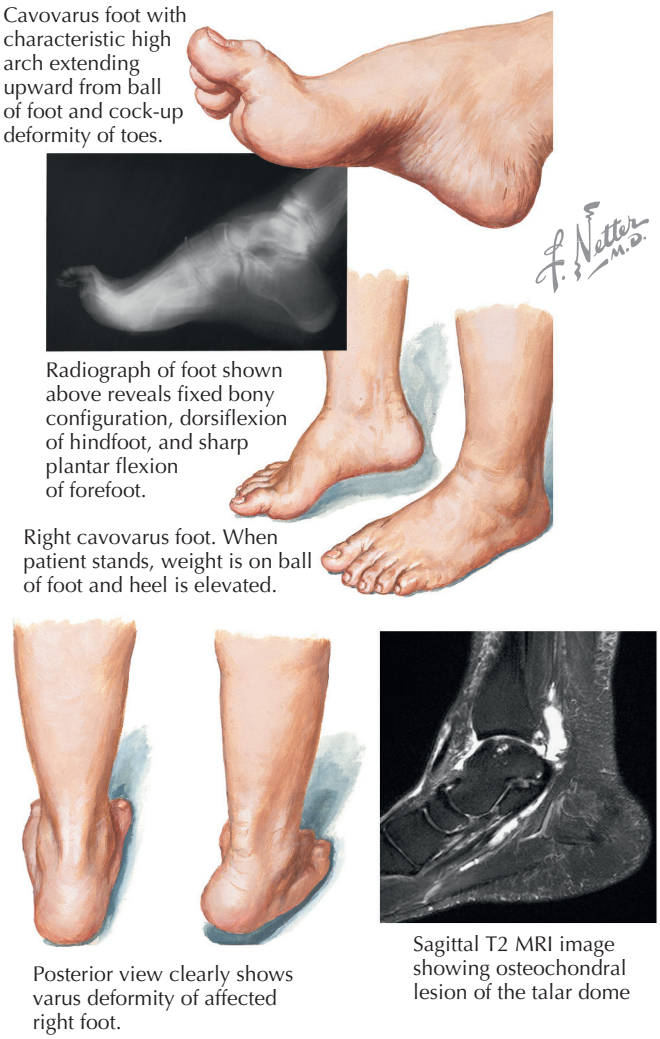



**Figure 56.6.** Ankle joint problems.

**Imaging:** Radiographs are helpful in diagnosing lesions. CT or MRI is preferred to better delineate the lesion and to assess for intra-articular loose bodies (Fig. 56.6). When combined with intra-articular contrast, can show the status of the cartilage; a segment is considered unstable as the dye tracks beneath the osteochondral fragment.

**Treatment:** Osteochondral lesions that are small, stable, and asymptomatic may be nonsurgically treated. Small lesions considered unstable can be excised; larger osteochondral lesions that are displaced may be fixed back into their native positions. Microfracturing can be performed in the presence of a healthy bone bed. Osteochondral autograft and allograft transplantation is also available, particularly in larger lesions or those that have failed previous surgical treatments. Autologous cartilage implantation may also be an option. Surgery is performed using either arthroscopic or open techniques.

## Chronic Lateral Ankle Instability

**Description:** The most common ligament to be injured is the anterior talofibular ligament (ATFL), and the second most common is the CFL. Chronic instability may result owing to incomplete healing; often associated with other ankle pathology, such as talar osteochondral lesions or talar process fractures

**Presentation:** Patients will complain of persistent instability with numerous inversion episodes. Instability may be exacerbated with small events such as stepping on a pebble or walking on uneven ground.

**Examination:** Always compare with the contralateral side. Stress examinations will reveal increased translation or subluxation of the talus.

**Treatment:** Nonsurgical treatment is the mainstay for acute ankle instability. Initial management focuses on controlling pain and swelling. Early functional rehabilitation has shown excellent results. Patients will work on strengthening, range of motion, balance, and proprioception training. Excellent results can be obtained with appropriate physical therapy. An orthosis may be necessary if subtle cavus foot is present and contributing to inversion events (see Fig. 56.6). Failure of nonsurgical treatments with ongoing symptomatic instability is an indication for lateral ligament reconstruction.

## Soft Tissue Impingement

### ANTEROMEDIAL AND ANTEROLATERAL IMPINGEMENT

**Description:** May occur after inversion injuries; anterior impingement caused by tearing of anterior capsule and synovial tissues; lateral impingement caused by torn ATFL or tearing of anterior inferior fascicles of syndesmosis; medial impingement less common, and caused by tearing of anterior band of deltoid ligament; capsule and synovium may also cause impingement at this location

**Examination:** Physical examination can be very specific in reproducing the pain. Patients are often able to reproduce the pain and describe its exact location.

**Imaging:** MRI and arthrograms may show the lesion but are not very specific.

**Treatment:** Usually, arthroscopic debridement is adequate. Open debridement with arthrotomy is rare.

### POSTERIOR ANKLE IMPINGEMENT

**Description:** Uncommon; caused by tearing of the posterior tibiofibular ligament, which forms a labrum-like structure at the posterior tibial plafond

**Mechanism:** Can be torn with ankle trauma; the unstable tissue can flip into the posterior ankle and become entrapped with plantarflexion.

**Examination:** Test for impingement with plantarflexion. Patient is often able to reproduce symptoms.

**Imaging:** MRI findings on the sagittal view can suggest the presence of an impingement lesion if the labral rim is seen to be detached in this view.

**Treatment:** Arthroscopic debridement is possible posteriorly with the patient in the prone position. Open debridement is also appropriate.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 57 CARTILAGE PROBLEMS IN SPORTS

Arthur T. F. Chou • Mark D. Miller

## GENERAL PRINCIPLES

### Articular Cartilage

- Functions to decrease joint friction and distribute load across the joint; also referred to as *hyaline cartilage*
- **Composition:** Water (65%–80%), collagen (10%–20%, predominantly type II), proteoglycans (10%–15%, aggrecan is most responsible for the hydrophilic property), and chondrocytes (5%) (Fig. 57.1)
- **Viability:** Articular cartilage is avascular, and chondrocytes are nourished via diffusion from synovial fluid.
- **Structure:** Organized into three primary layers—superficial, middle, and deep; the tidemark separates these layers from the calcified cartilage and subchondral bone (see Fig. 57.1).
- Location: Articular surfaces, ribs, and nasal septum

### Fibrocartilage

- Functions in direct tendon and ligament insertions and helps in the healing of articular cartilage lesions
- **Composition:** Primary collagen is type I collagen. Fibrocartilage is not as durable as hyaline articular cartilage.
- **Location:** Tendon/ligament junction with bone, menisci, and annulus fibrosis of the intervertebral disc

### Articular Cartilage Injuries

- Healing is enhanced by motion of the involved joint.
- **Deep lesions:** Cross the tidemark and penetrate the subchondral bone; vascularity from the subchondral bone promotes fibrocartilage healing (type I collagen) rather than the preferred articular cartilage.
- **Superficial lesions:** Do not penetrate the subchondral bone and therefore have no intrinsic healing potential secondary to the avascular nature of articular cartilage

### Apophysis

- Cartilaginous prominence adjacent to the physis
- Site of tendon attachments before skeletal maturity
- Secondary ossification centers develop later with eventual osseous fusion.
- **Traction apophysitis:** Repetitive microtrauma caused by the pull of attached tendons; results in partial avulsion and inflammation of the apophysis; common in active children and adolescents; excessive force may result in avulsion fracture of the apophysis.
- **Osteochondrosis:** General term for disorders affecting one or more ossification centers in children; encompasses conditions such as traction apophysitis and avascular necrosis

## HISTORY AND PHYSICAL EXAMINATION

### History

- History should focus on the nature of injury and symptoms of the involved joint.
- Acute injuries typically result in focal chondral or osteochondral injuries, as opposed to the more generalized nature of degenerative lesions.
- These injuries may not be initially identified and are occasionally diagnosed after the persistence of symptoms.
- Chronic symptoms may also be secondary to various osteochondroses.

### Physical Examination

- Few, if any, physical examination tests are specific for the evaluation of articular cartilage injury.
- A complete examination of the involved joint should be conducted.

### Imaging Studies

- Imaging studies are essential for the evaluation of cartilage injuries.
- **Plain radiographs:** Useful in ruling out fractures and identifying various osteochondroses and osteochondral lesions such as osteochondritis dissecans (OCD); also beneficial in identifying intra-articular loose bodies, assessing limb alignment, and evaluating joint space
- **Computed tomography (CT):** Helpful in assessing cartilage lesions with associated osseous involvement
- **Magnetic resonance imaging (MRI):** Gold standard for the evaluation of articular cartilage; can identify subchondral edema; focal chondral defects may be underestimated (Table 57.1). Newer MRI techniques such as delayed gadolinium-enhanced MRI of cartilage can identify proteoglycans, while the measurement of T2 relaxation times are sensitive to collagen architecture.

## SPECIFIC INJURIES AND PROBLEMS

### Hip

#### Focal Chondral Defect

**Description:** Localized, full-thickness loss of articular cartilage with exposed subchondral bone (Fig. 57.2)

**Mechanism of injury:** Typically, a direct blow to the greater trochanter; forces are transferred to the articular surfaces of the femoral head and acetabulum.

**Presentation:** History of injury with failure of full recovery, catching or locking with vague hip and groin pain

**Physical examination:** Nonspecific

**Differential diagnosis:** Avascular necrosis, femoroacetabular impingement (FAI), hip dysplasia, degenerative arthritis, labral pathology, and femoral neck stress fracture

**Diagnostics:** Radiographs are helpful in evaluating joint space and ruling out other conditions. MRI may demonstrate localized defect or subchondral edema. MRI arthrography has higher detection rates.

**Treatment:** Arthroscopic chondroplasty, drilling, or microfracture for localized lesions; excision of unstable or loose fragments to alleviate mechanical symptoms (see Fig. 57.2)

**Prognosis and return to sport:** Return to sport when symptoms allow after debridement or excision of fragments. Chondral reparative procedures such as microfracture require partial weight bearing for 6–8 weeks; early range of motion encouraged

#### Femoroacetabular Impingement (FAI)

**Description:** Abnormal contact between the femoral head–neck junction and acetabulum; results in injury to the articular cartilage and labrum; may be a cause of chronic hip pain in athletes; two types defined based on the primary location of pathology:

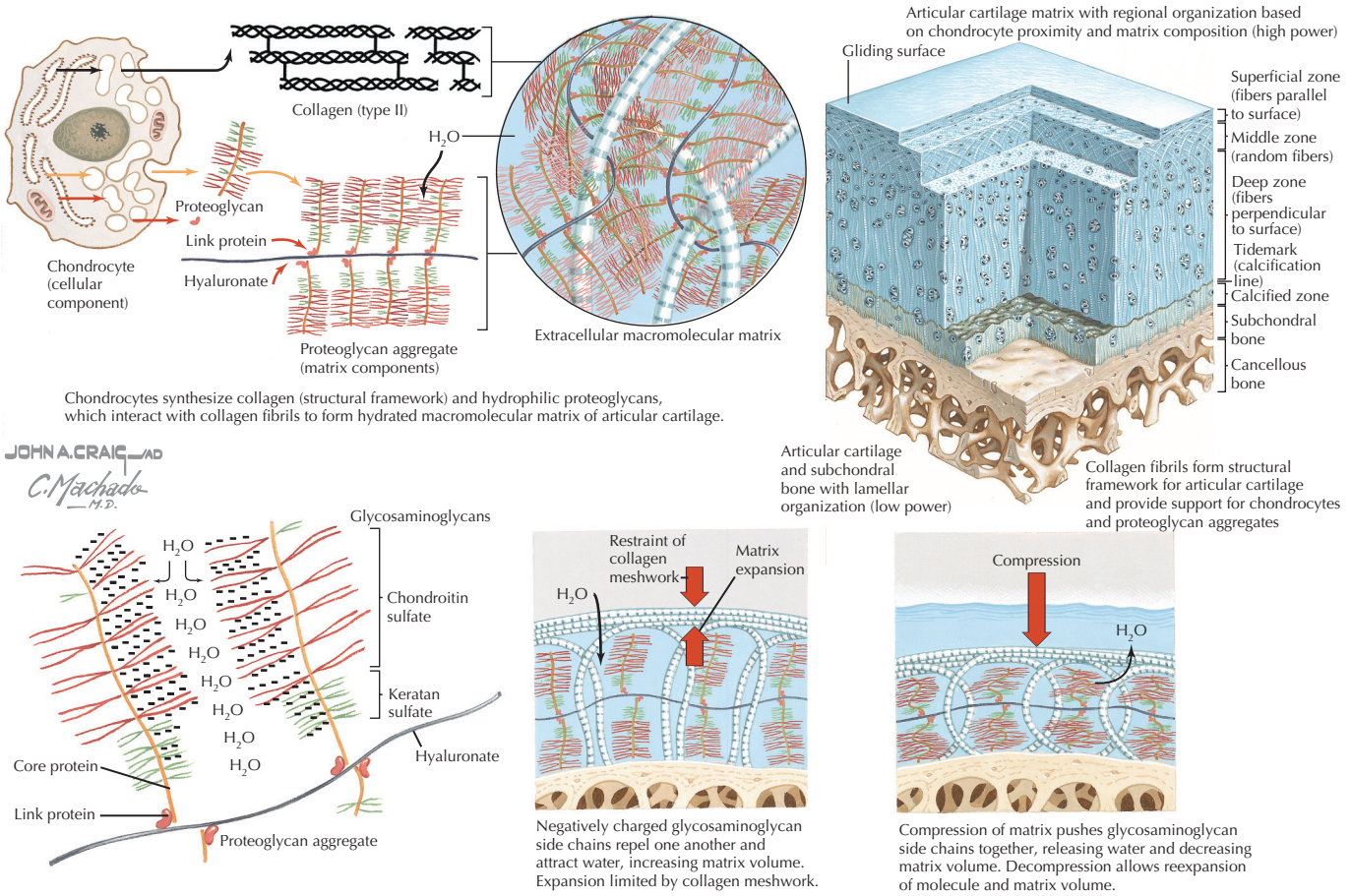



**Figure 57.1.** Composition and structure of articular cartilage.

**TABLE 57.1 CARTILAGE SIGNAL INTENSITIES ON MRI**

| | T1-Weighted Images | T2-Weighted Images |
|---|---|---|
| Hyaline cartilage | Gray | Gray |
| Fibrocartilage | Dark | Dark |

**TABLE 57.2 FACTORS INVOLVED IN FEMOROACETABULAR IMPINGEMENT**

| Type of Factor | |
|---|---|
| Dynamic | A. Decreased offset and asphericity of femoral head–neck ratio (CAM-type lesion)<br>B. Acetabular overcoverage<br>C. Extra-articular impingement |
| Static | A. Undercoverage of hip (dysplastic hip)<br>B. Femoral anteversion<br>C. Femoral valgus |

- **CAM type:** Femoral deformity; "pistol grip" deformity of the femoral head with decreased head–neck offset and asphericity, leading to abutment of this region with the normal acetabulum
- **Pincer type:** Acetabular deformity; increased acetabular retroversion leading to abutment of the normal femoral head–neck junction on the acetabular rim
- **Mixed type:** Combined deformity of femoral head and acetabulum, most common (up to 80%)

**Mechanism of injury:** Etiology unknown; combination of static factors and dynamic factors (see Table 57.2)

**Presentation:** Anterior hip pain with difficulty squatting, cutting, and starting/stopping

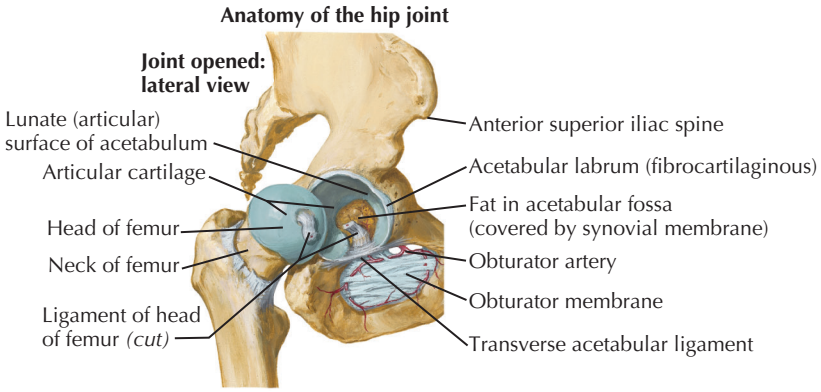
**Physical examination:** Pain with flexion, internal rotation, and adduction indicate a positive impingement test; range of motion often limited

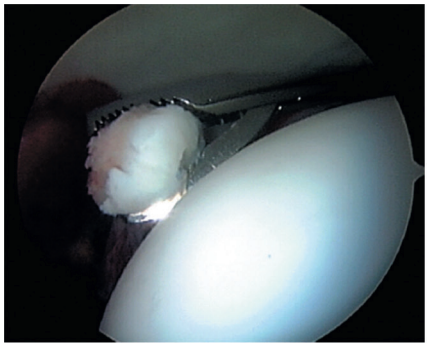
**Differential diagnosis:** Traumatic chondral defect or labral tear and hip dysplasia

**Diagnostics:** Radiographs demonstrate the "pistol grip" deformity of the femoral neck or signs of acetabular retroversion such as a crossover or posterior wall sign. MRI may help identify chondral injury and labral tears.

**Treatment:** Nonsurgical options include activity modification, anti-inflammatory medication, adductor strengthening, and hip-motion exercises. Typical surgical options include arthroscopic osteoplasty for CAM-type lesions and acetabular rim trimming for pincer-type lesions. Chondral lesions are addressed as described previously with chondroplasty, drilling, or microfracture. Labral repair may also be required. Open surgical dislocation and mini-open and arthroscopic repairs all are effective approaches to correct deformity.

**Prognosis and return to sport:** FAI may be a precursor to osteoarthritis. Arthroscopic osteoplasty, rim trimming, and labral repair require partial weight bearing for 4 weeks. When chondral lesions are addressed, partial weight bearing for 6–8 weeks is necessary.



Loose body from chondral injury viewed from the posterolateral portal during hip arthroscopy

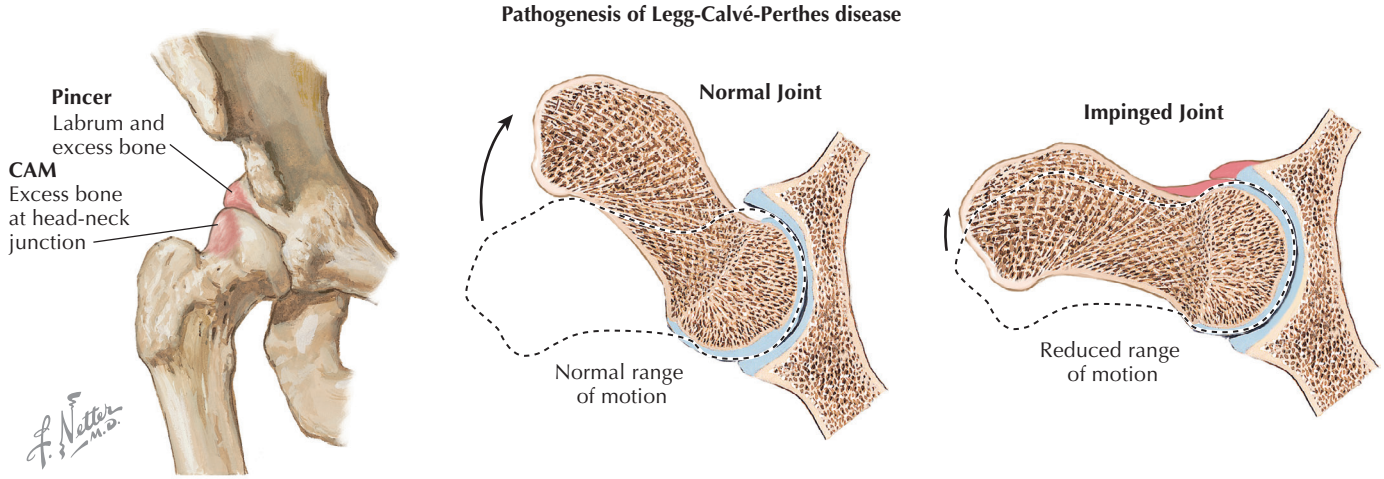



**Figure 57.2.** Hip anatomy and femoroacetabular impingement. (Arthroscope image from Miller M, Cole B. *Textbook of Arthroscopy*. Philadelphia: Saunders, Elsevier; 2004.)

### Apophyseal Injuries

**Description:** Several traction apophyses are present in the hip and pelvis of skeletally immature patients; ischial tuberosity is most commonly involved (Table 57.3).
**Mechanism of injury:** Traction apophysitis secondary to overuse in adolescent patients; common in running sports
**Presentation:** Activity-related pain
**Physical examination:** Localized tenderness, discomfort with range of motion, and tension on the involved musculotendinous unit
**Differential diagnosis:** Apophyseal avulsion fracture, muscle strain, or rupture
**Diagnostics:** Radiographs may show irregularity of the involved apophysis and rule out avulsion fractures.
**Treatment:** Activity modification, local modalities, and anti-inflammatory medications
**Prognosis and return to sport:** Return to sport as symptoms allow

**TABLE 57.3 TRACTION APOPHYSES OF THE HIP**

| Traction Apophysis | Muscle Attachments |
|---|---|
| Iliac crest | Internal and external obliques |
| Anterior superior iliac spine (ASIS) | Sartorius and tensor fascia lata |
| Anterior inferior iliac spine (AIIS) | Rectus femoris |
| Ischial tuberosity | Hamstrings |
| Greater trochanter | Abductors |
| Lesser trochanter | Iliopsoas |

## Knee

### Focal Chondral Defect

**Description:** Localized, full-thickness loss of articular cartilage with exposed subchondral bone (Fig. 57.3AB); medial femoral condyle most commonly involved, followed by lateral femoral condyle, femoral trochlea, and patellar facets; the proximal tibial articular surface is relatively protected by the overlying meniscus and thus less susceptible to injury
**Mechanism of injury:** Generally traumatic injury with shear or rotational forces
**Presentation:** History of injury, possible delayed effusion, pain with persistent weight bearing, and mechanical symptoms
**Physical examination:** Possible effusion and tenderness to palpation
**Differential diagnosis:** Meniscal tear, OCD, and degenerative arthritis
**Diagnostics:** Not visualized on radiographs; MRI best delineates but may underestimate the involvement and size of focal lesions (see Fig. 57.3C)
**Treatment:** Nonsurgical treatment (anti-inflammatory medications, steroid and hyaluronic injections, and physical therapy) often provides limited benefits. A spectrum of arthroscopic options exists when nonsurgical measures fail.
- **Chondroplasty:** Loose chondral flaps debrided with careful preservation of surrounding normal articular cartilage; provides temporary relief at best (see Fig. 57.3D)

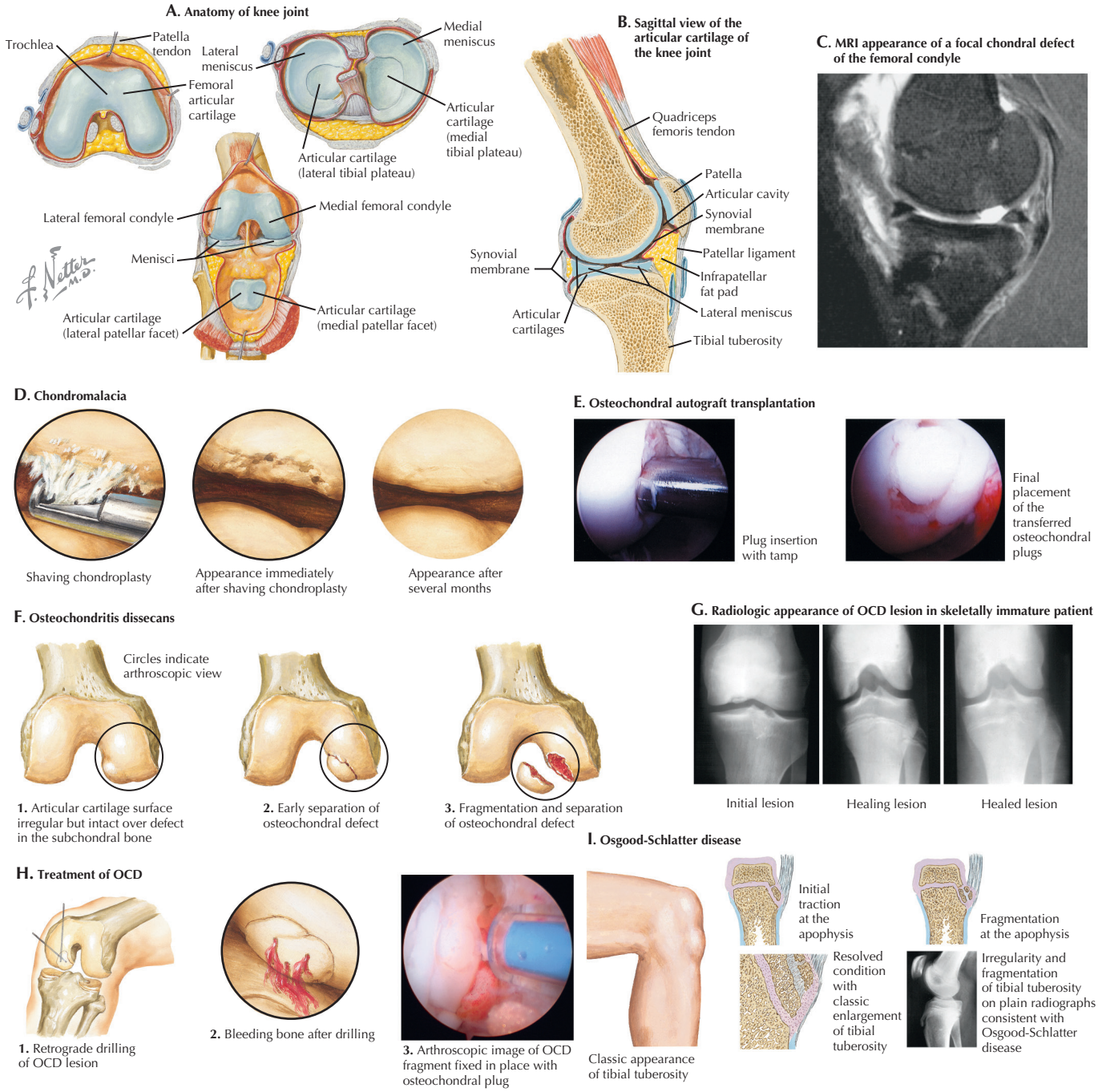



**Figure 57.3.** Knee injuries and problems. (**C** and **E,** from Miller M, Cole B. *Textbook of Arthroscopy*. Philadelphia: Saunders, Elsevier; 2004.)

- **Microfracture:** Considered first line of treatment for small symptomatic lesions; multiple holes created to penetrate subchondral bone to promote bone marrow stimulation; bleeding into the defects promotes influx of pluripotent mesenchymal cells with resultant fibrocartilage formation, which is less desirable than hyaline cartilage
- **Osteochondral autograft transfer (OAT):** Transfer plugs of normal osteochondral tissue from nonweight-bearing regions of the knee into the defect. Rapid healing occurs while articular gaps are filled with fibrous tissue; limited by donor site availability (see Fig. 57.3E)
- **Fresh osteochondral allograft (OCA) transplantation:** Defect filled with a single osteochondral plug from a cadaver; main advantage is a single-stage procedure that allows replacement of a large defect with viable hyaline cartilage; primary concerns include potential disease transmission and immunologic reactions and can be technically demanding
- **Autologous chondrocyte implantation (ACI):** Two-stage procedure; first, chondrocytes are harvested and cultured. After several weeks, the cultured cells are re-implanted under a periosteal patch, which contains pluripotent mesenchymal stem cells. The chondrocytes proliferate and produce hyaline-like cartilage. Newer techniques allow implantation on a scaffold without the need of a periosteal patch.

**Prognosis and return to sport:** No weight bearing for up to 8 weeks; early range of motion encouraged and continuous passive motion may be beneficial; first line of therapy is microfracture, but certain studies have suggested young athletes experience the

best results with restorative procedures (e.g., OAT and ACI); return to sport typically delayed by 3–6 months

### Osteochondritis Dissecans (OCD)

**Description:** Localized separation of subchondral bone and overlying articular cartilage (see Fig. 57.3F); typical location is lateral aspect of the medial femoral condyle
**Mechanism of injury:** Can be caused by repetitive trauma, endocrine disorders, vascular insufficiency, and familial predisposition
**Presentation:** Often vague activity-related pain, possible effusion, and mechanical symptoms if the affected fragment becomes loose within the joint
**Physical examination:** Effusion and tenderness to palpation
**Differential diagnosis:** Meniscal tear, focal chondral defect, and intra-articular loose body of other etiology (e.g., subsequent to patellar dislocation)
**Diagnostics:** A tunnel (notch) view best demonstrates OCD lesions on radiographs (see Fig. 57.3G). MRI is better for delineating size and stability of the lesion. Synovial fluid behind the lesion indicates poor healing potential with nonsurgical management.
**Treatment:** Adolescents with open physes have a much better prognosis, which may be observed with restricted weight bearing. Surgical treatment options recommended for unstable or loose fragments, failure of conservative management, and lesions in skeletally mature patients; consists of debridement, drilling, and internal fixation with addition of bone grafting in select cases (see Fig. 57.3H)
**Prognosis and return to sport:** Prognosis best in patients with open physes; Return to sport when symptoms abate; with surgical fixation of lesions, return to sport often delayed 3–6 months to allow adequate healing of the lesion

### Osgood–Schlatter Disease and Sinding–Larsen–Johansson Syndrome

**Description:** Osteochondroses that occur at traction apophyses in knees of skeletally immature patients; Osgood–Schlatter affects the tibial tuberosity (patellar tendon insertion) and Sinding–Larsen–Johansson the inferior pole of the patella (patellar tendon origin); most common during periods of rapid growth (see Fig. 57.3I)
**Mechanism of injury:** Traction apophysitis secondary to mechanical stress from the extensor mechanism and chronic avulsion of the tibia; overuse syndrome
**Presentation:** Activity-related anterior knee pain, particularly with jumping and kneeling
**Physical examination:** Localized tenderness, prominent tibial tuberosity, and pain with resisted knee extension
**Differential diagnosis:** Patellar tendonitis, tibial tubercle physeal injury, and patellar sleeve fracture with acute injury
**Diagnostics:** Radiographs may show fragmentation or irregularity of the tibial tuberosity or inferior patellar pole and associated soft tissue swelling (see Fig. 57.3I).
**Treatment:** Activity modification, hamstring stretching, local modalities, bracing with compression sleeve or Cho-Pat strap, anti-inflammatory medications; rarely, ossicle excision for re-calcitrant cases
**Prognosis and return to sport:** Self-limiting condition; return to sport as symptoms allow

## Foot and Ankle

### Osteochondral Lesions of the Talus (OLT)

**Description:** Focal osteochondral defect involving the dome of the talus (Fig. 57.4A); anterolateral lesions are typically traumatic, shallow, and more likely to be displaced. Posteromedial lesions are more common, usually deeper, atraumatic, and nondisplaced.
**Mechanism of injury:** Often occur in the setting of ankle fracture or inversion ankle sprain with recurrent lateral ankle instability
**Presentation:** Persistent pain following an ankle sprain or healed fracture, possible mechanical symptoms if fragment is unstable or loose within the joint
**Physical examination:** Localized ankle tenderness and swelling, may have an increased talar tilt and anterior drawer sign consistent with associated lateral ankle instability
**Differential diagnosis:** Recurrent lateral ankle instability and lateral talar process fracture
**Diagnostics:** Radiographs often normal, rule out fracture; CT and MRI better to delineate size, depth, and displacement of the lesion
**Treatment:** Observation with immobilization in skeletally immature patients and stable lesions in adults; unstable, displaced, and loose fragments require surgical intervention. Options include arthroscopic excision of the fragment with microfracture, OAT, OCA transplantation, and ACI. Occasionally, large fragments may be reduced and fixed if recognized acutely. Currently available literature favors microfracture with bone marrow stimulation in lesions with a diameter <15 mm and OAT for larger cystic lesions.
**Prognosis and return to sport:** Acute injuries are treated conservatively, and avoid weight bearing for 6 weeks in all surgically treated ankles. Return to sport is often possible at 3–6 months.

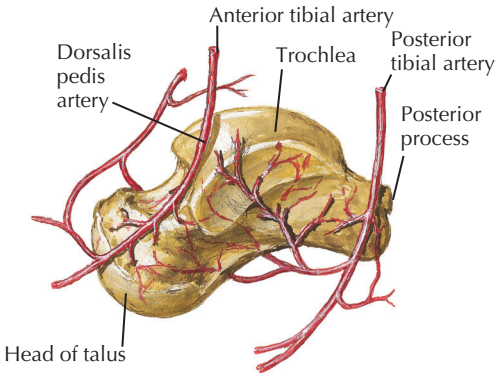
### Sever Disease

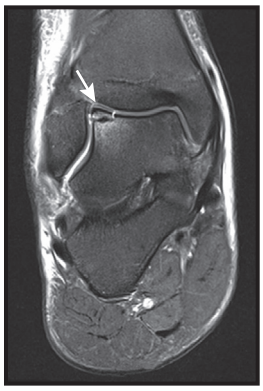
**Description:** Osteochondrosis at the insertion of the Achilles tendon on the calcaneal tuberosity in skeletally immature patients; most common during periods of rapid growth
**Mechanism of injury:** Traction phenomenon on the calcaneal apophysis from the strong gastrocsoleus complex; overuse syndrome
**Presentation:** Skeletally immature patient with activity-related posterior heel pain; bilateral in up to 50% of cases
**Physical examination:** Localized tenderness of the posterior heel, pain with medial–lateral compression, tight heel cords, pain on stretching of triceps surae, and positive squeeze test
**Differential diagnosis:** Achilles tendonitis, retrocalcaneal bursitis, calcaneal stress fracture, plantar fasciitis, and calcaneal bone cyst
**Diagnostics:** Diagnosis is based on clinical symptoms, but radiographs may show irregularity, sclerosis, and fragmentation of the calcaneal apophysis.
**Treatment:** Activity modification, anti-inflammatory medications, heel cord stretching, and heel cups or lifts
**Prognosis and return to sport:** Self-limiting condition; return to sport as symptoms allow

### Freiberg's Infarction

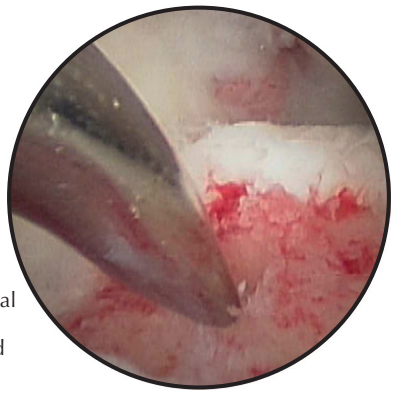
**Description:** Osteochondrosis of the second metatarsal head; most common in adolescent and young adult females
**Mechanism of injury:** May be secondary to acute trauma, repetitive microtrauma, or other conditions leading to osteonecrosis and collapse of the subchondral bone
**Presentation:** Acute pain (metatarsalgia), worse with weight-bearing activities, and second metatarsophalangeal (MTP) stiffness
**Physical examination:** Localized tenderness of the second metatarsal head, swelling, and limited MTP range of motion
**Differential diagnosis:** Metatarsal fracture, stress fracture, and metatarsalgia of other etiology (e.g., transfer metatarsalgia secondary to conditions of the neighboring hallux)
**Diagnostics:** Early radiographs normal; radiographs later show collapse with flattening of the second metatarsal head, possible osteophytes, and articular cartilage destruction (see Fig. 57.4B)
**Treatment:** Protective footwear, orthotics such as metatarsal pads or bars, and anti-inflammatory medications often alleviate

**A. Anatomy of the talus**



MR image of acute displaced lateral talar osteochondral lesion *(arrow)*



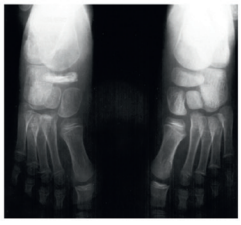
Arthroscopic photograph of lateral talar osteochondral lesion being treated with microfracture

**B. Freiberg's infraction**

Note the bony changes and flattening of the second metatarsal head.

**C. Köhler's disease**

Boy walks with painful limp, bearing weight on outside of foot to relieve pain.



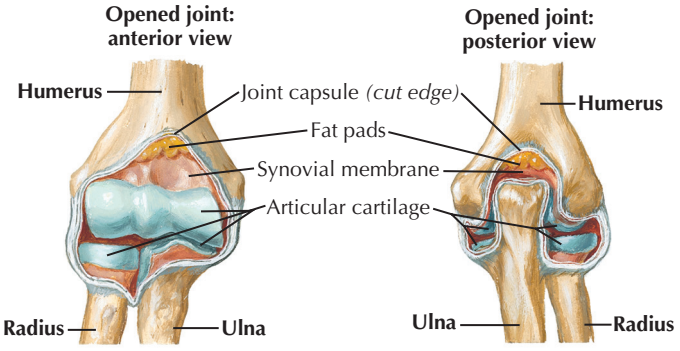
AP view showing sclerosis and flattening of the tarsal navicular

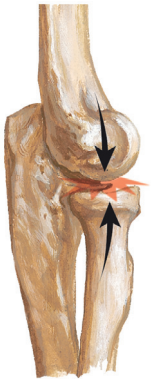
Lateral view showing sclerosis and flattening of the tarsal navicular

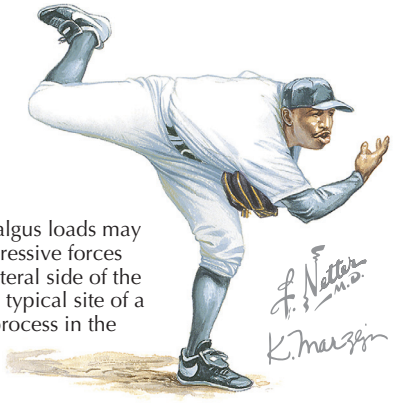
Soft, longitudinal arch support and 1/8 inch-thick lateral heel wedge help relieve foot pain until revascularization and ossification of navicular occur.

**Figure 57.4.** Foot and ankle injuries and problems.

**A. Anatomy of the elbow joint**



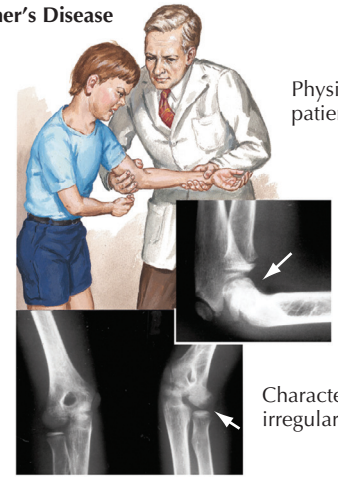



Repetitive valgus loads may create compressive forces across the lateral side of the elbow at the typical site of a pathologic process in the capitellum.

Sagittal MR image of the elbow shows a loose osteochondritis dissecans fragment of the capitellum.

**B. Panner's Disease**



Physical examination of patient with Panner's disease

Areas of lucency consistent with bony absorption

Characteristic lateral radiograph showing irregularity of the capitellum

**Figure 57.5.** Elbow injuries and problems.

symptoms in mild cases. Advanced cases may require immobilization in a walking cast or surgical intervention. Surgery can be categorized into articular restoration or realignment procedures and include synovectomy with core decompression of the metatarsal head, osteochondral plug transplantation, cheilectomy, osteotomy, or partial resection of the metatarsal head.

**Prognosis and return to sport:** Most patients show good prognosis with conservative management and may return to sport with resolution of symptoms.

### Köhler Disease

**Description:** Osteonecrosis of the tarsal navicular; usually presents in childhood

**Mechanism of injury:** Idiopathic

**Presentation:** Persistent midfoot pain in a young child (see Fig. 57.4C); more common in boys (approximately 80% of cases); may or may not recall an injury

**Physical examination:** Localized tenderness

**Differential diagnosis:** Accessory navicular and navicular stress fracture

**Diagnostics:** Radiographs show sclerosis and flattening of the navicular (see Fig. 57.4C).

**Treatment:** Immobilization and activity modification

**Prognosis and return to sport:** Self-limiting condition; navicular reconstitutes over time; return to activity when symptoms resolve, often 6–8 weeks

## Elbow

### Osteochondritis Dissecans (OCD)

**Description:** Osteochondral fragmentation with localized separation of subchondral bone and overlying articular cartilage (Fig. 57.5A); typically involves the capitellum of dominant hand

**Mechanism of injury:** Repetitive trauma and compression across the lateral elbow may damage the blood supply to the capitellum; common in adolescent throwing athletes and gymnasts

**Presentation:** Lateral elbow pain and possible mechanical symptoms if the fragment is unstable or loose within the joint

**Physical examination:** Localized tenderness over radiocapitellar articulation, possible effusion, often lack full extension; posterolateral rotatory instability test should be performed to examine potential elbow instability.

**Differential diagnosis:** Panner disease, lateral ligament injury or tendinosis, capitellum or radial head fracture, and loose body of other etiologies (e.g., following elbow dislocation)

**Diagnostics:** Radiographs may show sclerotic changes, loose bodies, and focal lesions in the anterolateral capitellum with rarefaction and irregularity of articular surface; MRI and CT useful for evaluation and classification of such lesions

**Treatment:** Conservative management in adolescents with stable lesions; large, unstable, or loose fragments typically require surgical intervention. Options include fragment removal with chondroplasty or microfracture versus fixation with drilling and possible bone grafting. Osteochondral autograft transplantation may reduce progression to osteoarthritis and lead to better long-term results.

**Prognosis and return to sport:** Return to sport varies based on treatment regimen. Conservative management allows return to sport when symptoms resolve. Surgical management warrants delayed return to sport, particularly if fragment reattachment is attempted. Early range of motion and strengthening are encouraged.

### Panner Disease

**Description:** Osteochondrosis of the capitellum in young children; an avascular segment develops and subsequently re-vascularizes over time.

**Mechanism of injury:** Likely lateral compression overuse injury

**Presentation:** Activity-related lateral elbow pain; typically young patients with OCD lesions and generally not associated with trauma

**Physical examination:** Localized tenderness, may lack full extension (see Fig. 57.5B)

**Differential diagnosis:** Capitellum OCD, stress fracture, lateral ligament injury, or tendinosis

**Diagnostics:** Radiographs often normal, may show fissuring and fragmentation of the capitellum on initial radiographs (see Fig. 57.5B)

**Treatment:** Activity modification, local modalities, and anti-inflammatory medications

**Prognosis and return to sport:** Self-limiting condition; return to sport when symptoms resolve

## Wrist

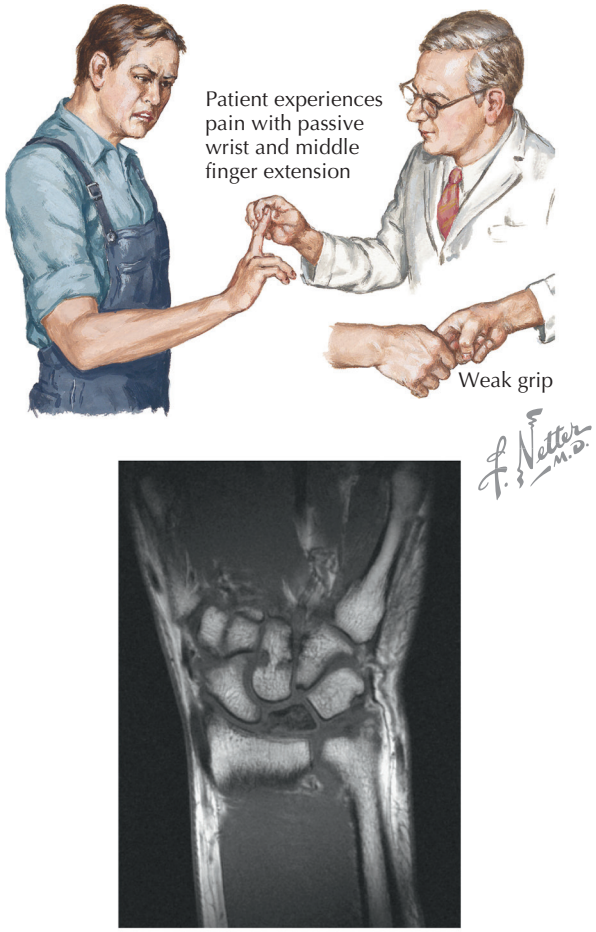
### Kienböck Disease

**Description:** Avascular necrosis and collapse of the lunate

**Mechanism of injury:** Etiology still unknown; possible causes include disruption of vascular supply, abnormal lunate morphology, and negative ulnar variance

**Presentation:** Chronic wrist pain, stiffness, and weak grip

**Physical examination:** Tenderness and swelling localized over the lunate and the radiocarpal joint; pain and limited range of motion with passive flexion and extension of wrist (Fig. 57.6)




MRI appearance of Kienböck disease

**Figure 57.6.** Kienböck disease.

**Differential diagnosis:** Tendonitis, scapholunate dissociation, and ganglion cyst

**Diagnostics:** Radiographs may show sclerosis and collapse of the lunate as well as ulnar negative variance. Advanced stages have associated degenerative changes. MRI may reveal diffuse signal changes over the lunate (see Fig. 57.6).

**Treatment:** Conservative measures as initial management; if persistent or worsening symptoms, surgical options include radial shortening versus ulnar lengthening in early stages and intracarpal arthrodesis versus proximal row carpectomy in advanced cases

**Prognosis and return to sport:** Return to sport as symptoms allow

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 58 ACUTE FRACTURES AND DISLOCATIONS IN ATHLETES

James P. Stannard • Steven L. Martin • Robert C. Schenck, Jr.

## TRANSPORTATION OF AN ATHLETE WITH FRACTURE OR DISLOCATION

- The need for and mode of transporting an injured athlete is determined following primary and secondary evaluations by the first responder. It must always be executed so that further injury is prevented.
- Planning the mode of transport and necessary equipment can help ensure that an appropriate technique is used (see Chapter 4, Sideline Preparedness and Emergencies on the Field).
- An athlete with a suspected spinal injury should not be moved by anyone other than certified medical personnel with appropriate stabilization. An ambulance should be contacted immediately (see Chapter 46, Neck Injuries), if not already present.
- If a serious fracture, such as that of the tibia, femur, or pelvis, is suspected, the safest and most efficient mode of transportation for a short distance is by means of a stretcher that is taken onto a utility vehicle or preferably by an ambulance.
- Suspected extremity fractures must be immobilized before transport (Fig. 58.1).

### Emergency Splint Equipment

- The ability to take care of an acute fracture in an athlete is directly related to one's level of preparedness and experience.
- Timely application of a splint to a fractured extremity minimizes the overall surrounding soft tissue injury and allows for safe transportation of the athlete for definitive care.
- Sports medicine providers (physician and trainers) should carry lightweight, easy-to-apply splint material in their sport medicine bag (see Chapter 4, Sideline Preparedness and Emergencies on the Field).
- **Basic mobile splint kit for a medicine bag** (Table 58.1):
  - Arm sling/shoulder immobilizer (medium and large sizes)
  - Alumafoam padded splints (several lengths that can be cut to size)
  - Structural aluminum malleable (SAM) splint
  - Knee immobilizer (universal size)
  - Webril (cast padding) and ace wraps (4 and 6 inches)
  - Plaster or fiberglass splinting material for self-made splints (prepackaged splints are available)
- **Advanced facility kit** (available at all high-volume sports complexes or via emergency medical services [EMS]):
  - Extremity vacuum splints or air splints (total body kits)
  - Cervical collar
  - Spine board
  - Femoral (Hare) traction splint
  - Crutches

## GENERAL PRINCIPLES

### Overview

- Braces or orthoses stop or limit range of motion, facilitate movement, or guide a joint through an arc of motion.
- Splints are used to immobilize and position one or several joints (see Fig. 58.1).
- Splints and braces are prescribed after a fracture to protect a partially healed fracture or to prevent the pain and soft tissue damage that occurs with motion.
- A cast is a stress-sharing device, allowing limited fracture motion and callus formation as a result of secondary bone healing. The joints above and below a fracture should be immobilized to prevent rotation and translation of the fracture fragments.

### Splint Treatment

- Splints are beneficial because they provide some stabilization at the fracture site, but they may be removed for rehabilitation treatment.
- After a cast is initially removed, splints are frequently used during activity or at night to reduce pain and discomfort and allow additional fracture consolidation.
- Joint stiffness is very common after cast removal and resolves after weeks to months of aggressive, but appropriate, rehabilitation.
- Immobilization of the joints above and below the fracture site often leads to stiffening and the need for a prolonged rehabilitation program.
- A cast brace can provide partial immobilization while allowing some range of motion and weight bearing on a limb.
- Once a fracture achieves an appropriate degree of stability as a result of callus formation, the cast can be replaced with a splint or brace, allowing motion of the joints proximal and distal to the fracture, without compromising the support at the fracture site.

### Initial Evaluation

- Immediately after an athlete sustains a fracture or dislocation, the neurologic and vascular status must be evaluated and documented.
- Temporary immobilization measures should be employed as the patient is transported to the hospital (see Fig. 58.1).
- The mechanism of injury can provide clues regarding what type of injury has occurred.
- In all upper extremity fractures, assess the following:
  - Open or closed fracture
  - Patient complaints of pain, swelling, or paresthesias
  - Vascular status distal to the injury
  - Active and passive range of motion of wrist and/or digits
  - Radial, median, and/or ulnar nerve function and possible compression; axillary nerve function should also be documented with shoulder injuries
- In all lower extremity fractures, assess the following:
  - Open or closed fracture
  - Patient complaints of pain, swelling, or paresthesias
  - Vascular status distal to the injury (pulses, color, and capillary refill)
  - Active and passive range of motion of all metatarsophalangeal and interphalangeal joints
  - Nerve function and possible compression

## UPPER EXTREMITY INJURIES

### Distal Humerus Fractures

- Fractures that have minimal displacement and are in near anatomic alignment can be treated with casting or splinting if they are stable enough to allow early elbow motion.

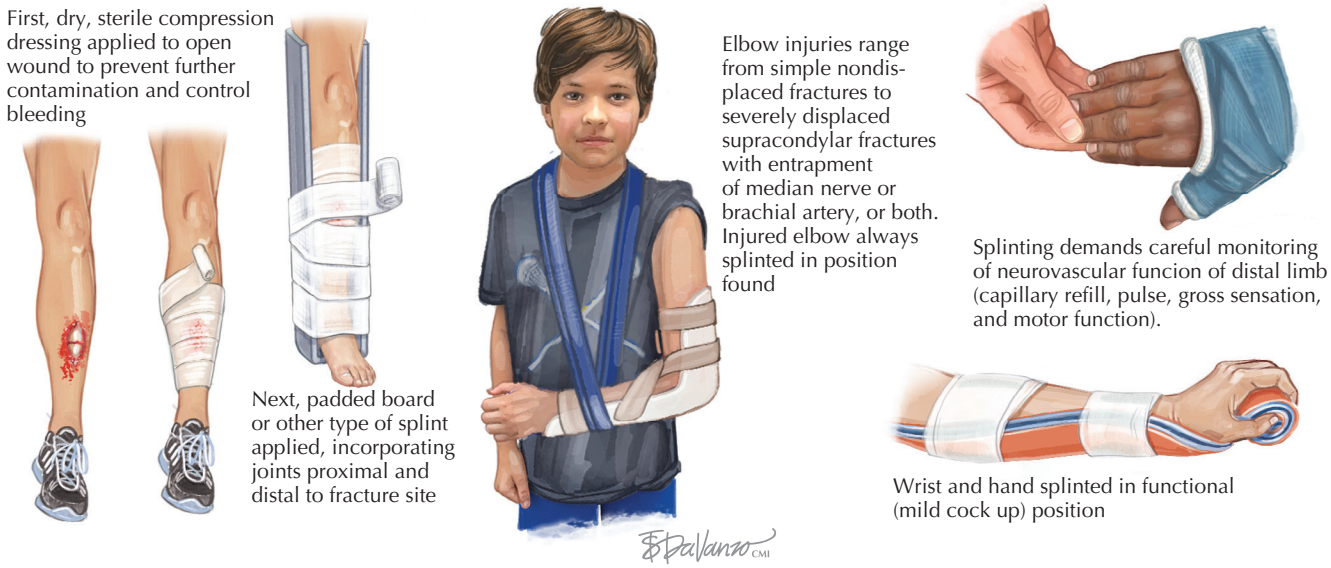


**Figure 58.1.** Prehospital care of fractures.

**TABLE 58.1 SPLINTS USED PER INJURY LOCATION**

| | |
|---|---|
| **Clavicle and Acromioclavicular (AC) Joint** | Arm sling |
| **Shoulder and Proximal Humerus** | Arm sling and a 6-inch ace wrap swathe |
| **Distal Humerus and Elbow** | Air or vacuum splint |
| **Forearm and Wrist** | Fiberglass/plaster splint, structural aluminum malleable (SAM) splint, prefabricated orthosis |
| **Hand** | Buddy taping, Alumafoam splint |
| **Spine** | Cervical collar and spine board |
| **Hip and Femur** | Femoral traction splint |
| **Knee and Proximal Tibia** | Knee immobilizer |
| **Tibial Shaft, Ankle, and Foot** | Air or vacuum splint |

- Elbow stiffness and permanent motion loss are significant risks following elbow trauma.
- Cast or splint treatment of these injuries should initially allow no motion at the elbow.
- Most distal humerus fractures require surgical stabilization, followed by early functional motion of the elbow.

## Olecranon Fractures

- Minimally displaced fractures of the olecranon with <2-mm displacement and intact elbow extensor mechanism can be treated with cast or splint immobilization.
- Early elbow motion is necessary for good functional results.
- The cast should be adequately padded to avoid skin breakdown at the edges of the cast or splint.
- The cast should volarly extend to the distal palmar crease and dorsally to the metacarpophalangeal joints to allow full motion at the interphalangeal and metacarpophalangeal joints.
- Once there is early evidence of healing, the elbow should be mobilized to prevent stiffness.
- An elbow-hinged brace can protect the fracture and maintain motion of the elbow. Often, some terminal extension is lost, but functional range of motion is maintained.
- Aggressive rehabilitation is critical as the loss of some terminal extension may have implications on athletic performance even if it does not affect activities of daily living.
- Displaced fractures of the olecranon require open reduction and internal fixation (ORIF), followed by early elbow range of motion to minimize the development of elbow stiffness.

## Radial Head Fractures

- Minimally displaced fractures can be treated in a sling with early elbow range of motion.
- For fractures requiring ORIF, short-term immobilization is needed, followed by early bracing mobilization.
- A hinged elbow brace is typically used to allow protection of the fixation during initiation of early range of motion.
- Most severe fractures have better outcomes with radial head replacement than with ORIF, even in young patients.

## Distal Radius Fractures

- Minimally displaced fractures can be treated with a long-arm or Münster cast, followed by a short-arm cast.
- Rigid immobilization for 4–6 weeks, followed by a removable splint, is recommended.
- Displaced fractures require reduction before splinting and should be immobilized in a long-arm splint.
- The cast should be volarly trimmed to the proximal palmar crease and distally to the metacarpophalangeal prominences to allow free finger movement. The cast should be trimmed to allow the thumb full opposition with the small finger.
- Distal radius fractures requiring ORIF require long-arm splints in the immediate postoperative period, followed by short-arm casting or removable gauntlet splint depending on the pattern of injury.

## Scaphoid Fractures

- For nondisplaced or minimally displaced fractures, thumb spica cast immobilization is recommended. The wrist should be in a

neutral position relative to flexion, extension, and radial deviation.
- Fractures requiring open reduction should be immobilized in a thumb spica splint in the immediate postoperative period, followed by a thumb spica cast.
- Athletes—and other high-activity and high-demand patients—frequently have better outcomes with ORIF of scaphoid fractures.

## Metacarpal and Phalangeal Fractures

- Dislocations of the proximal interphalangeal (PIP) joint should be immobilized using static dorsal extension splinting (Fig. 58.2A).
- It is important to secure the proximal phalanx to the splint to ensure that the PIP joint does not extend when a patient flexes the metacarpal phalangeal joint.
- Metacarpal neck and shaft fractures are best treated with a cast or splint.
- The splint is applied with the hand in "safe" position with the wrist in extension, the metacarpal phalangeal joints flexed, and the proximal and distal interphalangeal joints in extension. Displaced fractures require reduction before splinting.
- Phalanx fractures that are minimally displaced can be treated with "buddy taping," wherein the fractured finger is taped to an adjacent finger. The adjacent finger functions as a splint but allows continued range of motion. Displaced fractures require reduction before splinting.

## Clavicle Fractures

**Description:** The most common fracture, accounting for approximately 5%–10% of all fractures; 85% involve the middle third of the clavicle (Fig. 58.2B)

**Mechanism of injury:** Most result from a fall onto the ipsilateral shoulder.

**Initial on-field management:** The fracture displacement and pain level is significant and clinical diagnosis can be made on the field; the athlete should be assisted off the field with the arm held at his or her side. Initial management with a sling with or without a swathe is sufficient. A 6-inch ace wrap can be used

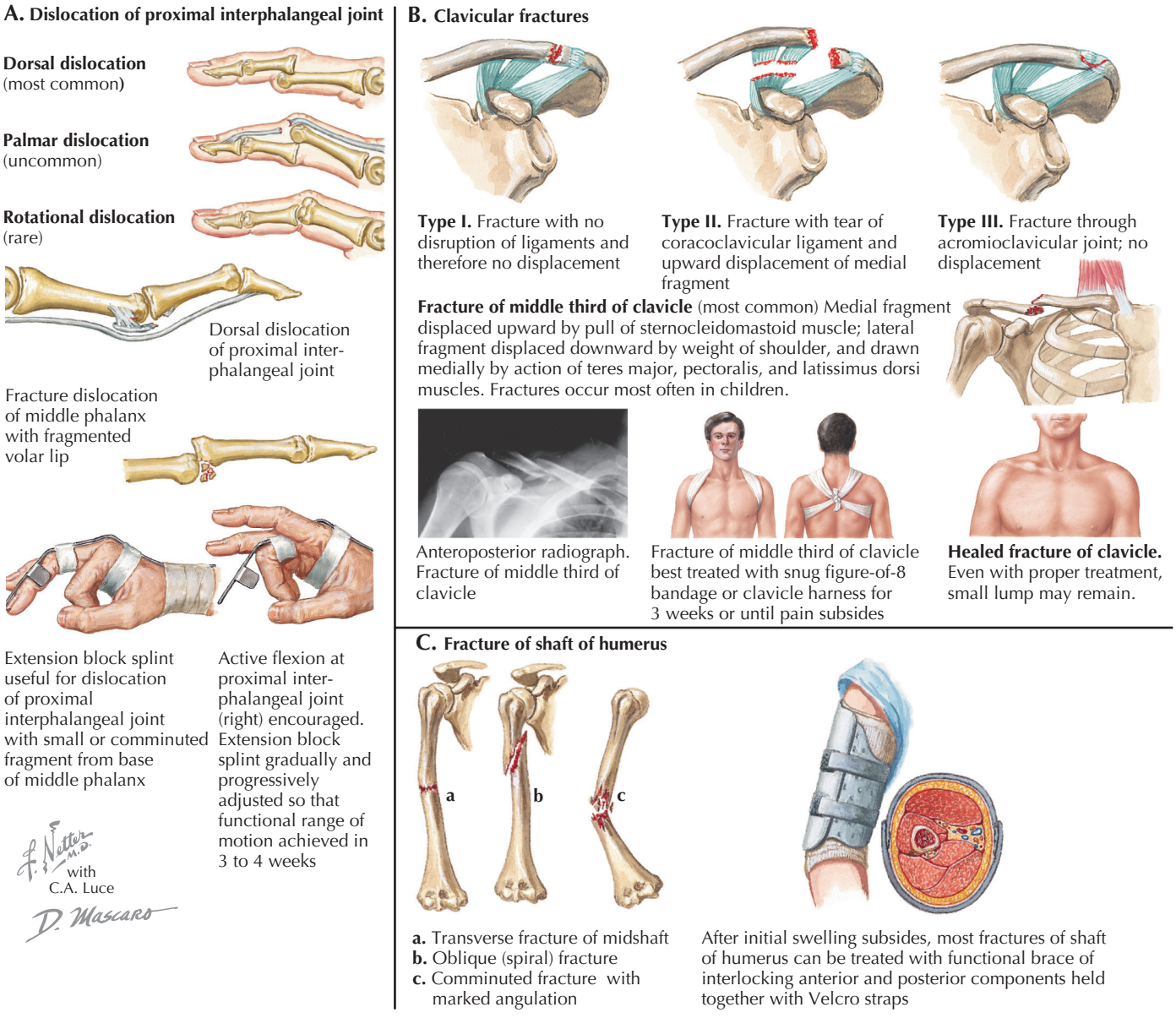


Figure 58.2. Upper extremity fractures and dislocations (proximal interphalangeal, clavicle, and humeral shaft).

for a swathe and improves comfort by supporting the elbow and immobilizing the arm to the body.

**Evaluation:**

- **Inspection:** Look for obvious deformity and inspect for skin breakdown or tenting.
- **Palpation:** Check for neck, sternoclavicular joint, midclavicle, acromioclavicular joint, scapula, and proximal humerus tenderness.
- **Neurovascular examination:** Evaluate the upper extremity including the axillary nerve. Sensation may not be completely reliable, so motor function of the deltoid should be carefully assessed.

**Radiographs:** An anteroposterior (AP) and an AP with a cephalic tilt of 20 degrees are sufficient.

**Treatment:** Nonsurgical treatment for several closed clavicle fractures with application of a figure-of-eight collar or arm sling. Indications for surgical treatment include skin compromise secondary to severe displacement, open fractures, and a floating shoulder with neurovascular compromise. Level I studies have shown that ORIF has improved results with significantly displaced clavicle fractures. Shortening and overlap of >2 cm in an active individual should usually be treated surgically. The method of fracture stabilization is generally with a contoured plate or an intramedullary device. Problems with conservative care include a higher incidence of both nonunion and malunion.

**Prognosis and return to sport:** Several recent reports suggest a faster return to sport with surgical intervention than with conservative care. One report with intramedullary fixation reported a return to training in 6 days and a return to competition in 17 days with 12 high-performance athletes. Prognosis for athletes is very good following clavicle fractures.

## Humeral Shaft Fractures

**Overview:** Uncommon, accounting for approximately 1% of fractures in trauma registries (see Fig. 58.2C)

**Mechanism of injury:** More frequently from a fall onto the involved extremity but can occur from a direct blow; on occasion, rotational injuries can also occur in certain sports such as arm wrestling

**Presentation:** Painful, unstable extremity with swelling and bruising

**Physical examination:** Gentle palpation of the shaft of the humerus should be performed, noting any areas of malalignment and severe pain

**Associated injuries:** Associated nerve injuries are relatively common and should be documented; radial nerve injuries are most common. Most common fracture type associated with a nerve injury is a transverse mid-diaphyseal fracture. Document a motor neurologic examination of the axillary, musculocutaneous, radial, median, and ulnar nerves. Skin should also be examined to rule out any open fractures. Shoulder and elbow joints should be examined and imaged.

**Diagnostics:** AP and lateral radiographs of the entire humerus should be obtained. In addition, radiographs of the shoulder and elbow should also be recorded to help evaluate for associated pathology.

**Treatment:** In most cases, nonsurgical management of midshaft fractures is appropriate. Treat with functional bracing and by allowing active flexion and extension of the elbow during the healing process to prevent elbow stiffness; motion helps reduce the fracture and aids in the healing process. Isometric muscle exercises in the functional brace can aid in maintaining alignment and assist in fracture healing. Patients who have sustained significant nerve injuries that prevent active range of motion are not candidates for functional bracing. The humerus can heal with an angulation of up to 30 degrees and shortening of 2–3 cm without functional problems in most patients. However, the functional needs of a high-performance athlete are different, and accepting that degree of malalignment is discouraged. If functional bracing is elected to treat an athlete, special care should be taken to follow the healing process and evaluate the alignment. If alignment is not maintained in the dominant arm of a skilled athlete, consideration should be given to surgical stabilization of the fracture. Surgical treatment usually involves either plating or use of an intramedullary nail (see Fig. 58.2C).

**Prognosis and return to sport:** Prognosis for healing and good functional recovery following isolated humerus shaft fractures is good. Patients who have associated nerve injuries or shoulder dislocations have a less favorable prognosis but may still be able to return to competition if the nerve injury resolves.

## Forearm Fractures

**Description:** Forearm injuries represent approximately 5% of all fractures and include isolated ulnar fractures and fractures of both the radius and ulna. Monteggia and Galeazzi fractures involve the proximal and distal radioulnar joints, respectively (see Fig. 58.3A, B, and C). Monteggia fractures involve fracture of the ulna with dislocation of the radial head. Galeazzi fractures involve fracture of the radius shaft (usually at the level of the junction of the middle and distal third) with injury to the distal radial ulnar joint.

**Mechanism of injury:** Isolated ulnar fractures are seen in contact sports athletes resulting from a direct blow to the arm (see Fig. 58.3D). Moreover, fractures of both radius and ulna are seen in contact athletes, which may be open or closed.

**Initial on-field management:** The injured extremity should be stabilized with a splint, and the athlete must be taken off the field for further evaluation.

**Evaluation:**

- **Inspection:** Determine if the fracture is open or closed.
- **Palpation:** Assess both the elbow and wrist of the injured forearm to be sure there is no damage to either the proximal or distal radioulnar joint.
- **Neurovascular examination:** Check vascular status of the arm by palpation of the radial pulse in addition to assessment of the capillary refill at the nail bed of each digit. The radial nerve sensation is tested by lightly touching the dorsum of the first web space. The ulnar nerve sensation is tested by lightly touching the small finger. The median nerve sensation is tested by lightly touching the volar side of the index finger.

**Radiographs:** Should include two views of the forearm and must always include adequate views of both the elbow and wrist

**Treatment:** In skeletally mature athletes, surgical treatment is recommended to restore anatomic alignment in **displaced radial and ulnar shaft fractures.** The musculature of the proximal forearm makes maintenance of closed reduction difficult. For **undisplaced fractures of the ulna,** management of injury is symptomatic and includes prohibition of sporting activities until clinical and radiographic evidence of fracture union is obtained; bracing or casting is used. Serial radiographs are recorded to ensure no further displacement and to monitor eventual healing. **Displaced fractures** may require surgical fixation. Isolated ulnar fractures are at risk for delayed or nonunion.

**Return to sport:** In general, a minimum of 4–6 weeks

## Compartment Syndrome in Forearm

**Description:** An increase in pressure in a muscle compartment leading to damage to the structures within the compartment, including muscles and nerves

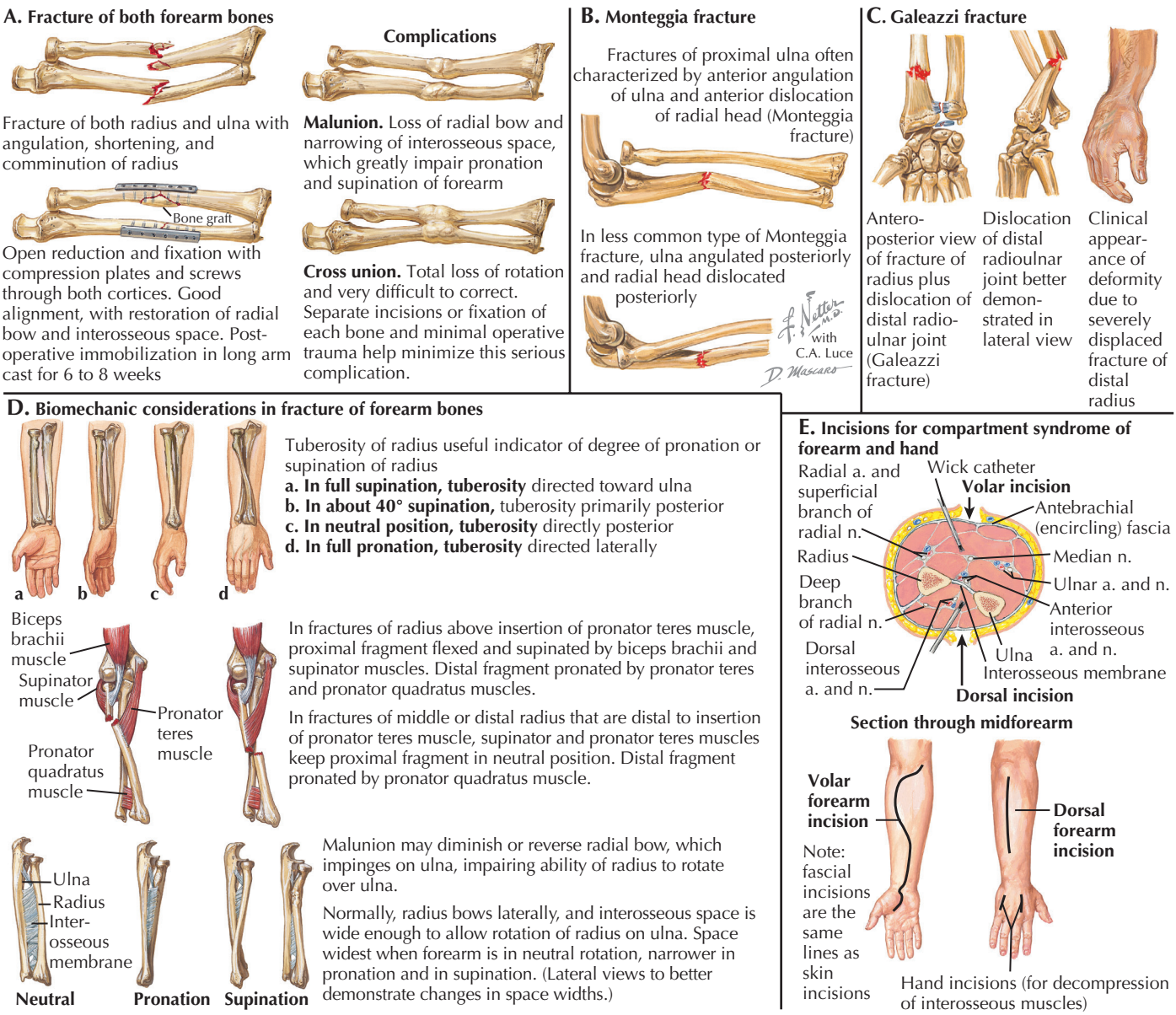


**Figure 58.3.** Upper extremity fractures and dislocations (forearm and compartment syndrome).

**Mechanism of injury:** Typically the result of high-energy impact (either forceful or crushing) to the forearm, and occurs more frequently in the volar compartment; in athletes with forearm injuries, a tense swelling and an out-of-proportion pain may suggest compartment syndrome. Excessive pain may be the first clue to an early compartment syndrome and cannot be ignored in athletes. A higher muscular volume may put the athlete at an increased risk of compartment syndrome.

**Evaluation:**

- **Inspection:** The forearm should be evaluated for swelling accompanied by severe pain.
- **Palpation:** Assess the compartment compressibility by gently pressing against the forearm compartments and comparing it to the contralateral side. Tense, noncompressible compartments or severe pain with passive motion of the fingers out of proportion to the injury should increase suspicion for compartment syndrome.
- **Neurovascular examination:** Assess vascular status of the arm by palpating the radial pulse in addition to assessment of the capillary refill at the nail bed of each digit. Radial nerve sensation is tested by lightly touching the dorsum of the first web space. Ulnar nerve sensation is tested by lightly touching the volar small finger. Median nerve sensation is tested by lightly touching the volar index finger.

**Radiographs:** Two views of the forearm should be obtained to ensure that there is no fracture.

**Treatment:** If compartment syndrome is diagnosed, emergent fasciotomies should be performed (see Fig. 58.3E).

## LOWER EXTREMITY INJURIES

### Tibial Plafond Fractures

- A long leg cast is only suitable for fractures with minimal displacement and no damage of the articular joint surface.
- Comminuted fractures usually have soft tissue damage that requires delayed surgery, which requires spanning external fixation before surgical fixation to allow soft tissue recovery and avoid complications.

## Foot Fractures

- Fractures involving the forefoot, midfoot, or hindfoot can be immobilized using a short leg splint until swelling subsides.
- Fractures that are minimally displaced can be treated with a short leg cast.
- Fractures of the foot phalanges can often be treated with a hard-soled shoe and "buddy taping" (Fig. 58.4A).

## Hip Fractures and Dislocations

**Description:** Intertrochanteric hip fractures and femoral neck fractures are rare in athletes; hip dislocation and femoral head fractures are more common injuries among athletes and younger individuals (see Fig. 58.4B, C, and D). Fracture dislocation of the hip in a young, athletic patient should be viewed as a surgical emergency and a career-threatening injury. There is a correlation between the duration for which a hip remains dislocated and the development of osteonecrosis of the femoral head.

**Presentation:** Patients with posterior hip dislocations present with shortened extremities with the hip flexed, adducted, and internally rotated; those with anterior hip dislocation present with shortened extremities with the hip flexed, abducted, and externally rotated.

**Physical examination:** No attempt should be made at reduction of the hip before obtaining good quality radiographs. A neurologic examination should be documented before attempting reduction.

**Diagnostics:** Radiographs are key and should be obtained before attempting reduction, to prevent severe displacement of associated minimally displaced fractures. AP and lateral views of the hip joint as well as Judet views (45-degree oblique views) of the acetabulum must be recorded.

**Treatment:** After obtaining radiographs and ruling out an associated fracture, closed reduction of the hip can be attempted under conscious sedation. It is important to have complete relaxation of the hip muscles before attempting reduction. If the patient has an associated femoral neck fracture, he or she

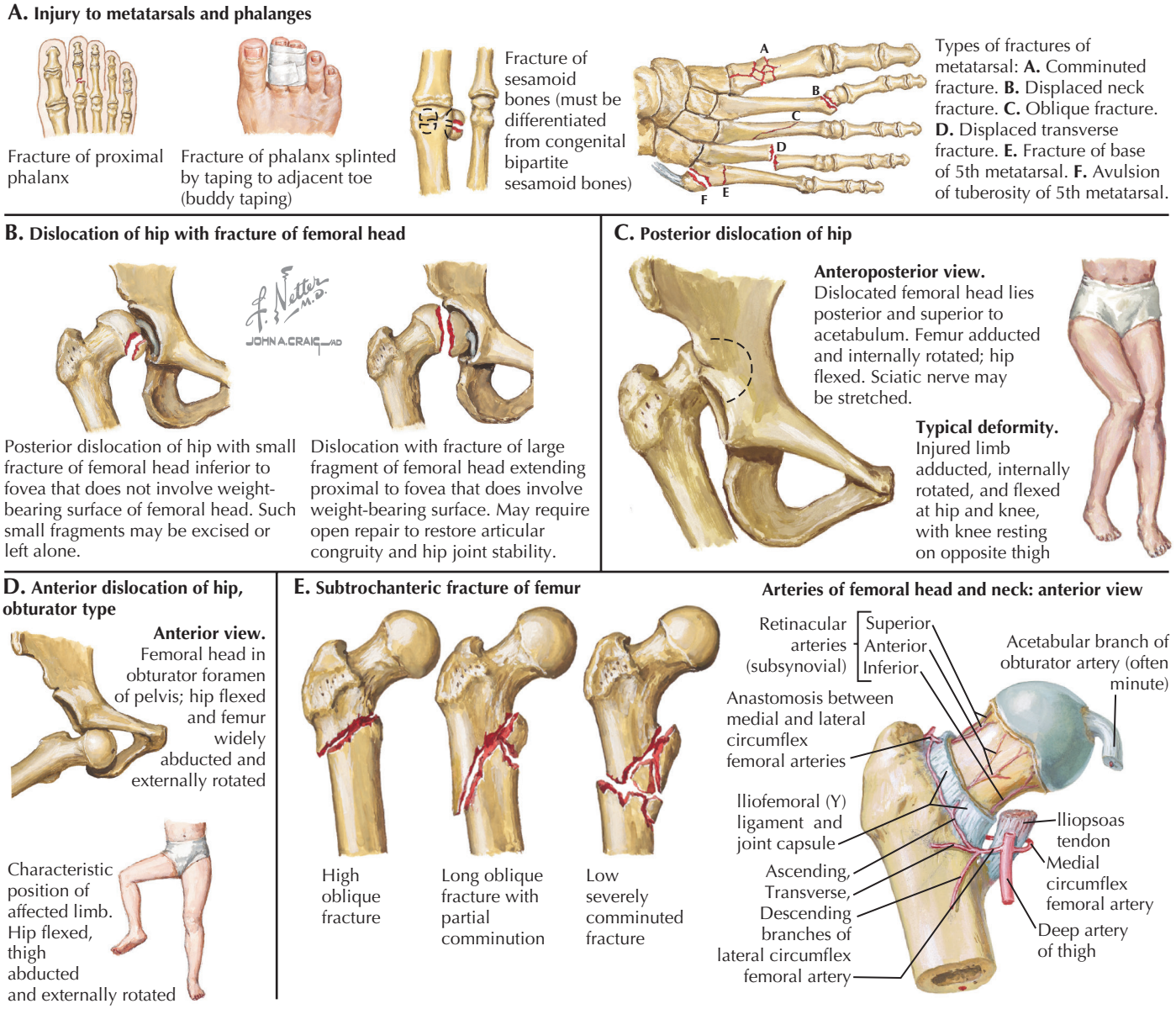


**Figure 58.4.** Lower extremity fractures and dislocations (foot, hip, and femur).

should be expeditiously taken to the operating room. If there is an associated acetabulum fracture, it is reasonable to attempt a closed reduction as long as there are no large bone fragments in the joint that may block reduction. An attempt at a closed reduction should be made by an experienced physician. If that fails, the patient should be taken to the operating room for a reduction under general anesthesia. There are three primary methods for achieving closed reduction of a posterior hip dislocation.

- **Bigelow maneuver:** This is accomplished by having an assistant provide downward pressure on the anterior superior iliac spine while the physician pulls in-line traction, flexes the hip 90 degrees, and applies internal rotation and adduction until reduction is achieved.
- **Allis maneuver:** In this case, an assistant stabilizes the pelvis while the physician pulls in-line traction, flexes the hip to 90 degrees, and gently alternates between internal and external rotation in an attempt to reduce the hip.
- **Stimson maneuver:** In this case, the patient is placed prone with the affected extremity hanging off the end of the examination table. Both the hip and knee are flexed 90 degrees, and a downward force is applied to the calf.
- Anterior hip dislocations are reduced with the patient supine using traction and counter-traction until reduction is obtained.

**Prognosis and return to sport:** Long-term results following hip dislocations are poor, even if reduction is prompt and appropriate. Athletes who sustain a hip dislocation with or without fracture should be counseled that they may have sustained a career-ending injury.

## Femur Fractures

**Description:** Uncommon sports injury; requires high-energy trauma

**Associated injuries:** Carefully assess athletes for other injuries. Femur fractures can be associated with substantial blood loss. Common systemic injuries include head, chest, and abdominal injuries. The most important associated skeletal injury is a fracture of the femoral neck. It is critical to diagnose such injuries and avoid any additional displacement of the neck fracture; a displaced femoral neck fracture puts the blood supply to the femoral head at risk and may lead to avascular necrosis of the femoral head (see Fig. 58.4E).

**Presentation:** Severe pain and inability to bear any weight on the involved leg

**Physical examination:** Displaced femur fractures present with malalignment of the limb and severe pain. Document the neurologic and vascular status of the leg. Minimize movement of the injured extremity until radiographs can be obtained. Imaging should include views of the femoral neck. Evaluate for skin lacerations, which can indicate an open fracture, and assess for compartment syndrome. Development of compartment syndrome represents a surgical emergency and should raise concern for vascular injury.

**Diagnostics:** Plain radiographs; AP and lateral views are generally adequate to characterize the injury. If the femoral neck is not clear on plain hip radiographs, splints must be removed and an internal rotation view of the femoral neck should be obtained. If the patient has an asymmetric pulse, emergent arteriogram or computed tomography (CT) angiogram should be obtained.

**Treatment:** Initial treatment should concentrate on stabilization of the extremity (see Fig. 58.4E). Gentle longitudinal traction should be applied, and subsequently, a splint should be applied. A Hare traction splint can help provide pain relief. Definitive treatment of femur fractures requires surgical stabilization. Intramedullary nails are the most commonly used implants.

**Prognosis and return to sport:** Long-term prognosis is good. Athletic activities should be deferred for 4–6 months. Abductor muscle weakness and dysfunction following antegrade intramedullary nailing is common. Knee pain is common after retrograde femoral nailing. Loss of muscular strength is common and may take ≥1 year to completely recover. Physical therapy should concentrate on rehabilitation of muscles around the hip, particularly the abductors.

## Knee Dislocation

**Description:** Occurs when the tibia is no longer in contact with the femur; associated with ligament, nerve, and vascular injury (see Fig. 58.5A); seen in contact sports such as football. Increasing incidence is being recognized both with high energy mechanisms such as motor vehicle collisions and athletic injuries. Most dislocations spontaneously reduce and require careful evaluation to avoid missing the diagnosis.

**Mechanism of injury:** Result of a large force applied across the knee joint; knee joint is disrupted with dislocation of the tibiofemoral articulation with associated multiligament injury. Typical mechanism is hyperextension with the foot fixed in place.

**Initial on-field management and evaluation:**

- Gross deformity may be apparent, but it may look relatively normal if the dislocation has spontaneously reduced.
- **Palpation:** The knee joint, leg, and thigh should be palpated to assess for possible fracture or associated soft tissue injury.
- **Neurovascular examination:** The dorsalis pedis and posterior tibial pulse should be manually palpated and compared to the contralateral side. Careful sensory and motor examination should be performed and documented, with particular attention to peroneal nerve function.
- **Ligament examination:** Careful knee ligament examination should be performed to assess any cruciate and collateral ligament injury.

**Radiographs:** Three views of the knee should be obtained to assess for fracture. The most common associated fracture (by far) is a tibial plateau fracture. Two views each of the femur and the tibia should also be obtained to check for associated injuries. Magnetic resonance imaging (MRI) is recommended to confirm ligament injury and to assess the extent of the injury.

**Treatment:** Requires reduction of dislocation followed by assessment of vascular status (see Fig. 58.5A); pulses of the involved extremity should be palpated. Any discrepancy in pulse should prompt immediate evaluation for vascular injury by means of diagnostic studies such CT or MR angiography. Neurologic evaluation should be performed to assess tibial and peroneal nerve function. If knee dislocation is suspected, the athlete should be immediately transferred to a medical facility for evaluation by an orthopedic and vascular surgeon. Vascular injury requires immediate intervention by the vascular surgery team. Multiple ligament injuries in knee dislocations will require surgical reconstruction. Rehabilitation following a knee dislocation is intensive and prolonged (see Fig. 58.5A).

## Tibial Shaft Fractures

**Description:** Generally low- to medium-energy fractures in athletics; despite the lower energy mechanism, a tibial fracture can be a devastating and career-ending injury for an athlete. Early stabilization with a well-supervised rehabilitation program is critical for favorable outcomes (Fig. 58.5B).

**Mechanism of injury:** Noncontact rotational injuries occurring in sports such as downhill skiing to direct blow-type injuries occurring in contact sports such as football or soccer

**Initial management:** Deformity, instability, and pain confirm the diagnosis. Immediate splinting before transportation is

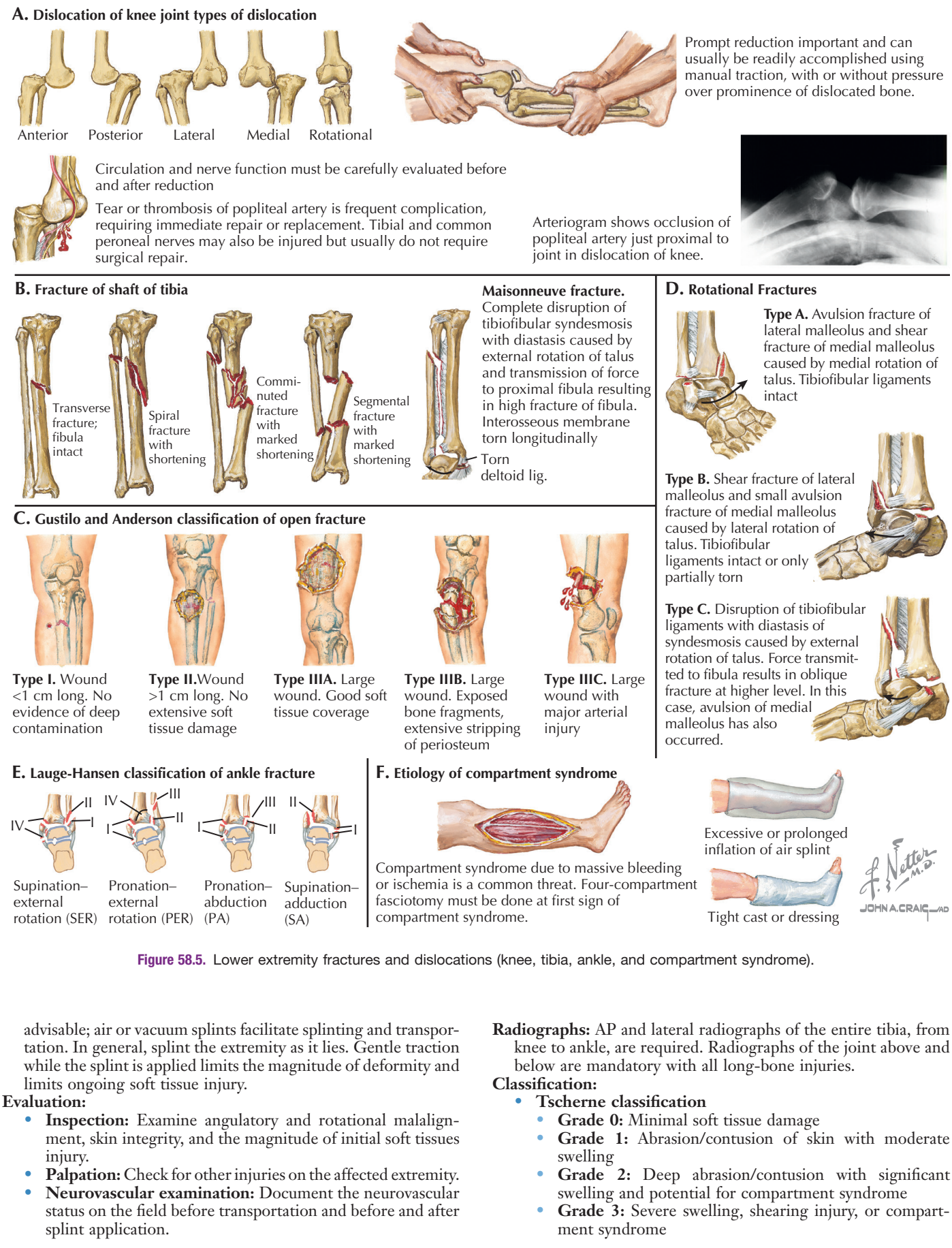


**Figure 58.5.** Lower extremity fractures and dislocations (knee, tibia, ankle, and compartment syndrome).

advisable; air or vacuum splints facilitate splinting and transportation. In general, splint the extremity as it lies. Gentle traction while the splint is applied limits the magnitude of deformity and limits ongoing soft tissue injury.

**Evaluation:**

- **Inspection:** Examine angulatory and rotational malalignment, skin integrity, and the magnitude of initial soft tissues injury.
- **Palpation:** Check for other injuries on the affected extremity.
- **Neurovascular examination:** Document the neurovascular status on the field before transportation and before and after splint application.

**Radiographs:** AP and lateral radiographs of the entire tibia, from knee to ankle, are required. Radiographs of the joint above and below are mandatory with all long-bone injuries.

**Classification:**

- **Tscherne classification**
  - **Grade 0:** Minimal soft tissue damage
  - **Grade 1:** Abrasion/contusion of skin with moderate swelling
  - **Grade 2:** Deep abrasion/contusion with significant swelling and potential for compartment syndrome
  - **Grade 3:** Severe swelling, shearing injury, or compartment syndrome

- **Gustilo classification of open fractures:** An open wound over the fracture remarkably increases the likelihood of an infection; the more severe the open wound, the greater the likelihood of infection (Fig. 58.5C).
  - **Grade I:** Open wound <1 cm
  - **Grade II:** Larger open wounds (1–10 cm) with slight contamination
  - **Grade III:** Severe soft tissue damage
    - **A:** can be closed primarily
    - **B:** severe soft tissue wound with one or all of the following: severe contamination, significant periosteal stripping, flap coverage, or VAC required for closure
    - **C:** vascular repair or reconstruction required

**Treatment: Closed stable fractures** can be treated with casting and functional bracing. However, athletic individuals frequently benefit from surgical stabilization that allows early functional rehabilitation. Locked intramedullary nailing is the treatment of choice for most closed **unstable diaphyseal tibial fractures**. Open reduction with plate osteosynthesis is used in cases in which the fracture extends too close to the adjacent joint for adequate intramedullary nailing. **Open fractures** are treated with aggressive soft tissue management with debridement and irrigation and early closure or coverage. The success of treatment is related more to the adequacy of the soft tissue management than it is with the fracture fixation method. External fixation is reserved for **high-energy, severe open grades** of tibial shaft fracture where the extent of the soft tissue zone of injury demarcation is poorly defined during initial debridement.

## Ankle Fractures

**Description:** Ankle injuries are among the most common injuries treated by the sports medicine physicians (see Fig. 58.5D).

**Mechanism of injury:** Most ankle injuries involve a planted fixed foot with a rotational force applied to the leg. The position of the foot and the magnitude and direction of the force determines the pattern of injury. How quickly the foot is allowed to disengage from the turf often determines whether an ankle sprain or an ankle fracture occurs.

**On-field management:** Unstable ankle injuries and those associated with obvious deformity should be splinted on the field before transportation. With a stable ankle injury, the athlete can be assisted to the sidelines where further treatment can be administered. Initial management includes neurovascular evaluation, splint application, extremity elevation, and ice therapy.

**Evaluation:**

- **Inspection:** Look for deformities, including foot alignment. Note areas of swelling including the syndesmotic region and foot. Document any areas of skin contusion, abrasion, or disruption.
- **Palpation:** Start by checking for associated injury to the knee or proximal fibula. Palpate the medial side, lateral side, syndesmosis, hindfoot, and midfoot. Localize all areas of direct tenderness and try to differentiate from ligamentous areas versus bone.
- **Stability:**
  - **Squeeze test:** Squeeze the midleg, compressing the tibia and fibula together, looking for syndesmotic pain.
  - **External rotation test:** In the absence of any obvious deformity, a lightly applied external rotation force to the foot can help identify a fracture. Stability and the absence of pain on external rotation rule out most unstable ankle fractures.
  - **Drawer test:** Stabilize the tibia with one hand and pull the foot forward. Look for excessive anterior translation of the talus out of the mortise, and compare with the uninjured ankle.
- **Neurovascular examination:** Document the presence or absence of pedal pulses, capillary refill, and color of the toes. Check all sensory dermatomes of the foot and ankle.

**Radiographs:** Key examination findings that indicate a need for radiographs in the absence of deformity include pain at the malleoli, tenderness at the tip or posterior edge of the malleolus, severe swelling, and inability to bear weight. **Initial radiographs** should include a high-quality AP, a mortise (AP with the leg 15 degrees internally rotated), and a lateral radiograph. CT scans and MRIs are occasionally required for occult fractures and to rule out osteochondral lesions of the talus.

**Classification:**

- The **Weber/AO system** depends on the level of the fibular fracture.
  - Type A: Fibular fracture is below the level of the plafond.
  - Type B: Fibular fracture is at the level of the plafond.
  - Type C: Fibular fracture is above the level of the plafond and is generally associated with an injury to the syndesmotic ligament.
- **Lauge–Hansen classification** is based on the position of the foot and the direction of the deforming force (see Fig. 58.5E).
- **Anatomic classification** is based on the number of malleoli fractured and the level and pattern of fracture: isolated lateral malleolus, bimalleolar, and trimalleolar.
- **Syndesmotic ligament injury:** Examination findings suggestive of injury include tenderness over the anterior aspect of the syndesmosis and a positive squeeze or external rotation test. Radiographic findings include an increase in the medial clear space (distance between the lateral wall of the medial malleolus and medial wall of the talus) of >4 mm and the distal tibiofibular space (distance between the posterior border of the tibia and the inner border of the fibula) of >6 mm. However, injury may not be radiographically apparent; thus, routine stress radiographic testing is necessary to detect any instability.
- **Maisonneuve fracture:** Fracture of the proximal fibula associated with a rotational injury to the ankle; this is often overlooked. Patients often do not complain of pain in the region of the proximal fibula when more painful distal injuries are present; more likely when deltoid ligament is ruptured or the medial malleolus is fractured without a fracture of the lateral malleolus. Full-length AP and lateral radiographs of the tibia and fibula should be obtained in such cases.

**Special tests:**

- **External rotation stress test:** The syndesmosis is disrupted with an external rotation force against a fixed foot in both supination external rotation (SER) stage IV and with pronation external rotation injuries. In cases where in it is not clear if the syndesmosis is completely torn, an AP radiograph should be recorded with application of external rotation stress to the foot.
- **Ankle joint effusion:** A large ankle joint effusion on plain radiographs is often an indication of an occult fracture. A CT scan or MRI may be indicated in such cases.

**Treatment:**

- **Isolated lateral malleolus fracture:** Nonsurgical treatment of isolated distal fibular fractures without an injury to the medial side (SER II) is successful in a majority of cases. The fracture can be treated with casting or an ankle orthosis with early weight bearing and early ankle range of motion.
- **Bimalleolar and trimalleolar fractures:** Displaced bimalleolar and trimalleolar fractures are best treated with ORIF to re-establish the integrity of the ankle mortise and joint congruity. Fixation of the posterior malleolus is indicated with an articular surface involvement of >25% and with persistent posterior talar subluxation.

- **Associated soft tissue ligamentous injuries**
  - **Deltoid ligament disruption:** Such injuries are nonsurgically treated if the medial clear space reduces anatomically with reduction and fixation of the lateral malleolus. Persistent medial widening demands inspection of the syndesmosis. After internal fixation of the lateral malleolus, external rotation stress radiographs should be re-recorded to confirm stability of the syndesmosis. If syndesmotic instability is present, indicative of a SER IV injury, this should be addressed as noted below.
  - **Syndesmosis:** Fixation of the syndesmosis is indicated when evidence of a diastasis is present. Fixation methods are evolving but start with anatomical restoration of both fibular length and the tibiofibular relationship at the mortise.

## Compartment Syndrome of Lower Limb

**Description:** Surgical emergency usually associated with fracture of the involved extremity. Acute compartment syndrome represents myoneural ischemia that is caused by elevated intracompartmental pressure. Exertional compartment syndrome is a related entity that involves athletes, but it does not represent an emergency.

**Presentation:** Can be initiated by several conditions, including fractures, crush injuries, severe contusions, or vascular injuries (see Fig. 58.5F); reported in almost every muscle compartment of the arms, legs, and trunk; most cases involve the legs. Classic symptoms are described by the five Ps: pain, pallor, pulselessness, paresthesia, and paralysis. The earliest and most reliable symptom is pain that is out of proportion with the injury; other symptoms are less reliable. Do not wait to see if other symptoms occur before making the diagnosis and initiating treatment.

**Physical examination:** Primary diagnosis is based on physical examination and not compartment pressure measurements. Firm compartments and severe pain are adequate to make the diagnosis. Possible loss of pulses distal to the compartment, pallor of the extremity, or nerve dysfunction in the form of either paresthesia or paralysis

Paresthesia is another relatively early sign (with pain) and should be carefully evaluated if present.

**Diagnostics:** Various commercial devices have been developed to measure compartment pressures in limbs; the pressure threshold that requires a fasciotomy is extremely controversial and should mainly be based on clinical examination.

**Treatment:** Surgical emergency requires surgical release of the fascia over the entire length of the involved compartment; both skin and fascia should be left open after release. Negative pressure wound therapy to aid in the management of fasciotomy wounds can be very helpful.

**Prognosis and return to sport:** Prognosis is highly variable and largely depends on the timing of the diagnosis. This is a limb-threatening condition with a high likelihood that the athlete will not be able to function at the same level as before the injury. Prognosis is favorable in patients who undergo an early fasciotomy. As noted previously, a higher muscular volume within the compartment may put the athlete at an increased risk of compartment syndrome. Possibility of an evolving compartment syndrome must be considered and dealt with after injury before the athlete is allowed to fly home after a competition.

## Open Fractures

**Overview:** Can be severe injuries that threaten the athlete's career, and possibly the athlete's leg; it is critical that every team physician understand certain key principles regarding the treatment of open fractures. The team physician should not hesitate to engage trauma surgeons, who have substantial experience with severe open fractures, to assist and provide the ideal treatment.

**Presentation:** Significant bleeding and an obviously open wound, or it may involve virtually no bleeding and only a small wound or abrasion (see Fig. 58.5C). Always assume a fracture is open until proven otherwise.

**Physical examination:** Remove all clothing and pads to allow careful evaluation of the skin; inspect for lacerations and abrasions. Open fractures should be classified according to the system developed by Gustilo and Anderson (see Fig. 58.5C).

**Diagnostics:** Orthogonal radiographs of the fracture are the primary diagnostic studies required. Compartment syndrome is more common with open fractures than with closed fractures.

**Treatment: Should be initiated on an urgent basis**; although present data suggest that several open fractures do well as long as they are treated within the initial 24 hours, optimally, the patient should be taken to the operating room as soon as the appropriate team and equipment can be assembled. Initial management of the wound, including thorough examination with gentle cleansing of any gross contamination, if possible, is key. Several open fractures that occur during athletic competitions are grossly contaminated with soil and grass; it is critical to perform an aggressive and thorough irrigation and debridement in order to avoid osteomyelitis. Several new treatment options have been developed that may be useful with the most severe open fractures:

- The use of negative pressure wound therapy has been reported to be associated with a decreased incidence of osteomyelitis following severe open fractures (level I data).

**Prognosis and return to sport:** Prognosis for a severe open fracture is driven by both the type of fracture and the open wound. It may be a career-ending injury, and the athlete and coach need to understand that. However, with aggressive treatment and incorporating contemporary treatment methods, completely recovery is possible, and the athlete can thus successfully resume his or her athletic career.

# RETURN TO SPORTS AFTER EXTREMITY FRACTURE

## General Principles

- Return to sport has to be individualized based on the type of fracture, fixation method, healing, and type of sport activity and position played.
- Returning to a noncontact overhead sports such as tennis is much different than returning to a collision sport such as football.
- The individual and the team physician must weigh the benefits of an early return against the short- and long-term risks of re-injury.
- The overriding principle is the health and well-being/safety of the athlete.
- The sport-specific evaluation on the field or court is more important than any test in the office environment. Feedback from the athletes themselves is also crucial.

### Upper Extremity

- In most cases, the time to return to sport with an upper extremity fracture is much less than that with a lower extremity fracture.
- A certain degree of cardiovascular fitness, speed, and agility can all be maintained from the beginning.
- In addition, the injury can be protected with taping, padding, splinting, and bracing.
- Return to sport is often a multiple of the time required for fracture union (0.5–1.0 times the fracture healing duration).
- The lower extremity rule would apply to an overhead athlete.

### Lower Extremity

- Rehabilitation starts immediately, but because there is a period of limited weight bearing, return of full strength, speed, and agility takes longer.
- As a general rule, time taken to return to sport is a multiple of the time required for fracture union (1.5–2.0 times the fracture healing duration).
- Rehabilitation goes through three major phases: protected phase, partial protected phase, and unprotected phase.
- Fixation method and fracture healing determine how long each phase lasts and must be guided by experience, clinical examination, and radiographs.
- Biologically friendly anatomical reduction and biomechanical sound initial fixation allows faster progression to the unprotected phase of rehabilitation and shortens the return-to-play duration.

## Hardware

**Hardware left in situ:** Playing with hardware must be individualized based on the type of sport (collision, contact, or noncontact), type of hardware, location of hardware, and stress concentration.

**Type of hardware:**

- **Plate:** Stress shielding occurs under a plate, and an area of stress concentration exists at the end of the plate. Risk of fracture at the end of the plate goes up in collision sports such as football. Protective padding to distribute loads over a broader surface area is advisable.
- **Intramedullary nail:** Minimal stress shielding occurs with a load-sharing device such as an intramedullary nail and stress concentration is better tolerated in the metaphyseal and epiphyseal regions at the ends of the nail. Minimal protection is required after fracture union.

**Hardware removal:** Most hardware is left in place until the athlete finishes competitive sports. Symptomatic hardware, particularly intramedullary nail interlocking screws, can be removed in the off-season.

- Re-fracture after plate removal occurs through screw hole sites. An extended period of protection may be required after removal, particularly with forearm fractures.
- Re-fracture after screw removal for fifth metatarsal base fractures has been reported, and screws should be left in place until the athlete has finished competitive sports.
- Intramedullary devices pose minimal risk and are left in place until competitive sports are finished.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# STRESS FRACTURES

# 59

Christopher C. Kaeding • Kurt P. Spindler

## GENERAL PRINCIPLES

### Definition

Stress fractures are fatigue-failure injuries of the bone affecting physically active people, including military recruits, track and field athletes, and ballet dancers. With the increased role of exercise for elderly people and patients with chronic disease, stress fractures should not be overlooked in nontraditional populations.

### Etiology

- Stress fractures are overuse injuries that present over a continuum of fatigue failure of the bone, from microfracture to complete structural failure. Excessive repetitive stress alters the balance of bone remodeling. Bone formation (osteoblastic activity) lags behind resorption (osteoclastic activity). Stress can be compressive, tensile, or rotational.
- Initially, increased osteoclastic activity at a site of stress leads to a more porous, weakened cortex. Above a certain threshold, additional stress cycles lead to the development of microscopic cracks, further decreasing bone strength. Continued repetitive stress leads to microfracture propagation and coalescence, resulting in a stress fracture. With further stress, frank fracture and displacement can occur (Fig. 59.1).

### Epidemiology

- Individual sports place stress on specific anatomic sites, which are at increased risk of stress fracture. Site specificity is determined by the athletic population (Table 59.1).
- Certain sports are more commonly associated with stress fractures: **running** (**69%**), fitness class (8%), racket sports (5%), basketball (4%), and other sports (14%).
- Female military recruits have 10 times the fracture risk of males; female athletes have 3.5 times the fracture risk of males.
  - Risk is activity dependent: higher risk for female runners and gymnasts.
  - Females' higher risk may be related to underlying menstrual irregularities and associated decreases in bone density (e.g., with female athlete triad) or anatomic and biomechanical factors specific to females.
- The most common bones injured, reported as percentage of all stress fractures: tibia (49.1%), tarsals (25.3%), metatarsals (8.8%), femur (7.2%), fibula (6.6%), pelvis (1.6%), sesamoids (0.9%), and spine (0.6%); bilateral injuries in 16.6% of cases
- Age relationship: Femoral and tarsal fractures more common in older patients; tibial, fibular, and upper extremity fractures more common in younger patients

## PROTECTIVE AND RISK FACTORS

See Table 59.2.

### Bone Characteristics

- **Composition:** Mineral deposition around collagen matrix; the bone resists compression; the collagen matrix (connective tissue) resists tension
- **Bone remodels in response to stress** (Wolff's law).
- Allows protective increases in cortical thickness, density, and diaphyseal diameter (Fig. 59.2)
- Remodeling is a function of number, frequency, and duration of loading cycles. It is influenced by strain volume, application rate, and duration per cycle.
- Osteoclasts activated by stress results in increased activity over 30–45 days, resulting in increased bone porosity.
- Osteoblasts migrate into porous areas and build matrix. This process begins approximately 30 days after stress and culminates with new bone formation over approximately 180 days.
- If a weakened bone is subject to additional stress, **osteoclastic activity can overwhelm osteoblastic activity, resulting in further weakened bone and microfractures.** Each load cycle may propagate microfractures until symptomatic stress fracture results. Increased microfractures have been demonstrated in areas of bone porosity.
- **Bone geometry** may determine the risk.
  - Larger and higher-density bones more resistant to fatigue fractures
  - Long bones with increased diameter resist bending in response to load. During basic training, recruits with tibial stress fractures had significantly smaller tibial widths and cross-sectional areas than those without stress fracture.

### Soft Tissues (Intrinsic Factors)

- Good muscle strength is protective. Muscle contractions attenuate ground reaction forces developed at impact during running and jumping.
- Bone strain increases as muscles fatigue.
- Contraction of strong opposing muscle groups on a single bone may produce tension across the bone and promote stress fracture.
- No relationship between stress fracture and muscle size or flexibility
- General fitness is protective. Military recruits with higher activity levels before enlistment developed fewer stress fractures during basic training.

### Endocrine

- Low testosterone or estrogen levels can increase the risk of stress fractures.
- Oligomenorrheic or amenorrheic female athletes are at a risk secondary to decreased estrogen levels and increased osteoclastic activity.
- High-intensity training may suppress menses.
- Delayed menarche, often observed in athletes, may result in decreased bone density but probably does not increase the risk of stress fractures.
- Cigarette smoking lowers estrogen levels in a dose-dependent manner, resulting in higher osteoclastic activity.

### Nutritional

- Stress fractures are associated with a lower fat intake, a lower caloric intake, eating disorders, and weight <75% of the ideal body weight.

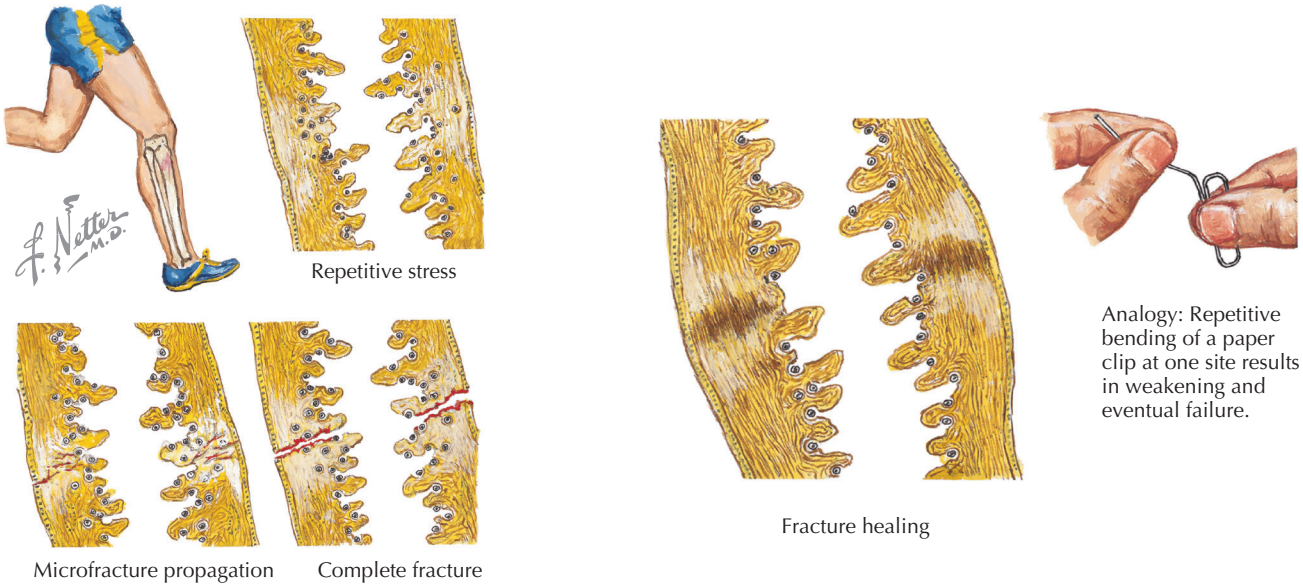



**Figure 59.1. Etiology of Stress Fracture.** Site specificity of stress fractures in track and field athletes is determined by the anatomic stress demanded by the sport.

**TABLE 59.1 STRESS FRACTURE SITES ASSOCIATED WITH SELECTED SPORTS**

| Track | Football | Basketball | Gymnastics | Softball (Fast-Pitch) | Dance | Running |
|---|---|---|---|---|---|---|
| Navicular | Tibia | First rib | Pars interarticularis | Rib | Metatarsals | Tibia |
| Tibia | Pars | Tibia | Radius | Ulna | Fibula | Fibula |
| Metatarsals | 5th metatarsal | 5th metatarsal | Midfoot | Humerus | Pars interarticularis | Metatarsals |
| Fibula | | Tarsal | Navicular | | Tibia | |

**TABLE 59.2 ETIOLOGIC FACTORS FOR STRESS FRACTURES**

| Intrinsic Factors | Extrinsic Factors |
|---|---|
| Alignment abnormalities | Overt |
| Femoral neck anteversion | Continued self-abuse |
| Pronation/supination | Inappropriate training |
| Tibial torsion | Inappropriate technique |
| Leg length discrepancy | Inappropriate equipment |
| Muscle imbalance | Harsh environment |
| Muscle weakness | Covert |
| Flexibility | Joint instability |
| Genetic predisposition | Extrinsic pressure |
| Aging/hormonal | Biomechanical fault |

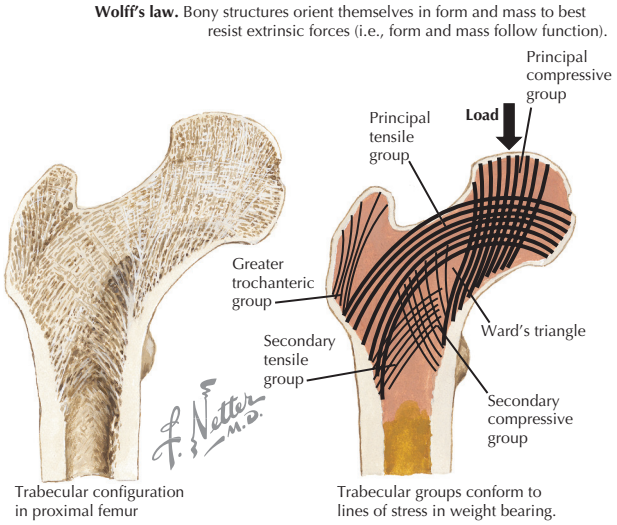



**Figure 59.2.** Bone architecture in relation to physical stress.

- Calcium intake correlates with bone density; low calcium intake does not correlate with stress fractures.

## Training Intensity

- Training errors include rapidly escalating frequency, duration, and intensity of training; 60% of running injuries are associated with training errors.
- Stress fractures are increased during the initial 2 weeks after increased training intensity, with increases >30% in duration or intensity over a single season, and in freshman runners adjusting to collegiate training demands.
- Multiple factors compound the risk
  - Independent of risk associated with menstrual dysfunction, athletes training >5 hours/day have a 6-fold increased risk of stress fractures.
  - A dancer who is both amenorrheic for >6 months and training >5 hours/day is 93 times more likely to develop a stress fracture than an eumenorrheic counterpart who trains less.

### Extrinsic Factors

- **Surface**
  - **Hard running surfaces** increase impact forces.
  - Unidirectional running on **cambered track surfaces** may predispose to injury.
  - Large epidemiologic studies reveal no association between stress injury and running surface.
- **Environment** (e.g., hills or uneven terrain)
- **Technical faults in training or performance—**may predispose to stress injury

## DIAGNOSIS

### Time to Diagnosis

- It takes an average of 5–16 weeks to diagnose a stress fracture. Delayed diagnosis can be detrimental, particularly with high-risk stress fractures.

### History

- Maintain a high index of suspicion.
- Certain affected anatomic areas are particularly challenging to diagnose.
- Recall of specific detailed historical facts may be challenging for athletes.
- Recent change in activity level over the past 2 months and preseason training
- Recent change in equipment or playing surface
- Activities added to already demanding physical performance
- **Insidious onset of pain,** usually initially after activity; with fracture progression, pain moves earlier into activity and may eventually occur with minimal activity (e.g., walking) or even at rest.
- Vague pain in affected region, occasionally localized
- Pain relief with rest and/or reduced activity
- Prior stress fractures
- **Females:** Menstrual irregularities, recent weight changes, eating disorder history or behaviors, and nutrition
- **Older athletes,** particularly **females:** osteoporosis risk factors (e.g., smoking, family history, corticosteroid use, body weight, Caucasian race, menopause, and hormone replacement)

### Physical Examination

- Point tenderness, edema, warmth, and palpable callus in certain cases (Fig. 59.3); in areas where bone not easily palpated, such as femoral neck, gentle range of motion (ROM) may elicit pain
- Tenderness with percussion or with sound waves (tuning fork or ultrasound)
- Specific tests:
  - **Stork test or one-legged standing hyperextension test:** Back extension with resultant hip flexion while standing on one leg increases pain in pars interarticularis stress fractures.
  - **Fulcrum test:** Performed by placing fist or arm of examiner under suspected fracture site and applying "bending" force to distal extremity while proximal extremity is kept relatively immobilized by anatomic restraints; positive test results in pain over the fracture site; commonly used to evaluate femoral shaft stress fractures but may be used in other "long bone" areas
  - **Hop test:** Hopping on affected leg reproduces pain at the fracture site, usually with femoral shaft stress fractures.
  - **Tuning fork test:** Vibrating tuning fork over fracture site results in pain at the fracture site; high rate of false positives

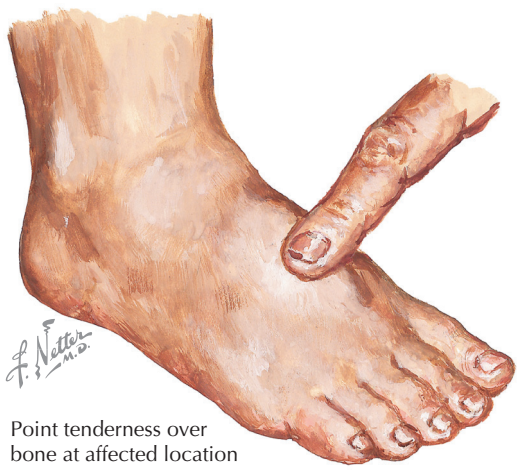



**Figure 59.3.** Physical examination of stress fracture.

### Radiologic Studies

- **Plain radiographs** usually negative in the early phase, particularly during initial 2–3 weeks; two-thirds of initial radiographs are negative, but half ultimately prove positive; radiographs are specific but not sensitive.
- **Ultrasound:** Not reliable for diagnosis; several studies in progress
- **Bone scan:** Approximately 100% sensitive but not specific; not helpful for guiding return to play; uptake on bone scan normalizes over 12–18 months, often lagging behind resolution of clinical symptoms; particularly useful in tarsal, femur, pelvis, and tibial plateau fractures; helps differentiate stress fractures from periostitis (shin splints). Stress fractures are **usually positive in all phases of triple-phase technetium bone scan** (angiogram, blood pool, delayed). Periostitis is often negative in the angiogram and blood-pool phase and positive in the delayed image phase.
- **Single-photon emission computed tomography (SPECT):** More specific than planar bone scan; particularly helpful in detecting stress fractures of the pars interarticularis, pelvis, and femoral neck
- **Computed tomography (CT):** Delineates bone well, useful when diagnosis is difficult (e.g., tarsal navicular stress fractures); helpful with spinal or linear stress fractures
- **Magnetic resonance imaging (MRI):** Superior sensitivity and specificity over bone scan and CT for soft tissue abnormalities and edema; may delineate early injury. Findings include periosteal or marrow edema. MRI may be highly useful with certain stress fractures (such as hip and pelvis).

## CLASSIFICATION

- Reproducible classification system of stress fractures is important for accurate communication between clinicians and the study of stress fractures.
- Classification system should have implications for the treatment and prognosis of a stress fracture.
- Table 59.3 outlines a reproducible and generalizable classification system.

## GENERAL TREATMENT

- Because they are overuse injuries, most stress fractures respond well to relative rest and gradual reintroduction of sport. High-risk stress fractures demand rapid diagnosis and specific intervention to avoid complications.
- Stress fractures frequently heal in 6–8 weeks (usually low-risk), but certain fractures (mostly high-risk) heal more slowly over ≥3–4 months.

**TABLE 59.3 CLASSIFICATION OF STRESS FRACTURES**

| Grade | Pain | Imaging Findings (Radiograph, Bone Scan, CT, MRI) |
|---|---|---|
| I | (-) | Evidence of stress injury |
| II | (+) | No fracture line |
| III | (+) | Fracture line |
| IV | (+) | Displaced |
| V | (+) | Nonunion |

### Suggested Phases of Treatment

- **Phase I:** Pain control, usually 10–14 days
  - Controversial to use nonsteroidal anti-inflammatory drugs (NSAIDs) at analgesic doses. Use with caution because prolonged high doses of NSAIDs may inhibit bone healing.
  - Physiotherapy (may include bone stimulator)
  - Flexibility and strengthening
  - Complete or relative rest depending on fracture site and severity of symptoms
  - Bracing may speed return to play in specific cases (e.g., long-leg pneumatic splint with anterior buttress in tibial shaft fractures)
- **Phase II:** Reintroduction of activity; may last for several weeks depending on the location and type of stress fracture; initiation of phase II varies with patient symptoms and depends on size and type of stress fracture.
  - Continue phase I treatment
  - Maintain aerobic fitness through pool running and cycling; newer elliptical trainers may also be helpful.
  - Gradually add impact activity, altering surface or equipment if possible. Run short distances on grass or soft surfaces and gradually increase running time.
  - Resume sports-specific training in noncompetitive settings.
  - Work out on alternating days and maximize rest between bouts of exertion.
- **Phase III:** Preparation for return to competition
  - Increase sports-specific plyometric conditioning drills. Add running drills, cutting drills, and selected skill work.
  - Begin lower-level competitive challenges.

## SPECIFIC STRESS FRACTURES

### Upper Extremity

Although much less common than in the lower extremities, stress fractures of the upper extremities should be included in the differential diagnosis for athletes who perform repetitive throwing, swinging, or overhead activities. Gymnasts and cheerleaders who use the upper extremities for weight bearing and twisting are at a risk of stress fractures below the elbow. Young athletes and throwing athletes (e.g., goalkeepers or javelin throwers) develop stress fractures of the humerus and shoulder girdle. Swinging athletes (golf and rowing) are at a risk of rib fractures.

#### Humerus

**Description:** **Proximal** and **medial epicondyle** fractures seen in younger throwing athletes and gymnasts. **Midshaft** fractures seen in adult throwing athletes and workers who perform heavy lifting

**History:** Pain with throwing or lifting heavy weights; may involve entire upper arm

**Examination:** Normal or pain on palpation or resisted motion

**Treatment:** Rest; immobilization, such as fracture brace; 6–8 weeks for healing; correction of biomechanical and technical faults

**Figure 59.4.** Lateral elbow, olecranon stress fracture *(arrow)*.

#### Ulna

**Description:** Reported in baseball and softball pitchers, rodeo riders, bowlers, golfers, and volleyball players

**History:** Pain over ulnar shaft during and after activity; fracture location varies by activity. Fractures develop proximally in volleyball players secondary to repetitive and explosive wrist flexion. Fractures occur distally in softball players (underhand activity). Midshaft stress fractures reported in tennis; pronation with backhand stroke at ball strike and follow-through are painful

**Examination:** Pain, edema, and local heat over involved areas; painful pronation and supination

**Radiographic studies:** Radiographs typically helpful, shows periosteal reaction; bone scan or MRI used to confirm. MRI may demonstrate edema in interosseous membrane.

**Treatment and return to play:** Rest from activity, resume gradual activity when pain free. Extension block splints used sometimes; correction of biomechanical or technical faults; in all reports, return to play was in 4–6 weeks

#### Olecranon

**Description:** Occurs in throwing athletes; caused by recurrent valgus extension overload; young gymnasts may injure olecranon physis.

**History:** Pain with elbow extension/throwing

**Examination:** Point tenderness and pain with triceps extension against resistance

**Radiographic studies:** Radiographs may be negative. Nonunion risk is higher if radiograph shows sclerosis around area of lucency. Fracture site is under tension. Radiographs demonstrate unique transverse radiolucency extending from posterior nonarticular to articular surface (Fig. 59.4)

**Treatment:** Short-term immobilization to reduce triceps pull; surgery may be required for nonunion.

#### Radius

**Description:** Uncommon; reported in military personnel, cheerleaders, gymnasts, tennis players, volleyball players, softball players, pool players, and cyclists

**History:** Pain in shaft of radius with aforementioned activities, particularly with wrist supination

**Examination:** Pain over involved area

**Radiographic studies:** Radiographs show periosteal thickening and occasionally bowing; bone scan or MRI confirms diagnosis.

**Treatment:** Rest for 6 weeks; repetitive wrist weight-bearers (e.g., gymnasts or cheerleaders) may require longer rest

## Shoulder Girdle and Trunk

### Clavicle

**Description:** Cases reported in javelin throwers and springboard divers; mechanism of injury hypothesized to be repetitive deltoid and pectoralis major contractions during javelin throw and repeated water entry (diving) with wrists extended and forearms pronated
**History:** Insidious onset of pain over clavicle
**Examination:** Pain reproduced with abduction of the shoulder above horizontal plane of shoulder
**Radiographic studies:** Radiographs show periosteal reaction. Bone scan or CT is used to confirm diagnosis.
**Treatment:** Rest from activity; return to play in 8 weeks

### Scapula

**Description:** Stress fracture of coracoid process reported in female trapshooters secondary to repetitive impact of rifle butt; stress fractures at other scapular sites reported in gymnasts, joggers who carry dumbbells (superomedial scapula), and football players (associated with weightlifting; acromion)
**History:** Gradual onset deep aching in shoulder
**Examination:** Pain with palpation of coracoid process and bicipital groove; painful resisted adduction and forward flexion of arm
**Radiographic studies:** Axillary view necessary for plain radiographic diagnosis; West Point axillary and modified oblique views helpful for acromial stress fractures; MRI or bone scan confirms diagnosis
**Treatment:** Rest from trapshooting for 6 weeks; at 6 weeks, patient is usually pain free with full ROM and can gradually resume shooting
Other fracture sites: return to play in 8 weeks with rest; consider butt modification or padding when returning to play.

### Ribs

**Description:** First-rib fracture most commonly seen in baseball pitchers and basketball players; reported in weightlifting, tennis, table tennis, rugby, judo, gymnastics, and ballet; other rib fractures seen in softball (pitchers), golf, tennis, and rowing; diagnosis often delayed because misdiagnosed as back strain; typically posterolateral, although anterolateral and rib neck stress fractures have been reported in rowers; possibly related to serratus anterior fatigue in golfers
**History:** Insidious onset, but occasionally presents acutely without preceding symptoms
**Examination:** Pain with arm motion over supraclavicular area (first rib) or over the affected area; pain with trunk rotation (lower ribs); serratus anterior weakness
**Radiographic studies:** Radiographs often negative; usually positive if fracture is over a broad, flat, and thick portion of the first rib. Other ribs are difficult to diagnose using radiographs; bone scan or MRI helpful to confirm diagnosis
**Treatment:** Rest, avoidance of overhead and swinging activity, maintenance of aerobic fitness, general conditioning and strengthening of serratus anterior; gradual return to play when pain free

## Spine

### Spondylolysis: Pars Interarticularis Stress Fractures

**Description:** Most common in athletes undergoing repetitive hyperextension of spine (gymnasts, cheerleaders, divers, and weightlifters); L4 and L5 are most commonly affected vertebrae levels; common cause of pediatric low back pain. In orthopedic referral population, 47% of pediatric patients with low back pain had spondylolysis, compared with only 5% of adults
**History:** Athletes usually involved in certain repetitive back **extension load** activities; insidious onset of low back pain; patient ultimately complains of significant back spasms (often misdiagnosed as lumbar strain); short periods of rest may temporarily relieve the pain, but return to activity results in immediate exacerbation of symptoms
**Examination:** May have clinical hyperlordosis; pain to palpation over affected vertebrae; pain and muscle guarding with one- and two-leg standing trunk extension test (one-leg usually worse), trunk rotation and extension, hip extension test (prone), trunk extension test (prone), and combined hip and trunk extension test (prone); neurologic examination usually normal; occasionally may have associated radiculopathy
**Radiographic studies:** Radiographs have low sensitivity. Anteroposterior (AP), lateral, bilateral, and oblique views; if positive, classic defect of "collar" on neck (pars interarticularis) of Scotty dog is seen on oblique views; falling out of favor because of low sensitivity and high radiation. SPECT scan has a greater sensitivity and is becoming the gold standard for diagnosis. Thin-cut CT scan (1.5–2-mm cuts) may help identify the extent and age of fracture, partial versus complete, and sclerosis (Fig. 59.5). Combination of SPECT and CT findings may help determine the likelihood of healing and may help design the treatment protocol.
**Treatment:** Somewhat controversial; initially, modify activity and avoid back hyperextension; some consider nonrigid bracing if pain persists. At 2–4 weeks, begin physical therapy; if pain present at 4 weeks, thoracolumbosacral orthosis (TLSO) or low-profile antilordotic Boston brace is an option to unload posterior elements and prevent hyperextension. Treat until patient is symptom free. Healing can take 3–6 months and may not correlate with symptoms. Consider CT scan to assess healing. Consider surgical spinal evaluation if persistent pain after rigid bracing, particularly if neurologic symptoms appear or progress.
**Return to play:** As early as 8 weeks if patient remains pain free at rest, in hyperextension, and while performing aggravating activities

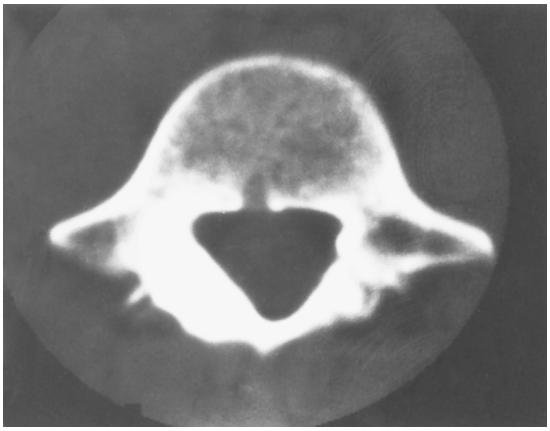


**Figure 59.5.** Axial CT image, pars interarticularis stress fracture with sclerosis.

## Pelvis

**Description: Pubic ramus** usually involved; uncommon; seen almost exclusively in women, military recruits, and long-distance runners or joggers
**History:** Pain in the inguinal, perineal, or adductor region that is relieved by rest; seen in female British Army recruits forced to match official 30-inch stride length while marching; when stride length decreased to 27 inches, no further stress fractures were reported, suggesting increased stride length may promote biomechanical stress at the pubic ramus

**Examination:** Antalgic gait, full ROM, pain over pubic ramus, and positive "standing sign" (inability to stand unsupported on affected side)
**Radiographic studies:** Radiographs initially negative; late in course, may show abundant callus; bone scan or MRI needed for early diagnosis
**Treatment:** Brief nonweight-bearing and rest from running; healing variable, typically 3–5 months

## Sacral Stress Fractures

**Description:** Fatigue fractures reported mainly in runners; high index of suspicion required; insufficiency fractures more common in osteoporotic bone (elderly)
**History:** Vague, poorly localized pain in the gluteal or groin area; rapid increase in mileage or training schedule often associated
**Examination:** Positive figure-4 (flexion, abduction, external rotation [FABER]) test and hopping test; normal ROM, but deep groin pain at extremes of motion
**Radiographic studies:** Radiographs usually negative; bone scan or MRI used for diagnosis
**Treatment:** Cessation of running, protected weight bearing, and relative rest lasting from 6 weeks to 8 months

## Femoral Stress Fractures

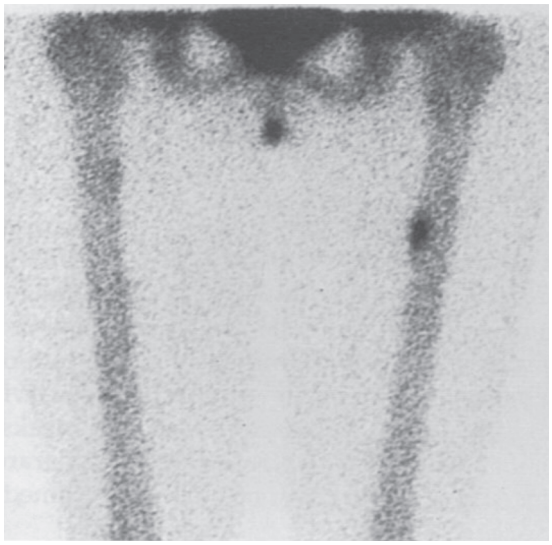
Femur sites most commonly involved with stress fractures, from most to least common: shaft, lesser trochanter, intertrochanteric region, neck, and greater trochanter; missed diagnosis of a femoral neck fracture can lead to a high morbidity

### Femoral Neck

**Description:** Seen most frequently in runners, dancers, and military recruits; diagnosis is typically delayed 5–13 weeks. Severe complications arise with progression to complete fracture and displacement. Complications may include avascular necrosis, nonunion, varus deformity, and chronic pain. **Superior** neck fractures are under tension and are a high-risk area for progression to complete fracture. **Inferior** neck fractures are compression-sided and are often conservatively managed.
**History:** Earliest sign: 87% report inguinal or anterior groin pain; aching pain precipitated by weight-bearing activities: 40% reported symptoms after long run, between 6–8 weeks of training. Symptoms appear with increase in training schedule; diagnosis often delayed
**Examination:** Antalgic gait (22%); pain with palpation in groin, hip, or anterior thigh (70%); pain at extremes of hip ROM (79% of 39 patients in one study); subtle limitation of flexion and internal rotation
**Radiographic studies:** Radiographs have a high false-negative rate, particularly early; bone or SPECT scan for early diagnosis; false negatives reported for up to 12 days after fracture. MRI is becoming more popular and is a sensitive study for identifying early marrow edema, which typically resolves in 8–12 weeks.
**Treatment** (Blickenstaff criteria modified by Fullerton): Complications in up to 30% of patients; return to play may be delayed for up to 2 years

- **Compression-sided fractures** (Blickenstaff type I): inferior neck
  - Type Ia: Callus without fracture line and positive bone scan; managed conservatively with nonweight-bearing and/or bed rest; gradual activity progression when pain free; weekly radiographs until patient can walk without pain using a cane
  - Type Ib: Definite fracture line but no displacement; treat with nonweight-bearing, or surgery; serial radiographs
- **Tension-sided fractures** (Blickenstaff type II): superior neck (Fig. 59.6)

AP hip, superior femoral neck (tension-type fracture).



Bone scan, increased uptake femoral shaft.

**Figure 59.6.** Femoral stress fractures.

  - A positive bone scan without a visible fracture line on radiographs is treated with bed rest or nonweight-bearing and crutches; serial radiographs to look for developing fracture lines
  - If fracture lines present, higher risk of displacement, avascular necrosis, nonunion, and malunion; early diagnosis and surgical management usually required; if no displacement, some experts advocate bed rest as first-line treatment in lieu of immediate surgery
- **Displaced** (Blickenstaff type III): surgical fixation recommended

### Femoral Shaft Stress Fractures

**Description:** Seen mostly in runners, particularly females; most common location is at the junction of proximal and middle thirds of the femoral shaft
**History:** Sudden increases in frequency, intensity, or duration; pain with running progresses to pain with activities of daily living
**Examination:** Antalgic gait (22%); normal ROM (94%); pain with palpation in groin, hip, or anterior thigh (70%); hopping on affected leg reproduces pain (hop test); positive in 70%; positive fulcrum or "hanging leg" test
**Radiographic studies:** Early radiographs negative; callus and lucent fracture line in 2–6 weeks; bone scan or SPECT scan for early diagnosis (see Fig. 59.6); MRI is much more sensitive; clearly identifies bone edema, which typically resolves in 8–12 weeks
**Treatment:** Conservative management of shaft stress fractures is successful; first-line interventions include protected weight-bearing with crutches for 1–4 weeks (length of time depends

on resolution of pain); activity modification; maintenance of aerobic fitness, skill, and strength; if pain free during day-to-day activities at 14 days, begin rehabilitation with low-impact, minimal weight-bearing exercise (cycling, swimming, pool running); time to full recovery varies, but reported as 5–10 weeks from diagnosis; resumption of athletic activity may take 8–16 weeks.

## Knee and Lower Leg Stress Fractures

### Patella

**Description:** Rare; reported in basketball, soccer, and high jump; may be longitudinal or transverse; reported after anterior cruciate ligament (ACL) and patellar tendon graft repair in patients with cerebral palsy and in young athletes

**History:** Anterior knee pain, worse with jumping, may have had prior bone tendon bone graft harvest for ACL reconstruction

**Examination:** Pain to palpation, pain with resisted knee extension

**Radiographic studies:** Radiographs may show definite fracture lines; must differentiate from bipartite patella; bone scan; MRI clarifies diagnosis and identifies bone edema

**Treatment: Transverse fractures** prone to displacement; nondisplaced fractures are treated with long leg or cylinder cast immobilization with the knee in full extension for 4–6 weeks, followed by quadriceps rehabilitation. Displaced fractures are treated with open reduction and internal fixation (ORIF). **Longitudinal fractures** occur in lateral patellar facet; if displaced, excise lateral fragment

### Proximal Tibia Fractures

**Description:** Infrequent; incidence unknown

**History:** Pain in anteromedial aspect of proximal tibia; weight-bearing precipitates pain

**Examination:** Localized tenderness and edema; need to differentiate from pes anserinus tendinitis, bursitis, and saphenous nerve entrapment

**Radiographic studies:** Acute stress fracture often has no radiographic findings. MRI is the most sensitive study (Fig. 59.7).

**Treatment:** Modification of offending activity usually by decreasing frequency and duration

### Tibial Shaft Stress Fractures

**Description:** Need to differentiate between compression stress fracture and tension stress fracture ("dreaded black line")

- **Compression stress fractures:** MRI suggests continuum between medial tibial stress syndrome and stress fracture, with varying degrees of periosteal edema; common sites include proximal or distal third tibia, posteromedial tibia (see Fig. 59.7); common in runners
- **Tension stress fractures:** Located in anterior or anterolateral cortex in central third of tibia; common in ballet dancers and jumpers

**History:** Initially, pain occurs after activity; later pain occurs with activity and activities of daily living

**Examination:** Localized pain, anterior or medial tibia; edema, palpable periosteal thickening, and pain with percussion; positive "tuning fork test" (beware of false negative; useful if pretest probability is high)

**Radiographic studies:** Radiographs may be positive if symptoms have persisted for 4–6 weeks; bone scan shows fusiform uptake, which differs from linear uptake of medial tibial stress syndrome. False-negative bone scans have been reported. MRI is more sensitive; used for grading and prognosis for return to play (grades 1 and 2 less symptomatic, return to sport 4–6 weeks; grades 3 and 4 often need more time for healing)

- Grade 1: mild periosteal edema (T2), normal marrow (T1, T2)
- Grade 2: moderate periosteal edema (T2), marrow edema (T2)
- Grade 3: severe periosteal edema (T2), marrow edema (T1, T2)
- Grade 4: severe periosteal edema (T2), marrow edema (T1, T2), distinct fracture line visible

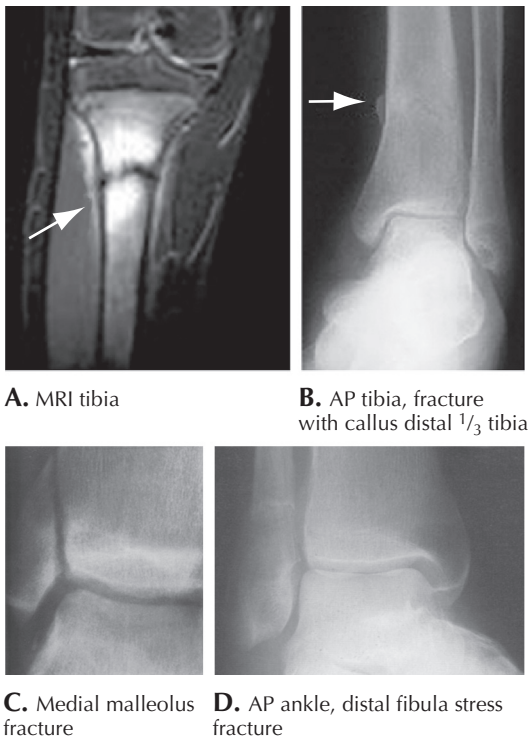
**Treatment:** Control pain, stop running, and use crutches if necessary

- **Compression lesions** may take 2–12 weeks to heal. Return to play is faster with three-panel, long-leg stirrup brace. Correct amenorrhea in females. Calcium supplementation recommended; correct biomechanical faults (consider foot orthotics); in general, return to play guided by MRI grade:
  - Grade 1: return to play in 2–3 weeks, after rest
  - Grade 2: return to play in 4–6 weeks
  - Grade 3: return to play in 6–9 weeks (initially painful with ambulation)
  - Grade 4: return to play in 12 weeks, after casting for 6 weeks and 6 weeks of nonimpact rehabilitation (single case)
  - When pain free: cross-training, nonimpact, and reduced-impact aerobics training; progress to alternating-day graded return to running
- **Tension lesions** achieve faster return to play with intramedullary rod. Conservative treatment may heal in 6 months but has a high rate of recurrence; may progress to complete fracture if missed

**Prevention** is the best treatment: strength and flexibility of gastrocnemius and soleus; correction of poor running technique; correction of biomechanical issues (e.g., pronation or pes planus) at preparticipation evaluations, particularly with history of stress fracture

### Medial Malleolar Stress Fractures

**Description:** Extend obliquely from plafond; inherently unstable and prone to nonunion; high index of suspicion key to early diagnosis



**A.** MRI tibia **B.** AP tibia, fracture with callus distal $^1/_3$ tibia

**C.** Medial malleolus fracture **D.** AP ankle, distal fibula stress fracture

**Figure 59.7.** Knee and lower leg stress fractures.

**History:** Insidious onset medial ankle pain, increased with exercise and relieved by rest
**Examination:** Tender over medial malleolus, ankle effusion
**Radiographic studies:** Radiographs usually negative early; bone scan or MRI usually required for diagnosis
**Treatment:** MRI or bone scan positive, no fracture on radiographs: stirrup immobilization for 4–6 weeks. Radiographs or CT demonstrate the fracture; consider ORIF (see Fig. 59.7). Nonunion requires bone graft and screw fixation.

### Fibular Stress Fractures

**Description:** Most common in distal third just proximal to distal tibiofibular syndesmosis
**History:** Limp, swelling
**Examination:** Point tenderness, localized edema; foot overpronation with hindfoot valgus common as lateral malleolus is at a risk
**Radiographic studies:** Radiographs not positive for 3–4 weeks (see Fig. 59.7); bone scan can assist with diagnosis.
**Treatment:** Conservative; for distal fractures, pneumatic ankle brace may be helpful.

## Stress Fractures in Foot

### Calcaneus

**Description:** Seen in military recruits and runners
**History:** Insidious onset of heel pain with running
**Examination:** Positive heel squeeze test; may have positive hop test; edema
**Radiographic studies:** Radiographs show endosteal callus perpendicular to the long axis of calcaneus; bone scan, MRI, or CT to confirm diagnosis (Fig. 59.8)
**Treatment:** Rapid healing with conservative treatment, including relative rest and activity modification; brief period of nonweight-bearing if ambulation painful; return to activity in 3–4 weeks

### Tarsal Navicular Stress Fractures

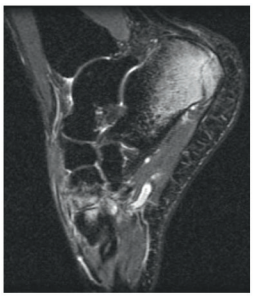
**Description:** Previously thought uncommon, now recognized in jumping and running athletes, soccer, basketball, and track athletes; vague symptoms often lead to delayed diagnosis. Complications include nonunion and chronic pain. Most fractures in sagittal plane and middle third secondary to poor blood supply; diagnosis requires a high index of suspicion and early imaging (bone scan, MRI)
**History:** Insidious-onset forefoot/midfoot pain, particularly with running and jumping; pain occasionally mild and usually relieved with rest
**Examination:** Pain at the "N spot" (dorsal aspect of the navicular); pain may be diffuse rather than localized.
**Radiographic studies:** Radiographs usually negative; bone scan or MRI can confirm diagnosis. CT or MRI helps determine the fracture site and the extent of healing (see Fig. 59.8).
**Treatment:** If no bicortical fracture, then nonweight-bearing cast immobilization for 6–8 weeks; if pain free (best guide to healing), a 6-week graduated program of weight-bearing activity. Consider foot orthotics on return to play. ORIF with bone graft for displaced fractures, failure of nonsurgical treatment, delayed union, and nonunion

### Metatarsal (MT) Stress Fractures

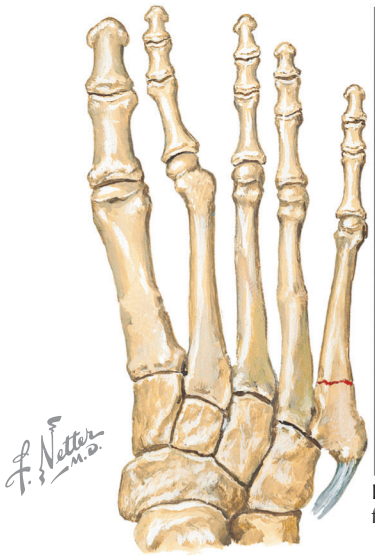
PROXIMAL DIAPHYSIS OF FIFTH METATARSAL

**Description:** Distal to the tuberosity and prone to nonunion; seen in basketball and, less commonly, football
**History:** Insidious onset of lateral foot pain, worse during and after activity; pain steadily worsens; occasionally, there is acute fracture.
**Examination:** Point tenderness distal to the tuberosity, usually in zone 3 (proximal diaphysis)
**Radiographic studies:** Radiographs usually show sclerotic changes around the fracture site. Bone scans are only occasionally necessary (see Fig. 59.8).
**Treatment:**

- Torg classification (proximal fifth MT fractures)
  - Acute: sharp fracture line with no sclerosis or cortical hypertrophy
  - Delayed union: history of previous injury, wide fracture gap, and intramedullary sclerosis
  - Nonunion: recurrent symptoms, wide fracture gap, and sclerosis
- In high-demand and recreational athletes, intramedullary screw has been demonstrated to permit faster return to play and decrease the nonunion rate. Usually weight-bearing


MRI calcaneus

CT foot, navicular stress fracture



Proximal diaphysis 5th metatarsal stress fracture

Base 5th metatarsal fracture x-ray

Distal $^1/_3$ 2nd metatarsal stress fracture with callus formation

**Figure 59.8.** Stress fractures in foot.

activity in 7–14 days, with training progression to completely unrestricted activity over 6–9 weeks; a small risk of fracture nonunion and fatigue fracture of the screw
- In nonathletes, a short-leg, nonweight-bearing cast for 6–8 weeks may be considered.

OTHER METATARSALS

**Description: First metatarsal MT:** 10% of MT stress fractures; associated with overpronation

**Second, third, and fourth MTs:** 90% of MT stress fractures; associated with running and training >20 miles/week; flatfeet (pes planus) increases impact stress to the MT. Most fractures in runners are distal (see Fig. 59.8). In ballet dancers, fractures occur proximally and often involve medial second MT secondary to en pointe work.

**History:** Localized pain and edema; onset insidious, worse after increase in training

**Examination:** Pes planus or overpronation; point tenderness and localized edema over MT

**Radiographs:** Weight-bearing AP, lateral, and oblique radiographs; in dancers with second MT pain, consider internal and external oblique radiographs; usually positive; bone scan helpful if negative

**Treatment:** Rest and a stiff-soled shoe or low-tide cast boot; gradual reconditioning to repetitive stress (at 2–3 weeks), such as pool running performed first in chest-deep water and progressing to more shallow depths as symptoms allow; gradual progression to biking, then running. Consider foot orthotics. In distal first MT stress fractures, dorsal displacement may require casting with dorsal pressure to prevent lateral metatarsalgia. In dancers, proximal second MT stress fractures may progress to nonunion and must be aggressively managed with casting or ankle-foot orthosis for 8 weeks.

SESAMOIDS

**Description:** Difficult diagnosis; differentiate from sesamoiditis and bipartite and tripartite sesamoids; 30% of population have bipartite sesamoids.

**History:** Pain over the plantar aspect of first metatarsophalangeal (MTP) joint; pain with "toe-off"

**Examination:** Pain on palpation, pain with resisted first toe plantarflexion, and pain over sesamoids with stretch into extreme of dorsiflexion

**Radiographic studies:** Diagnosis of sesamoid stress fracture by plain radiographs is challenging, and additional imaging (e.g., bone scan or MRI) is often required to differentiate stress fracture from bipartite sesamoid. MRI helps identify marrow edema.

**Treatment:** Conservative, initially nonweight-bearing cast for 6 weeks to prevent dorsiflexion of the first ray; gradual return to sports over several months; consider foot orthotics after casting; rarely surgical excision for delayed union or chronic pain

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 60 FOOT PROBLEMS

Kenneth J. Hunt • Melissa D. Koenig • Thomas O. Clanton

## GENERAL PRINCIPLES

**Overview:** Injuries and disorders of the foot can impose considerable dysfunction in an athlete. While most foot problems will improve with appropriate care, a clear understanding of normal anatomy and physical examination findings is vital to recognize injuries and abnormal processes to prevent worsening and long-term damage.

### Anatomy and Physiology of the Foot

**Bones:** Normal bony architecture includes 28 bones (7 tarsals, 5 metatarsals, 14 phalanges, and 2 hallux sesamoids) (Fig. 60.1A).
**Accessory bones:** Common accessory bones that can create symptoms in athletes are the accessory navicular (Fig. 60.1B), os trigonum, and os peroneum.
**Muscular anatomy:** Most lower leg extrinsic muscles have tendinous attachments on the foot. Foot intrinsic muscles are important for normal function (Fig. 60.1C).
**Neurovascular structures:** Three primary arteries/veins provide blood supply and five primary nerves provide innervation to the foot (Fig. 60.1C).

### Sport- and Athlete-Specific Issues

**Overview:** Most foot injuries in athletes are caused by acute or repetitive trauma. Several intrinsic and extrinsic factors can increase the risk of injury.
**Intrinsic factors:** Foot position, joint laxity and stiffness, nutrition, fitness level, training regimen, biochemical deficiencies, age, and body mass index
**Extrinsic factors:** Coaching, technique, environmental factors, footwear and equipment, and safety hazards
**Running and jumping sports:** Increased risk of bone stress injuries, tendinopathies, and fasciopathies
**Impact/collision sports:** Increased risk of acute fractures, tendon ruptures, and joint injuries

## HISTORY

**Overview:** An athlete's recollection of injury onset, mechanism, and location is an important part of the diagnostic process.
**Mechanism:** Acute, chronic, or acute on chronic; position of the foot during injury; was a pop felt or heard?
**Severity:** Was the athlete able to ambulate on his/her own after the injury? Was there an obvious deformity, swelling, or discoloration?
**Location:** Ask the athlete to indicate "with one finger" the location of the injury, pain, or symptoms.
**Previous injury:** Has the athlete experienced a similar injury in the past? Tendinopathies can flare up or rupture or bone stress injury can lead to fracture.

## PHYSICAL EXAMINATION

### Observation and Measurement

#### Standing Examination

**Alignment of the lower extremities:** View the patient from the front and back. Check for limb length inequality, pelvic tilt, genu varum, genu valgum, flexion, extension, and rotational abnormalities of both lower extremities.
**Foot alignment and mechanics:** Evaluate the medial longitudinal arch for pes planus, pes planovalgus, pes cavus, forefoot abduction, and forefoot varus (Fig. 60.2A). Check single- and double-leg heel rise to confirm that the heels invert and arches increase. Evaluate the forefoot for hallux valgus (or varus) and pronation; toe cross-over, cock-up, hammering or clawing of lesser toes (Fig. 60.2B); and metatarsus adductus.

#### Gait Analysis

**Inspect ambulation:** Assess all phases of gait for asymmetric movement on either side of the body, degree of toeing in or out, inversion of the heel, and supination of the foot. Observe the medial longitudinal arch during the stance phase. Assess for antalgic, neuropathic (e.g., steppage), and myopathic (e.g., Trendelenburg) gait and symmetric strength during push-off.

#### Seated Examination

**Visible abnormalities:** Note varicosities, erythema, ecchymosis, edema, and muscle wasting.
**Vascular:** Palpate posterior tibial and dorsalis pedis pulses. Assess capillary refill time. Absence of hair distally may indicate peripheral vascular disease.
**Skin:** Note the location of callus formation, scars, wounds, blisters, ulcerations, and discoloration. Visualize toenails and nail beds.
**Range of motion:** Check active and passive ankle, subtalar, and transverse tarsal and first metatarsophalangeal joint (MTPJ) motion; compare them to the uninjured side. Notable crepitus during range-of-motion examination indicates degenerative changes, fracture, or tissue thickening.
**Strength testing:** Test muscle groups against resistance—dorsiflexion, plantarflexion, inversion, and eversion; compare with the contralateral side.

### Palpable Anatomic Structures

**Bones:** Fifth metatarsal base, peroneal tubercle, first tarsometatarsal joint, navicular tuberosity, head of talus, sustentaculum tali, lateral malleolus, medial malleolus, and metatarsal heads (see Fig. 60.1A)
**Ligament attachments:** Calcaneofibular ligament, anterior talofibular ligament, superficial deltoid ligament, spring ligament, and plantar fascia (see Fig. 60.1C)
**Tendons:** Posterior tibialis, flexor hallucis longus (FHL), anterior tibialis, peroneal brevis and longus, extensor hallucis longus, and Achilles (see Fig. 60.1C)
**Bursae:** Calcaneal, retrocalcaneal

### Special Tests/Signs

**Thompson test:** Patient prone with affected leg extended; squeeze calf muscles to indirectly plantar flex the foot; failure of the foot to plantarflex (a positive test)
**Abduction stress test:** Manual abduction of the forefoot while stabilizing the hindfoot; assess for pain clinically and widening of Lisfranc joint on radiographs
**Calcaneal compression text:** Squeeze the posterior heel simultaneously from medial and lateral sides. Pain is a positive test and suggests stress fracture.
**Too many toes sign:** View the patient from behind, three or more toes visible lateral to the heel suggests forefoot abduction, often

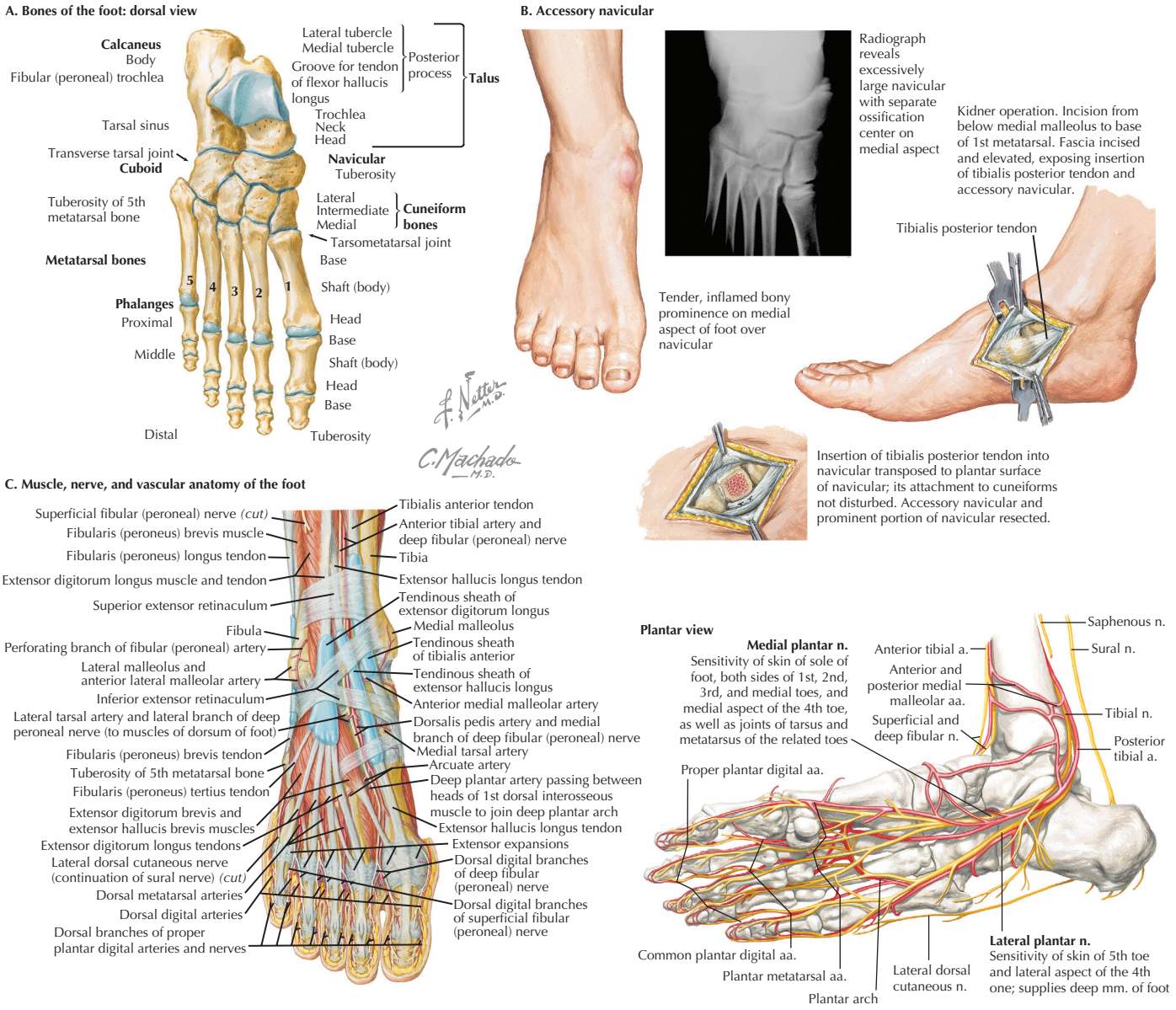



**Figure 60.1.** Bones, muscles, and vessels of the foot.

seen with posterior tibial tendon or spring ligament dysfunction (see Fig. 60.2A)

**Peek-a-boo heel sign:** Viewed from the front, indicates hindfoot varus alignment (Fig. 60.3)

## COMMON INJURIES AND DISORDERS OF THE FOOT

### Skin and Nail Problems

For details, see Table 60.1, Fig. 60.4, and Chapter 40, Skin Problems in the Athlete.

### Tendinopathies (Table 60.2)

**Etiology:** Repetitive or abnormal stress on a tendon causes macrotears (acute trauma) or microtears (chronic overuse) with resulting inflammation (tendinitis) and ultimately tendon degeneration (tendinosis).

**Common tendinopathies:** Posterior tibial, Achilles, peroneal, anterior tibial, and FHL tendinopathy

**Diagnostic considerations:** Plain radiographs show alignment, mechanical risk factors, bony avulsions, and bony contributions (e.g., Haglunds and/or calcification at Achilles tendon insertion). Magnetic resonance imaging (MRI) and ultrasound best to assess tendon status and confirm partial or complete ruptures

**Treatment:** Most tendinopathies will resolve with rest, ice, nonsteroidal anti-inflammatory drugs (NSAIDs), and addressing the risk factors. Acute tendon ruptures should typically be repaired in athletes (i.e., Achilles, posterior tibial, anterior tibial); peroneal tendon transfer can be considered in attritional peroneal ruptures; surgery for tendinopathy only if conservative treatments fail

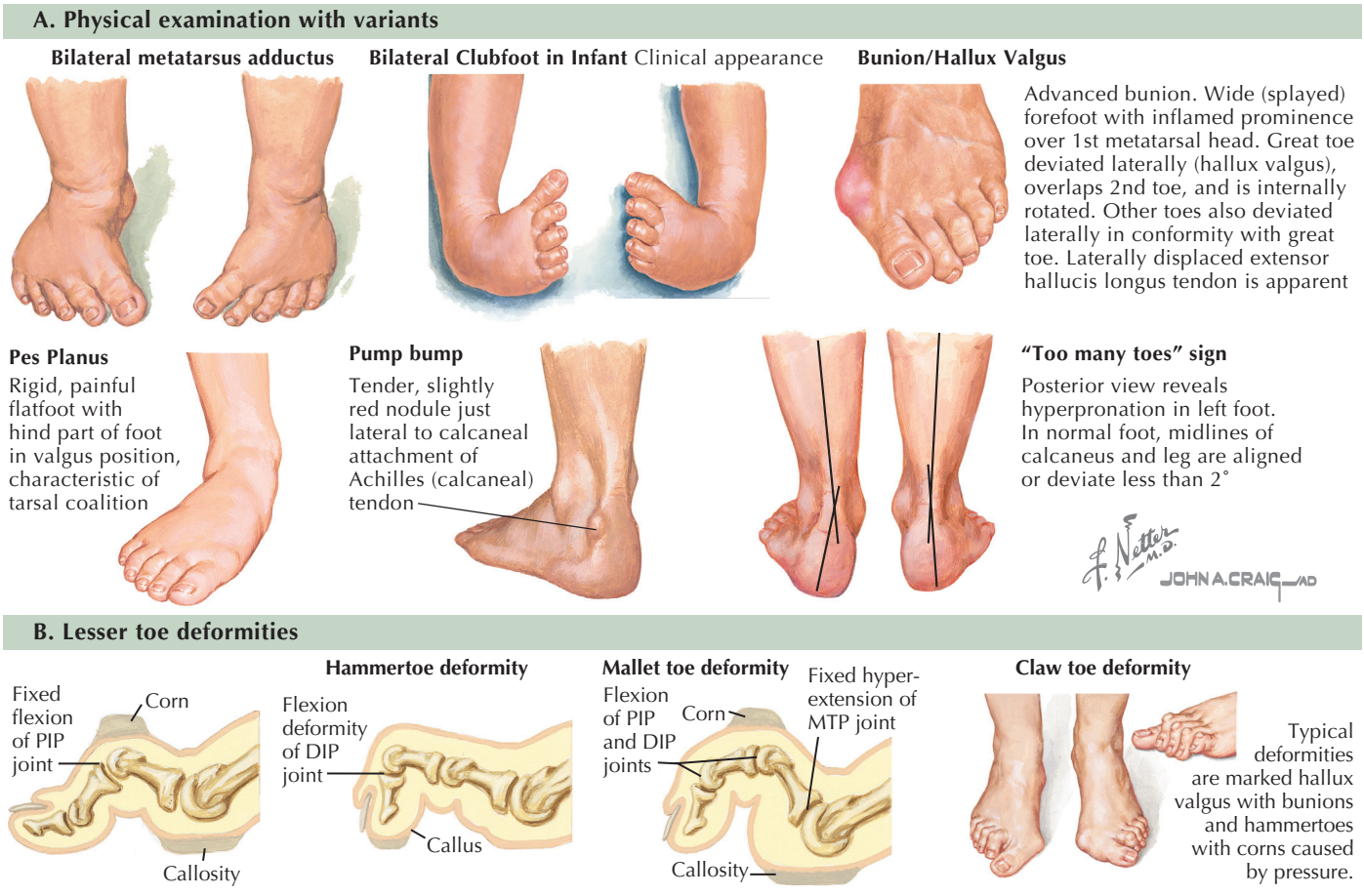



**Figure 60.2.** Physical examination of the toe and deformities.

## TABLE 60.1 SKIN AND NAIL PROBLEMS

| Type | Cause | Signs and Symptoms | Treatment |
|---|---|---|---|
| Calluses and corns | Excessive localized friction or pressure from tight shoes or structural abnormalities of feet, such as hammer toes | Pain; thickening and hardening of skin (soft corns between toes due to moisture) | Appropriate shoe fitting, padding (e.g., doughnut pad), pumice stone, resection of bony prominences, metatarsal pad, Budin splint |
| Blisters | Friction, pressure<br>Epidermal/dermal separation by serous fluid | Painful vesicles | Unruptured: sterile dressing, pressure relief<br>Ruptured: sterile cleansing, dressing (consider antibiotics for diabetic patients or signs of infection in any patient) |
| Warts | Virus (papilloma) | Pain at site; skin thickening with central core; flat or raised | Trichloracetic acid or salicylic acid, liquid nitrogen |
| Tinea pedis (athlete's foot) | Fungus | Dry or vesicular lesions; scaling, peeling, and cracking fissures in skin; deformed nails, hyphae, and buds on KOH wet mount | Dry: miconazole, clotrimazole, terbinafine, salicylic acid<br>Vesicular: wet dressings with Burrow's solution<br>Erythema or other signs of infection: consider antibiotics |
| Paronychia | Soft tissue infection around the nail | Inflamed nail margin with or without drainage | Warm water soaks, antibiotics; partial nail resection; appropriate nail-cutting techniques |
| Subungual hematoma | Trauma | Dark blood under nail; pain/pressure at site | Drainage (insert no. 18 needle or drill sterilely through nail)<br>Note: ensure traumatic history to distinguish from subungual melanoma |

**Figure 60.3.** Peekaboo heel sign in hindfoot varus.

## Heel Pain in the Athlete

### Plantar Fasciopathy

**Etiology:** Excessive tightness of gastrocsoleus complex pulling into Achilles tendon causes overload at plantar fascia origin on calcaneus during weight-bearing activities; result in microtears and inflammation (Fig. 60.5)

**Symptoms/signs:** Point tenderness/start-up pain, usually along medial tubercle of calcaneus; sometimes relieved with movement

**Imaging:** Radiographs may show plantar calcaneal enthesophyte (usually not the cause of pain). MRI (see Fig. 60.5) or ultrasound can show thickening of plantar fascia/partial tear.

**Differential diagnosis:** Plantar fascial rupture; tarsal tunnel syndrome; calcaneal stress fracture; heel pad atrophy; and Baxter's nerve entrapment

**Treatment:**

- Stretching, cushioned heel pad or orthoses, and supportive shoes (see Fig. 60.5)
- Decrease impact activities, ice/massage, and dorsiflexion night splint
- Refractory cases:
  - Corticosteroid injection: use with caution, risk of fat pad atrophy or plantar fascia rupture
  - Walking cast
  - Extracorporeal shock wave treatment
  - Percutaneous ultrasonic probe
  - Partial fasciectomy

### Sever's Disease (Calcaneal Apophysitis)

**Etiology:** Heel pain in children between 9 and 14 years of age participating in running sports; often biomechanical abnormality contributing to poor shock absorption, such as equinus, hallux valgus, pes cavus, and pes planus; occasionally, acute trauma (e.g., violent heel strike)

**Symptoms/signs:** Posterior heel pain, worse during and after activity and improves with rest, tender to palpation at or just inferior to Achilles insertion

Positive "squeeze" test: compression of medial and lateral aspects of calcaneal apophysis produces pain

Positive "Sever's test": heel pain aggravated by heel rise

**Imaging:** Radiographs characterized by fragmentation, sclerosis, and increased density of apophysis that mimic the appearance of osteonecrosis; changes are best viewed on lateral radiographs.

**Differential diagnosis:** Achilles tendinitis/strain, heel pad pain, retrocalcaneal bursitis, calcaneal stress fracture, and plantar fasciitis

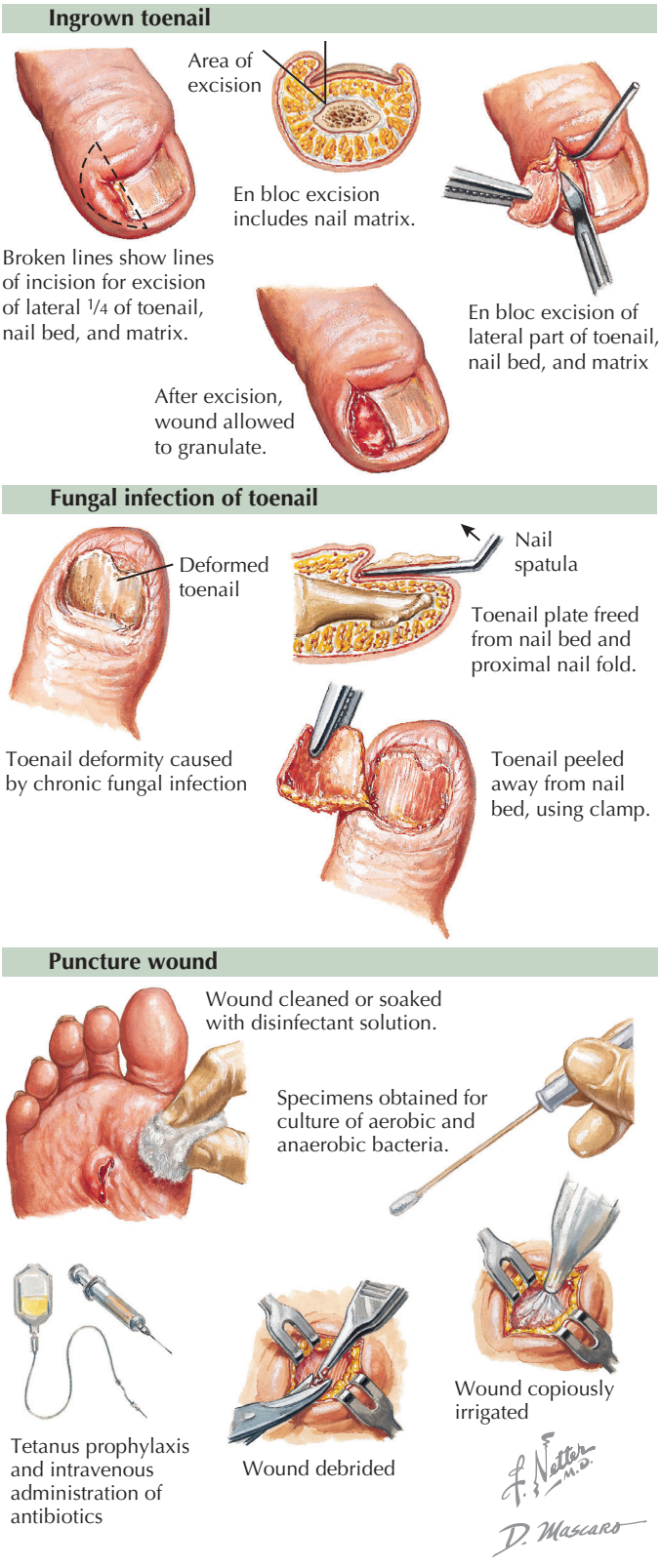
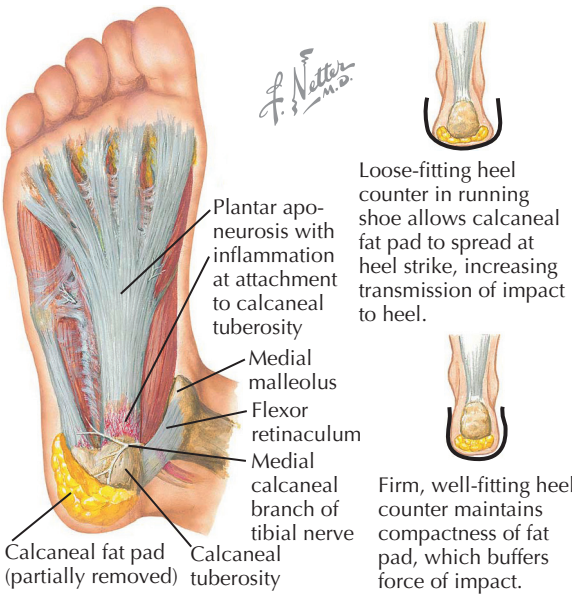



**Figure 60.4.** Nail problems in the foot.

## TABLE 60.2 INFLAMMATORY CONDITIONS

| Type | Signs and Symptoms | Treatment |
|---|---|---|
| Posterior tibial tendinitis | Medial arch pain, swelling, pain with resisted inversion, painful or inability to perform single heel rise, medial arch collapse, "too many toes sign": when viewed from behind, abducted forefoot allows more toes to be seen on the affected side | Initial: rest, ice, NSAIDs, orthoses with arch support, and medial heel wedge<br>Rehabilitation:* plantarflexion and inversion strengthening exercises, heel cord flexibility<br>Surgical: treatment ranges from osteotomy with tendon transfer to arthrodesis |
| Peroneal tendinitis | Lateral pain, particularly with active and resisted eversion; swelling; may complain of snapping sensation in cases of peroneal tendon subluxation; may be due to underlying tendon tear | Initial: rest, ice, NSAIDs, orthoses, cast, or boot<br>Rehabilitation:* eversion strengthening exercises; surgery may be required for recalcitrant cases, tears, or subluxation |
| Anterior tibial tendinitis | Pain in anterior medial foot, worse with active dorsiflexion; swelling and crepitation; seen in runners, hikers, and racquet sports | Initial: activity modification, ice, NSAIDs, and walking boot<br>Rehabilitation:* dorsiflexion strengthening exercises |
| Achilles tendinitis | Decreased gastrocnemius flexibility; pain, tenderness, swelling, and crepitation; pain with active plantarflexion; radiographs to look for Haglund's deformity or insertional calcifications | Rule out systemic conditions such as gout or spondyloarthropathy.<br>Initial: rest, ice, NSAIDs, and heel pads<br>Rehabilitation:* heel cord stretching and strengthening exercises; possible surgical debridement in chronic cases |
| Retrocalcaneal bursitis | Posterior heel/ankle pain; tenderness and swelling in bursa located between the Achilles tendon and the calcaneus | Initial: rest, ice, NSAIDs, heel cup, and shoe modification<br>Rehabilitation:* heel cord stretching, modalities, strengthening exercises |
| Sesamoiditis | Generic term; local tenderness (tibial side most common); pain worse with weight bearing; rule out avascular necrosis, sesamoid fracture. Bipartite sesamoid reported 10% tibial, rare fibular | Reduce weight-bearing stress at site; orthoses with sesamoid relief or Morton's extension; ice, NSAIDs, use of stiff-soled shoe or walking boot;<br>if conservative treatment fails: bone scan, computed tomography, or MRI to rule out stress fracture |
| Metatarsophalangeal joint synovitis | Pain at metatarsophalangeal joint (second most common), positive Lachman test (increased anterior-posterior translation), and joint crepitus; distinguish from interdigital neuroma | Metatarsal pad, figure-of-eight taping, Budin splint, NSAIDs, steroid injection (with caution), and surgery for failed nonsurgical treatment |

*Rehabilitation includes maintenance of aerobic, well-leg, and upper-body fitness; physical therapy modalities, such as phonophoresis and iontophoresis, may also be useful.
MRI, Magnetic resonance imaging; NSAIDs, nonsteroidal anti-inflammatory drugs.

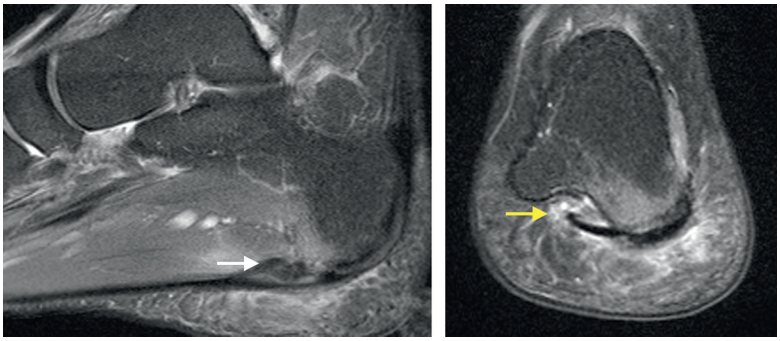





Loose-fitting heel counter in running shoe allows calcaneal fat pad to spread at heel strike, increasing transmission of impact to heel.



Firm, well-fitting heel counter maintains compactness of fat pad, which buffers force of impact.



T2-weighted sagittal *(left)* and coronal MRI *(right)* showing small focal tear of plantar fascia *(yellow arrow)* with diffuse thickening *(white arrow)* and surrounding soft tissue swelling

**Figure 60.5.** Plantar fasciitis.

**Treatment:** Rest (crutches in severe cases), activity modification, ice, heel lifts or cups, stretching and strengthening, NSAIDs; if still resistant—dorsiflexion night splint and rarely, short leg cast for 2 weeks, orthoses

## Forefoot Disorders

### Turf Toe

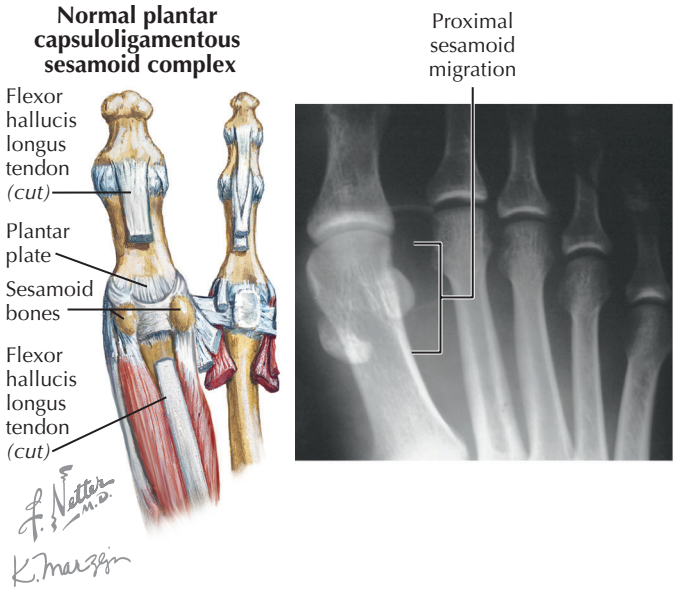
**Etiology:** Hyperextension of MTPJ of great toe leads to a spectrum of injuries from plantar plate tear to dislocation (Fig. 60.6)

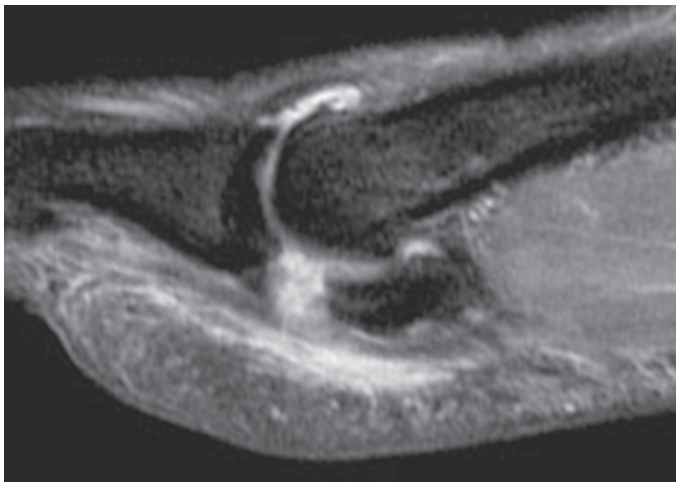
**Mechanism of injury:** Typically, an axial load on a foot fixed in equinus with MTPJ in hyperextension (e.g., football linemen during push-off)

**Symptoms/signs:** Pain, tenderness, and swelling at great toe MTPJ, and plantarflexion weakness; positive great toe Lachman test; compare with the contralateral side

**Imaging:** Radiographs may show proximal migration of sesamoids with plantar plate rupture (see Fig. 60.6). Stress fluoroscopy to evaluate sesamoid tracking of the sesamoids; MRI (see Fig. 60.6) to determine injury severity and surgical planning

**Differential diagnosis:** Phalangeal or metatarsal fracture, osteochondral injury, hallux rigidus, osteoarthritis, and gout



Plantar plate injury on MRI scan

**Figure 60.6.** Turf toe.

**Treatment:** Rest, ice/NSAIDs acutely, taping to limit MTPJ motion, walking boot until pain free, metatarsal pad to unload first metatarsal may be helpful; rigid shoe or insert to limit MTPJ hyperextension; surgery considered in complete rupture, unstable joint, sesamoid fracture, traumatic hallux valgus, or chondral injury with loose body

### Hallux Valgus (Bunion)

**Etiology:** Associated with increased ligamentous laxity, inappropriate shoe fit, pes planus, heredity, posttraumatic, and/or inflammatory conditions; more common in females

**Symptoms/signs:** Pain usually secondary to pressure and friction on prominent medial eminence (i.e., bunion); soft tissues may become inflamed, thickened, and painful (Fig. 60.7) causing nerve irritation; secondary deformities (e.g., hammertoes, callosities, and metatarsalgia) may contribute to pain and discomfort.

**Imaging:** Standing radiographs to assess hallux valgus and intermetatarsal angles, MTPJ congruity and arthritis, distal metatarsal articular angle (DMAA), and sesamoid position (see Fig. 60.7)

**Treatment:** Conservative measures to provide pain relief: shoes with adequate forefoot width and arch support; pads to cushion bunion, toe separators, and orthoses to help redistribute weight; surgery for cases that fail conservative management and limit sports participation and should be avoided in sprinters, high jumpers, pole vaulters, and ballet dancers if possible owing to the risk of decreased MTPJ range of motion; wait until end of athlete's career or until whenever possible

### Hallux Rigidus

**Etiology:** Limitation of MTPJ motion, particularly dorsiflexion; mechanical block due to periarticular osteophytes; usually posttraumatic, but can result from chronic turf toe, repetitive microtrauma (runners), primary arthrosis

**Symptoms/signs:** Tender palpable MTPJ osteophyte(s); decreased and painful first MTPJ dorsiflexion; pain increased with running, incline training, and wearing shoes with elevated heels (Fig. 60.8)

**Imaging:** Weight-bearing anteroposterior (AP), lateral, and oblique views; may be normal initially; nonuniform narrowing of first MTPJ with widening and flattening of the metatarsal head, loose body, marginal osteophytes; dorsal exostosis of first metatarsal is the hallmark.

**Treatment:** Shoe modifications (stiff sole, rocker bottom, extra-depth toe box), metatarsal bar, NSAIDs, orthoses with Morton's extension, and intra-articular steroid injections; surgical options include cheilectomy or fusion to relieve impingement and decrease pain; proximal phalanx osteotomy may benefit a running athlete by improving dorsiflexion movement.

### Lesser MTPJ Instability

**Etiology:** Overload of metatarsal head leading to repetitive stress and attritional tear of MTPJ plantar pate

**Symptoms/signs:** Pain under metatarsal head (2nd most common), hammertoe deformity, swelling, and toe subluxation or dislocation

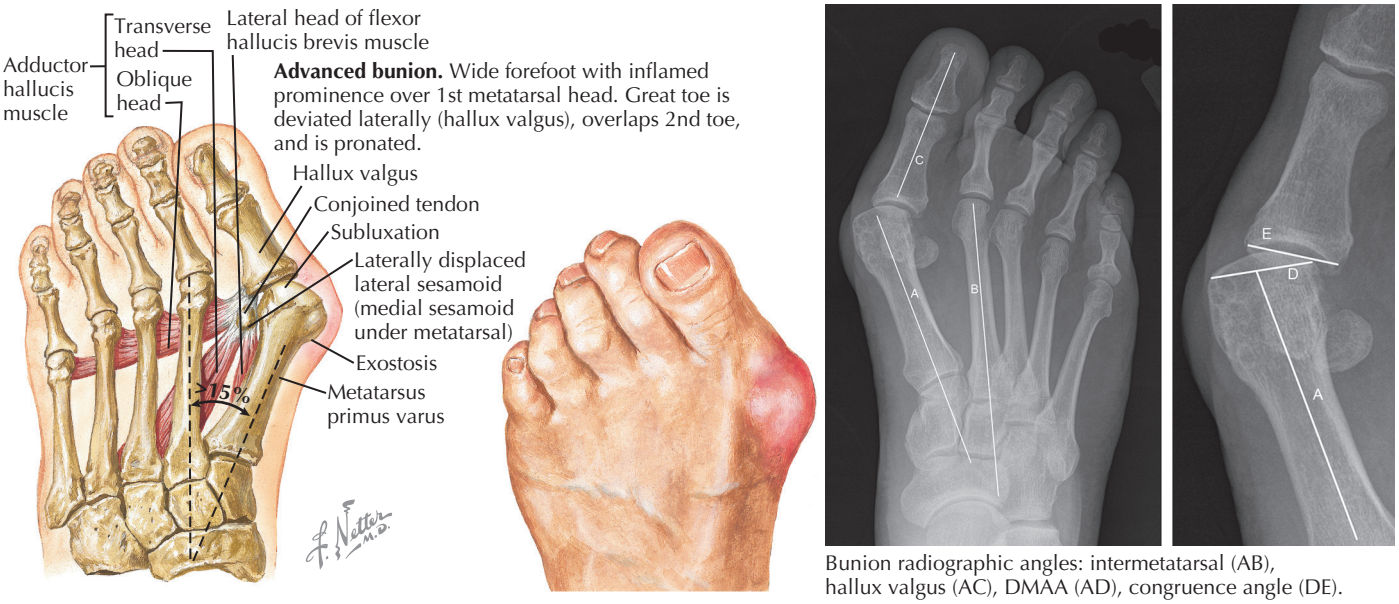
**Imaging:** Radiographs often show long second metatarsal and MTPJ subluxation or dislocation. MRI shows the grade of plantar plate injury.

**Treatment:** Rest, ice, splinting of toe, and stiff-soled shoes; surgery to repair plantar plate in recalcitrant cases

## Bone Stress Injuries

### Navicular Stress Fracture

**Etiology:** Common in running and jumping athletes; repetitive cyclic loading, explosive push-off; unique vascular anatomy




Bunion radiographic angles: intermetatarsal (AB), hallux valgus (AC), DMAA (AD), congruence angle (DE).

**Figure 60.7.** Bunions and hallux valgus.

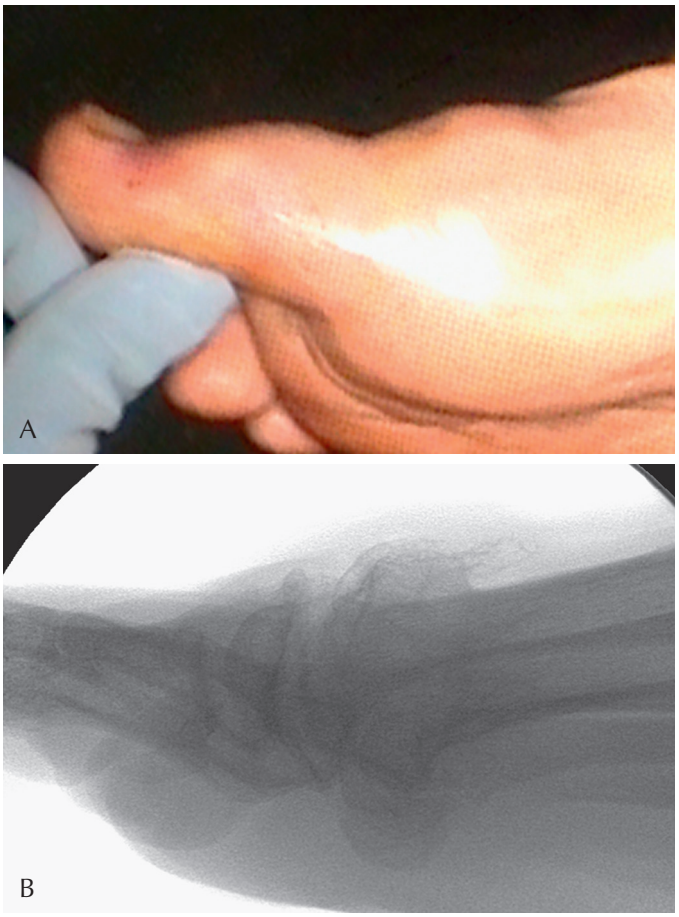



**Figure 60.8.** Hallux rigidus.

results in relatively avascular central one-third; frequently missed, delay in diagnosis by up to 4–7 months reported

**Symptoms/signs:** Ill-defined midfoot to anterior ankle soreness and cramping; insidious onset, progresses to frequent pain with activity; pain with percussion over navicular, N-spot tenderness to palpation, and symptom exacerbation with single leg hop

**Imaging:** Foot radiographs often negative; bone scan highly sensitive, uptake seen in all three phases; computed tomography (CT): gold standard, defines location, completeness, direction and displacement; fracture line usually proximal–dorsal to distal–plantar

**Treatment:** Nonweight-bearing short leg cast for 6–8 weeks; if remains tender to palpation, replace in cast, otherwise progressive return to activity; persistent symptoms, delayed union, or displaced fractures require surgery (internal fixation with or without bone grafting); recovery can take up to 1 year.

## Calcaneal Fractures

**Etiology:** Acute fractures are rare. Stress fractures more common, usually in endurance sports.

**Symptoms/signs:** Localized pain in heel, accentuated by weight bearing; mild swelling; pain with medial–lateral compression of calcaneus

**Imaging: Stress fracture:** May see line at posterior–superior aspect of the calcaneus perpendicular to the trabeculae; if negative, bone scan or MRI

**Treatment:** Modified rest with cessation of impact activities; cast or walking boot with crutches may be appropriate; gradual return to activity when clinical symptoms abate and serial radiographic assessment documents healing. Full healing can take 2–3 months. Consider shock-absorbing orthotic devices on returning to activity

**Traumatic:** Closed or open treatment depending on severity of fracture

## Jones Fracture

**Etiology:** Stress fracture of the fifth metatarsal at metaphyseal–diaphyseal junction in the region of or just distal to the fourth-to-fifth metatarsal articulation (Fig. 60.9); frequently seen in sprinters and jumpers; often have difficulty healing because of poor blood flow

**Treatment:** Cast for 4–6 weeks, followed by 4–6 weeks in walking boot; approximately 75% will heal with nonsurgical treatment, but 30%–50% will re-fracture. Surgical stabilization with intramedullary screw for acute fracture in athletes, nonunion, re-fracture, and cavovarus foot with lateral overload; bone grafting considered when treating delayed union or nonunion

Figure 60.9. Jones fracture.

### Sesamoiditis

**Etiology:** Usually repetitive trauma in running/jumping sports; contributing factors include mechanical overload caused by pes cavus, ankle equinus, and poor footwear. Anatomic variations that can contribute to mechanical overload include absent crista, variations in sesamoid size, and significant metatarsal rotation.

**Symptoms/signs:** Insidious, unilateral plantar forefoot pain with weight bearing, exacerbated by certain shoes or activities, focal to one or both sesamoids (tibial sesamoid most common); range of motion of the hallux MTPJ usually painless; direct tenderness and tenderness on resisted plantar flexion of the hallux indicate sesamoid pathology; attention to mechanical alignment of the foot is important.

**Imaging:** Plain radiographs (AP, lateral, and sesamoid views) show bony contour, bipartite sesamoid, fracture, and advanced avascular necrosis (AVN); MRI more sensitive for AVN, stress fracture, and injury acuity; bone scan and CT scan can also identify stress fracture and differentiate between bipartite and fracture.

**Treatment:** ***Sesamoiditis***: Reduce weight-bearing stress at site; orthoses with sesamoid relief or Morton's extension; ice, NSAIDs, use of stiff-soled shoe, carbon fiber insole, or walking boot; if conservative treatment fails, bone scan, CT, or MRI to rule out stress fracture

**Fracture**: Immobilization, protected weight-bearing (cast or boot), custom-molded orthosis to address mechanical contributors; if conservative treatment fails surgical options include partial or complete sesamoidectomy. Beware of transfer sesamoiditis and alignment changes.

## Acute Fractures

### Talus Fractures

**Etiology:**

- **Acute trauma:** Rare in sports, often a surgical emergency; talar neck fractures are associated with severe foot dislocation
- **Stress fractures:** Also rare; symptoms/signs include diffuse midfoot pain, anterior ankle tenderness on palpation, and painful active and passive motion of subtalar joint
  *Note:* Subtalar joint allows eversion/inversion.
- **Lateral process fractures:** "Snowboarder's fracture"; frequently misdiagnosed as a lateral ankle sprain; mechanism is acute dorsiflexion with inversion of foot and axial load.

**Imaging:** Radiographs usually provide definitive diagnosis. Subtle or stress fractures usually require bone scan or CT. Lateral process fractures are seen best on coronal CT images (Fig. 60.10).

**Treatment:**

- **Talus fractures:** Nondisplaced or stress fractures require rest, cessation of weight-bearing activity for 6 weeks followed by walking boot; displaced fractures usually require surgical open reduction and internal fixation (ORIF).
- **Lateral process fractures:** Treatment determined by fragment size, comminution, and displacement; nondisplaced—nonweight-bearing boot or cast for 6 weeks; displaced—ORIF or excision of fragment(s)

Figure 60.10. Coronal CT lateral process fracture.

## Midfoot/Forefoot Fractures

### Lisfranc Injury

**Etiology:** Traumatic injury: (i) twisting of forefoot (e.g., equestrian foot caught in stirrup during fall); (ii) axial load with foot in equinus (e.g., football and soccer); and (iii) crush injury (Fig. 60.11). Lisfranc ligament runs from plantar–medial cuneiform to the base of the second metatarsal; no intermetatarsal ligament between the first and second metatarsals; important to identify the injury, particularly in subtle cases

**Symptoms/signs:** Pain and inability to bear weight or push off, swelling and gross deformity if severe, may have spontaneous reduction after injury

**Imaging:** Weight-bearing AP, lateral, and oblique radiographs; compare with contralateral side; medial border of second metatarsal should parallel medial border of the middle cuneiform on AP; medial border of fourth metatarsal should parallel medial border of cuboid on oblique. Look for widening between first and second metatarsals and medial and middle cuneiform; MRI if suspicion is high and radiographs are negative

**Treatment:** Sprains can be placed in a CAM boot. Nondisplaced with ligament disruption in nonweight-bearing cast; displaced injuries require ORIF using screws, plates, or flexible devices.

### Metatarsal Fractures

**Etiology: Traumatic:** Direct blow to the foot or hard landing

**Stress:** More common and due to repetitive loading, associated with increased mechanical loading; seen in endurance running and jumping sports. Be aware of female triad (anorexia, amenorrhea, and osteoporosis).

**Symptoms/signs:** Localized pain with weight-bearing, swelling, and tenderness

**Imaging:** Plain radiographs confirm most acute fractures (Fig. 60.12); may be negative for stress fractures; bone scan or MRI may be necessary to detect early stress lesions; CT more specific for stress fractures

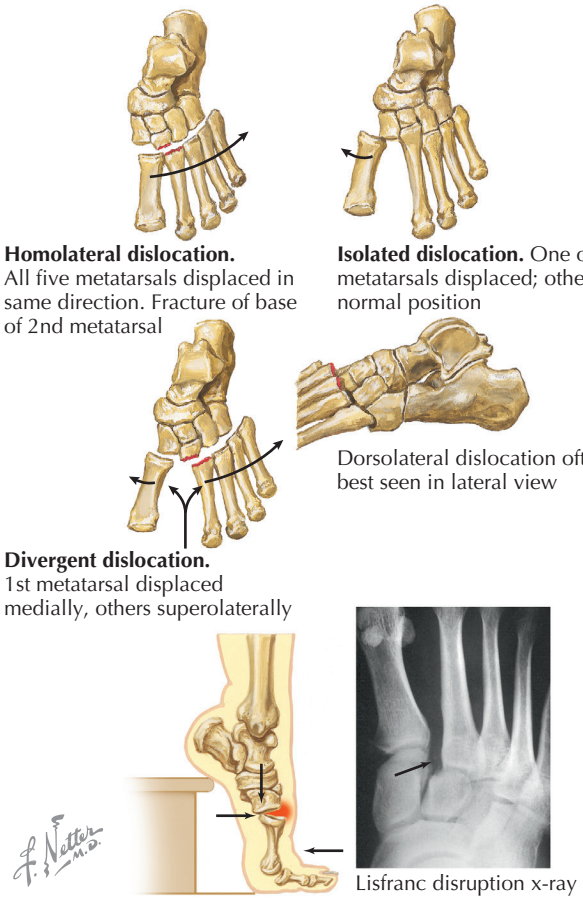



**Figure 60.11.** Lisfranc injuries.

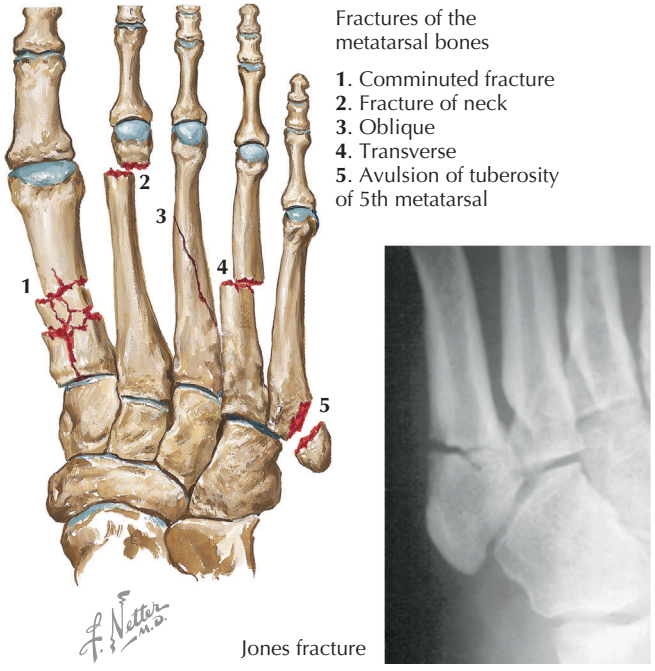



**Figure 60.12.** Metatarsal fractures.

**Treatment:**

- Acute fractures: ORIF if displaced or intra-articular
- **Second through fourth metatarsal stress fractures:** Nonsurgical treatment (first metatarsal stress fracture rarely seen); modified rest with cessation of weight-bearing activities and immobilization in cast or boot for 3 weeks may be used depending on the pain control needs; gradual return to activity when symptoms subside; base of the second metatarsal may show delayed or nonunion, thus requiring bone grafting and/or ORIF
- **Avulsion fracture of the base of the fifth metatarsal:** Common with ankle sprains; nonsurgical treatment successful in most cases; consider ORIF if displaced or nonunion

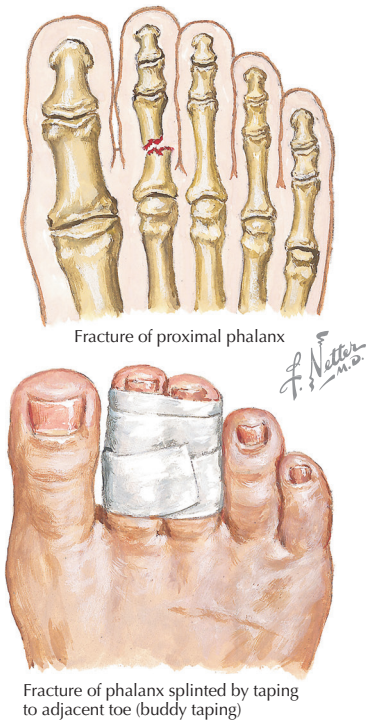



**Figure 60.13.** Phalangeal fractures.

### Phalangeal Fractures

**Etiology:** Trauma such as crush, direct blow, or jamming
**Symptoms/signs:** Pain, deformity, swelling, and ecchymosis
**Imaging:** Plain radiographs confirmatory
**Treatment:** Conservative unless open fracture or intra-articular fracture of great toe with displacement

- **Nondisplaced:** Buddy taping to adjacent toe and protection in stiff-soled shoe; symptoms dictate activity restrictions (Fig. 60.13)
- **Displaced:** Reduced by manipulation; subsequent buddy-taping/splinting
- **Complications:** Intra-articular fractures can result in joint stiffness and arthritis: consider ORIF.

## Neurologic Injuries

### Tarsal Tunnel Syndrome

**Description:** Entrapment of posterior tibial nerve at flexor retinaculum behind medial malleolus
**Etiology:** Trauma (subtalar dislocation or fractures); compression from space-occupying lesion (e.g., ganglion); systemic disorders (e.g., diabetes mellitus); biomechanical dysfunction (e.g., hindfoot valgus or tarsal coalition); idiopathic
**Symptoms/signs:** Aching pain at the medial foot, aggravated by prolonged weight bearing, radiating to plantar foot, plantar foot numbness/tingling; positive Tinel's sign (tapping over tibial nerve causes radiating pain along nerve distribution); Valleix phenomenon (neural percussion causes radiation of pain proximally along course of nerve)

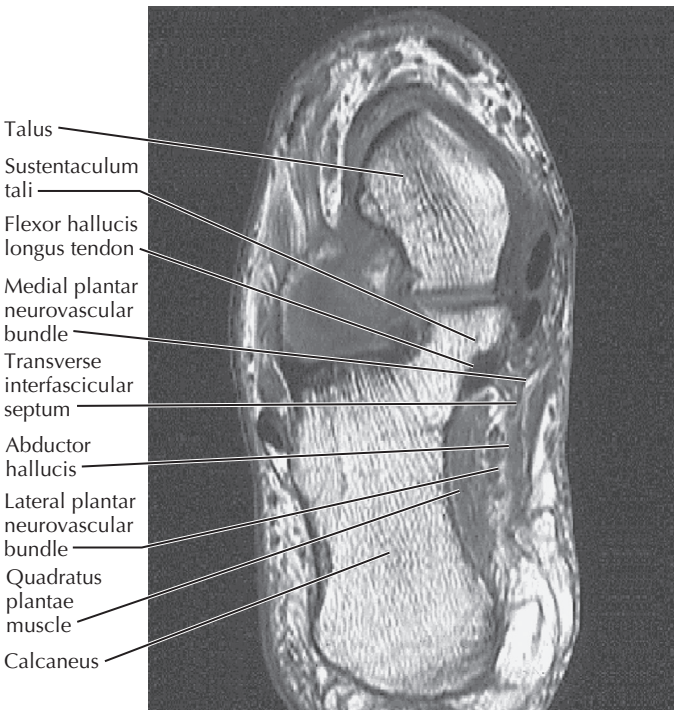



**Figure 60.14.** Tarsal tunnel anatomy.

**Diagnostics:** Radiographs obtained to assess foot alignment, malunion, and exostosis; MRI visualizes soft tissue space-occupying lesions (Fig. 60.14); electromyography (EMG) and nerve conduction studies are abnormal in 80% cases.
**Differential diagnosis:** Posterior tibial tendinitis, calcaneal stress fracture, gout, plantar fasciitis, and herniated lumbar disc
**Treatment:** Rest, NSAIDs, graduated return to activity; orthoses; local steroid injection; surgical decompression for intractable cases; best results with surgical decompression in cases caused by space-occupying lesions without nerve damage

### Interdigital (Morton's) Neuroma

**Description:** Impingement of interdigital nerves as they bifurcate at metatarsal heads
**Etiology:** Trauma and/or repetitive stress leads to chronic irritation of nerves as they cross under transverse metatarsal ligament to toes; fusiform swelling and pathologic changes occur in nerves.
**Symptoms/signs:** Pain, burning neuralgia with radiation into involved toes; symptoms worse with activity and narrow shoes; sensation of "walking on marbles"; third interspace most common (Morton's); tender to palpation between metatarsal heads, pain with squeeze of metatarsals; Mulder's click: squeeze the forefoot from medial to lateral while palpating the web space, positive test is a click or gritty sensation
**Differential diagnosis:** Metatarsalgia, metatarsal stress fractures, MTPJ synovitis, and neuropathy
**Radiographic assessment:** Usually negative; evaluate for stress fracture, MTPJ synovitis, and compression from exostosis
**Treatment: Initial:** NSAIDs, metatarsal pads, wide toe-box shoes, lower heel, corticosteroid injection
**Chronic:** Surgical excision of nerve; patient may lose sensation between involved toes but no functional deficits; recurrence from stump neuroma may occur

### Chronic Regional Pain Syndrome (CRPS)

**Description:** Pain syndrome accompanied by evidence of autonomic dysfunction
**Etiology: Type I:** unknown etiology, insidious onset; **Type II** can occur after injury or trauma; likely results from dysfunction of autonomic nervous system
**Symptoms/signs:** Onset is heralded by severe, diffuse, unrelenting pain with exquisite tenderness to even light touch, out of proportion to original injury; decreased range of motion and autonomic vasomotor signs, including warm or cool skin temperatures and decreased peripheral pulses. Skin may be moist/sweaty or dry/scaly with discoloration and swelling.
**Imaging:** Radiographs may reveal diffuse osteopenia of the involved foot. Bone scan may show delayed pattern of diffuse increased tracer throughout the foot, with juxta-articular accentuation of tracer uptake.
**Differential diagnosis:** Peripheral nerve injuries, polymyositis, lupus, Raynaud's disease, gout, and thrombophlebitis
**Treatment:** Prompt diagnosis and therapy improve the chances of permanent relief. Refer to a pain management specialist. Sympathetic blockade relieves pain. Physical therapy includes vigorously active exercises, weight-bearing activities, and direct stimulation of skin. Adjuvant pharmacologic agents and psychological evaluation required in more difficult cases. Occasionally, chemical or surgical sympathectomy is required.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 61 TAPING AND BRACING

Rebecca Ann Myers • Thomas A. Frette

## GENERAL PRINCIPLES

- Taping and bracing are used as **adjuncts** to sports protective equipment, treatment, and rehabilitation of an injury.
- Should not take the place of appropriate diagnostic, treatment, and rehabilitation of an injury
- Role in prevention, treatment, rehabilitation, and return-to-play decisions

### Functions of Taping and Bracing

**Prevention:** Aid in stabilization, support, and protection of uninjured or fully rehabilitated joints and soft tissues

**Treatment:** Minimize pain and swelling in the acute phase, aid in unloading painful structures, and provide support to unstable joints

**Rehabilitation:** Aid in early mobilization, muscle imbalances, neural control, proprioception, and protect healing injuries

### Physician's Role in Taping and Bracing

- **Determine the appropriateness** of taping/bracing.
- **Facilitate the selection process.**
  - **Identify** available options.
  - **Communicate** with the treatment team: athlete, parent, certified athletic trainer, physical therapist, and coach
- **Evaluate effectiveness** of the selected support.

### Implementation of Taping and Bracing

- **Athlete's acceptance:** Involve the athlete in the decision-making process; must be comfortable and functional; realize positive psychological effects of taping and bracing, which may improve athlete's confidence on returning to competition
- **Sporting equipment regulations:** Know the sport's regulations. Equipment modifications may be needed. Use materials that do not endanger other athletes. Exposed metal must be covered during contact sports. The environment will affect options.

### Practical Considerations

**Prescription:** Braces may require physician prescription, which needs to include diagnosis code, type of brace, length of need, and, occasionally, statement of medical necessity.

**Cost:** Taping materials and braces can be costly. Athletic departments may or may not cover full expense. Insurance coverage varies, and durable medical equipment may not contribute toward the patient's deductible.

**Marketing:** Types of tape, taping techniques, and braces are full of unsubstantiated claims and disclaimers of liability. New products should be viewed with an open, but critical, mind.

## PRINCIPLES OF TAPING

### Preparation

Decide on an appropriate technique. Gather required tape supplies. Place the athlete's body part in position of function and/or protection. Use an appropriate table height to optimize the taper's body mechanics.

### Types of Tape

- Nonstretch tape: good tensile strength with cloth backing that gives mechanical support to ligaments and joints; can also be used to reinforce stretch tape (e.g., standard trainer's athletic tape)
- Stretch tape: may be a one-way stretch (in length or width) or two-way stretch (in length and width); conforms to contours of the body and allows normal tissue expansion; typically requires scissors to tear and are more expensive (e.g., Kinesiotape, Elastikon, Lightplast, or Cover-Roll)
- Cohesive bandages: sticks to itself, waterproof, and reusable; may be used in place of stretch tape (e.g., Coban or Co-Flex)
- Hypoallergenic tape: alternative to standard zinc oxide non-stretch tape
- Size of body part determines the appropriate width.

### Skin Care

**Preventive measures:**

- **Shave hair:** Increases adhesion of the tape and reduces irritation and build-up of residue
- **Apply taping base** (e.g., tincture of benzoin): Increases adhesion of the tape and provides a protective layer between the tape and skin
- **Apply tape underwrap** (e.g., thin polyester urethane foam): Decreases skin problems and increases athlete's comfort; may not be appropriate for all uses
- **Apply lubricant to possible areas of irritation** (e.g., lace and heel areas of the ankle)

**Appropriate tape removal:** Use scissors or cutters with a blunt tip. Teach the athlete the appropriate removal technique. Cleanse the skin to remove tape residue. Treat skin irritations and wounds promptly; these problems can prevent further taping.

**Allergic reactions to tape materials:** Recognize and treat problems. Consider alternative tape supplies. Investigate other forms of support and protection.

### Application

- **Requires skill; proficiency results from practice**
- **Elements of appropriate taping technique:**
  - Tearing tape is a basic skill; tape must be torn often.
  - Every piece of tape should have a distinct purpose.
  - Place anchor strips proximal and distal to the injured area directly on the skin.
  - Bridge across injury; duplicate anatomy needing support
  - Weave strips to add strength, overlapping by at least one-half the width of the tape
  - Adapt two-dimensional tape to three-dimensional body part
  - Limit pressure around body prominences, particularly when vascular and neural structures are superficial.
  - Use stretch tape over muscle bellies to allow normal muscle expansion.
  - Inspect for and tape over any gaps in taping to prevent blisters and tape cuts.
- Avoid common problems that restrict circulation: applying too much tape; applying repeated circumferential strips without

tearing between turns; forcing tape to go in desired direction; be careful if using tape with acute injuries because swelling causes tightness

## PRINCIPLES OF BRACING

- Bracing material should be functional during sport and daily activities; comfortable, lightweight, and moldable braces enhance their functionality.
- Braces should fit appropriately and be adjusted as swelling improves and activity progresses.
- Braces may migrate during activity. Tape, straps, and undergarments may be used to decrease unwanted migration.
- Brace evaluation over time is necessary to ensure appropriate function and to assess need for part replacement.
- Over-the-counter braces are instantly available, less expensive, efficient to use, but may not offer as much support as more specialized braces.
- Custom applications constructed from thermoplastic and other materials available for specific athlete needs (e.g., clamshell brace or ankle foot orthosis). Consultation with experienced orthotist, prosthetist, or occupational therapist recommended.

**A.** Ankle taping

**B.** Lace-up ankle brace

**C.** Hinged stirrup ankle brace

**Figure 61.1.** Ankle taping and bracing.

## PROPHYLACTIC TAPING/BRACING

### Knee

- Controversial whether knee braces prevent knee injuries
- Hinged knee braces are commonly used by football linemen with the rationale that they will reduce the valgus force on the joint, preventing medial collateral ligament (MCL) sprains.
- Biomechanical studies show that braces can provide greater resistance to a valgus force, protecting the MCL and in certain cases, even the anterior cruciate ligament (ACL), but whether this translates to injury prevention is unclear.
- Braces have also been shown to impair performance and increase the incidence of injury in certain player groups. There are several variables in such studies (position, player size, and type of brace) and even when they are accounted for statistically, it is difficult to make clear conclusions.

### Ankle

- Prophylactic ankle taping and ankle braces are used in several sports. Certain studies show a lower incidence of ankle injury with taping and bracing. Whether taping and bracing decrease the incidence in primary and/or secondary prevention and whether one method is superior to the other remains unclear.
- Certified athletic trainers frequently utilize prophylactic taping (Fig. 61.1A, Video 61-1).
- Lace-up ankle braces and hinged stirrup braces are often used (Fig. 61.1, B and C).

### Wrist

- Wrist taping, wraps, or braces are commonly used in various sports (gymnastics, weightlifting, football lineman, and snowboarding).
- Wrist guards in snowboarding have a role in preventing serious injury in children and novice riders.

## SELECTED TAPING/BRACING OF UPPER EXTREMITY INJURIES

### Glenohumeral Instability

- Shoulder stabilization brace (Fig. 61.2) often used in sports
- Harness attaches upper arm to chest, preventing forceful abduction and external rotation of shoulder.

**Figure 61.2.** Shoulder stabilization brace.

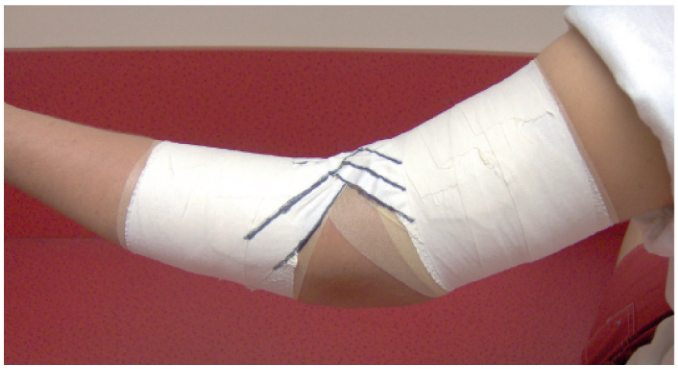
- Since shoulder joint is multidirectional, taping typically does not give enough support.
- Time and effort are probably better spent on rehabilitation.

### Elbow Hyperextension

- Taping technique (Fig. 61.3A): Use circumferential anchor strips around mid-upper arm and mid-forearm; criss-cross strips in "X" pattern over ventral elbow to limit elbow extension.
- Hinged elbow brace (Fig. 61.3B): Brace with medial and lateral supports; ventral straps in "X" pattern limit elbow extension. Hinges may also be locked to restrict flexion and extension. Medial and lateral supports prevent varus and valgus motion, which can protect radial or ulnar collateral ligament sprains, respectively.

### Lateral/Medial Epicondylosis

- Counter-force strap (Fig. 61.4) frequently used
- Velcro or elastic strap placed circumferentially around proximal forearm, designed to reduce contractile force on wrist extensor or flexor attachment at the elbow.



**A.** Elbow hyperextension taping

**B.** Hinged elbow hyperextension brace

**Figure 61.3.** Elbow hyperextension taping and bracing.

**Figure 61.4.** Counter-force strap for lateral epicondylosis.

### Wrist Sprain

- Taping technique (Fig. 61.5A): Tape wrist circumferentially; may use a foam pad or "fan" of tape over the ventral or dorsal wrist to prevent flexion or extension
- Wrist brace (Fig. 61.5B): soft elastic support with or without plastic or metal ventral and/or dorsal supports; braces that include the thumb (thumb spica splint extending across wrist to distal forearm, see Fig. 61.6C) are necessary to support radial-sided carpal bones such as the scaphoid.

### Thumb Sprain

- Thumb spica taping: (Fig. 61.6A, Video 61-2): Pieces of tape encircle thumb in a tear-drop pattern, anchoring ends on wrist; traction may be placed on either end of tape, offering support to any part of the first metacarpophalangeal joint.
- Thumb spica splint: (Fig. 61.6B and C): Neoprene wrap or brace with or without plastic or metal supports, which may extend to just below the radiocarpal joint or across the wrist to include the distal third of the forearm

### Proximal/Distal Interphalangeal Sprain

- Buddy taping is used to tape the injured finger to the adjoining uninjured finger. The tape joins the fingers at proximal and intermediate phalanges (Fig. 61.7).

## SELECTED TAPING/BRACING OF LOWER EXTREMITY INJURIES

### Hip Injury

- Multidirectional hip joint and adjacent buttock make bracing and taping of this area difficult. Elastic hip and groin spica wraps may be used in a figure-of-eight pattern around the waist and upper thigh.

**A.** Wrist taping

**B.** Wrist brace

**Figure 61.5.** Wrist taping and bracing.

**A.** Thumb spica taping

**B.** Thumb spica brace

**C.** Thumb spica brace

**Figure 61.6.** Thumb spica taping and bracing.

## ACL Injury

- Hinged knee brace (Fig. 61.8A): May be used for initial compression, support, and stability; custom braces may be used when returning to sport (Fig. 61.8B); evidence supporting efficacy in reducing and/or preventing injury limited.

**Figure 61.7.** Buddy taping.

**A.** Hinged knee brace

**B.** Custom ACL knee brace

**Figure 61.8.** ACL injury bracing.

## Medial or Lateral Collateral Ligament (MCL/LCL) Injury

- Hinged knee brace (Fig. 61.8A): Metal hinges provide medial and lateral support. Range of motion can be restricted using flexion and/or extension stops in order to decrease traction/stress on MCL or LCL.

## Patellofemoral Pain/Injury

- Patellofemoral taping (Fig. 61.9, Video 61-3): Used for patellar maltracking, fat pad impingement; Cover-Roll stretch tape is used over the anterior knee to protect the skin. Nonstretch tape is used to "pull" the patella into appropriate alignment. Patella may be pulled in any direction; important to assess mechanics with single or double leg squatting before and after taping.
- Medial/lateral patella-stabilizing brace (Fig. 61.10): Foam/plastic buttress provides medial or lateral patellar support, many adjustable. Certain braces use a pulley system that offers more support as the knee moves into flexion; can be used for patellar maltracking, instability and subluxation, or dislocation.

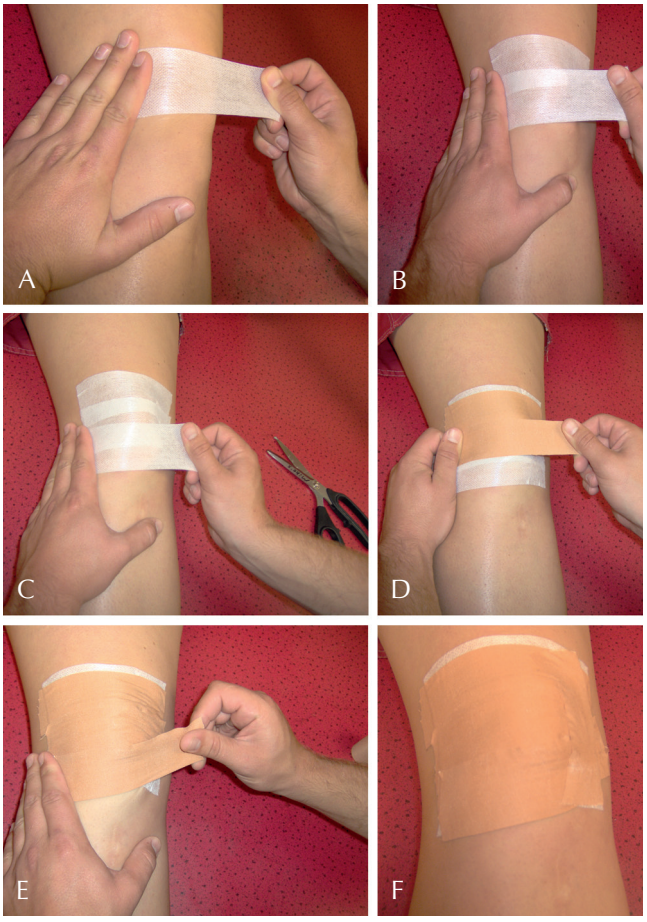



**Figure 61.9.** Patellofemoral taping.

**Figure 61.10.** Patella-stabilizing brace.

## Patellar Tendinopathy

- Patellar strap (Fig. 61.11): Circumferential elastic strap around the proximal leg beneath the patella, designed to reduce contractile force on the patellar tendon

## Knee Osteoarthritis

- Braces may be used to "unload" the affected medial or lateral compartment. Certain braces use a pulley system that allows more "unloading" as the knee moves into flexion. These braces may also be used for condylar bone bruises and after meniscal or cartilage repair procedures.

## Ankle Sprain

- Taping should not be performed on an acute sprain.
- Various taping techniques are used to support and provide protection to injured structures when an athlete returns to activity.
- Slip-on elastic support provides even compression to decrease edema in an acute sprain.
- Lace-up ankle brace uses medial and lateral stays and sometimes additional straps to restrict inversion/eversion (see Fig. 61.1B).
- Stirrup splints, based on semirigid orthosis, use Velcro straps to hold in place; designed for rehabilitative and functional uses. All are universally sized.

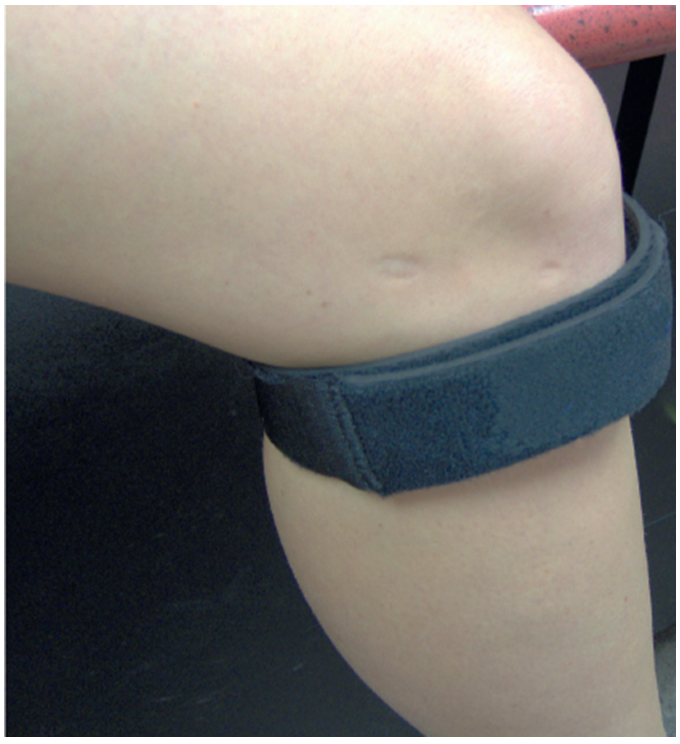


**Figure 61.11.** Patellar tendon strap.

- Inflatable ankle air cast/brace uses adjustable air pressure linings to improve individual fit, decrease edema, and prevent excessive inversion/eversion (Fig. 61.12).
- Gel cast/brace uses gel-filled linings to improve individual fit; can be placed in freezer to be used as a form of cryotherapy.

Figure 61.12. Ankle air cast.

Figure 61.14. Dorsal night splint.

Figure 61.13. Arch taping.

Figure 61.15. Turf toe taping.

## Plantar Fasciitis

- Arch taping (Fig. 61.13; Video 61-4): Supports longitudinal and transverse arch, which relieves strain on the plantar fascia
  - Patient lies prone with foot off the end of the examination table
  - A nonstretch, 0.5–0.75-inch athletic tape used to support the longitudinal arch by taping from the plantar aspect of the first metatarsal head around the posterior heel to the plantar aspect of the fifth metatarsal head.
  - A nonstretch, 1–1.5-inch athletic tape is used to support the transverse arch by taping from the lateral to the medial arch.
  - Distal and proximal ends of the strips supporting the longitudinal arch may be removed for comfort.
- Dorsal night splint (Fig. 61.14): Keeps the ankle at 90 degrees while sleeping, thus preventing ankle plantarflexion and offering gentle stretch of plantar fascia to reduce swelling at its medial calcaneal attachment.

## Turf Toe

- Taping (Fig. 61.15; Video 61-5): Anchor placed circumferentially around forefoot; pieces of tape placed in a tear-drop pattern with ends attaching either to dorsal or plantar side of the foot depending on the need for dorsal or plantar first metatarsophalangeal joint support and whether dorsiflexion or plantarflexion of the toe is being restricted.

## Orthotics

- **Common indications:** Lower-extremity kinetic chain conditions resulting from excessive pronation, cavus foot, or other foot dysfunctions or mechanical faults
- **Soft orthotics** provide inexpensive over-the-counter solution; easy break-in, some with a limited lifespan
- **Rigid/hard orthotics** provide customized solution for certain athletes, particularly road runners not involved in agility activities; good motion control, minimal cushioning

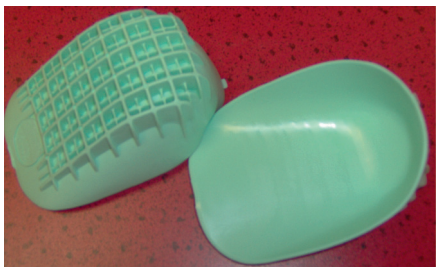


**Figure 61.16.** Tuli's heel cups.

**Figure 61.17.** Metatarsal arch pads.

- **Semirigid orthotics** provide support similar to rigid/hard orthotics for athletes involved in agility sports; good combination of support and cushioning; most popular for athletes
- **Sorbothane viscoelastic insoles** reduce impact-loading forces, particularly at the heel, provide minimal support.
- **Steel or carbon (e.g., Dynaflex or Spring Plate) shoe inserts** provide support to metatarsal fractures, midfoot sprains, and limit dorsiflexion of first metatarsophalangeal joint; essentially convert normal athletic shoe to "stiff" shoe and may be used in weaning from cast or boot
- **Heel cups** (e.g., Tuli's) decrease impact and improve shock absorption capabilities of calcaneal fat pad (Fig. 61.16)
- **Longitudinal arch pads** or Barton's wedges provide symptomatic relief from painful foot conditions; may be held in place with tape or glue or may use a self-adhesive to affix to shoe or orthotic
- **Metatarsal arch pads** and bars provide symptomatic relief from painful foot conditions such as interdigital neuroma and metatarsalgia; may be held in place with tape, glue, or self-adhesive (Fig. 61.17); used to splay and elongate metatarsals and tarsals

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- Video 61-1 Ankle taping
- Video 61-2 Thumb spica taping
- Video 61-3 Patellofemoral taping
- Video 61-4 Arch taping
- Video 61-5 Turf toe taping

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# INJECTIONS IN THE ATHLETE

# 62

Andrew V. Pytiak • Armando F. Vidal • Michelle Wolcott

## GENERAL PRINCIPLES

- Knowledge of anatomy is essential in administering injections safely and effectively.
- Use of local anesthetic injections in athletes may reduce the number of games missed because of injury but carries a theoretical risk of worsening the injury.
- Corticosteroid injections are widely used in the treatment of athletic injuries because of their potent anti-inflammatory properties, but these are not without undesirable side effects.
- Corticosteroid injections may not offer a clear therapeutic advantage over anti-inflammatory medications alone.
- Both physicians and athletes should be aware of any restriction regarding the use of corticosteroids during a competition.
  - The World Anti-Doping Agency requires the completion of therapeutic use exemption before administration of corticosteroids.
  - The International Olympic Committee Medical Code requires that any team doctor wishing to administer corticosteroids to an athlete by local or intra-articular injection must give written notification to the relevant medical authority before the competition.
  - **NCAA Statement on corticosteroid injections:** Corticosteroid injections should only be administered if a therapeutic effect is medically warranted, and the student-athlete is not subject to any significant short- or long-term risks.
  - After careful consideration, the physician and athlete must determine that the benefits outweigh the risks before proceeding with an injection.

## STERILE TECHNIQUE FOR INJECTIONS

- Mark the injection site by pressing the tip of a capped needle against the skin.
- Prep the injection site with Betadine or another appropriate antiseptic solution.
- Use prepacked sterile needles and syringes.
- Use single-dose vials of an injectable agent whenever possible.
- Change needles after drawing up the solution.
- Wear sterile gloves.

## COMFORT MEASURES

- **Ethyl chloride** is a topical spray that rapidly cools the skin, providing topical anesthesia.
- Benefit of a **subcutaneous local anesthetic** may be limited by subcutaneous injection pain.
- **EMLA cream** must be applied at least 1 hour before injection but has been shown to be highly effective.

## ACCURACY OF INJECTION

- Knowledge of surface landmarks and tactile feedback are essential for accurate needle placement.
- Clinician experience alone does not necessarily improve the accuracy of an injection.
- Joint aspiration in the presence of an effusion may assist in intra-articular needle placement but is not a guarantee.
- Injecting 1–2 mL of air with the injection may help verify intra-articular injection of the knee or shoulder. The presence of an audible "squishing" with range of motion indicates a successful intra-articular injection.
- Ultrasound guidance has been shown to improve the accuracy and clinical effects of certain injections in the hands of a skilled ultrasonographer.
- Image guidance has not shown a definitive advantage over traditional techniques for most injections, although it may be useful in certain situations, such as in obese patients or in patients who have failed to respond to previous injections.

## COMMON AGENTS

### Local Anesthetics

- **Lidocaine (Xylocaine)** is the most widely used anesthetic. Rapid onset of action with limited duration, approximately 30 minutes; maximum dose: 300 mg (30 mL of 1% lidocaine)
- **Bupivacaine (Marcaine)** has a slow onset of action with prolonged duration of effect, for up to 8 hours; maximum dose: 175 mg (70 mL of 0.25% bupivacaine)
- A combination of lidocaine and bupivacaine allows the rapid onset of action with a prolonged duration of effect.

### Corticosteroids

- Less soluble
  - Methylprednisolone acetate (Depo-Medrol)
  - Triamcinolone acetonide (Kenalog)
  - Triamcinolone hexacetonide (Aristospan)
- More soluble
  - Betamethasone sodium phosphate (Celestone)
  - Dexamethasone sodium phosphate (Decadron)
  - Prednisolone sodium phosphate (Hydeltrasol)
- Dosage
  - Dosage requirements vary and should be individualized on the basis of the condition being treated and the response of the patient (Table 62.1).
  - In chronic cases, injections may be repeated at intervals ranging from 1 week to ≥5 weeks depending on the degree of relief obtained from the initial injection.
  - No clear safety guidelines exist regarding the frequency and maximum number of injections. Judicious use of injections is recommended.

## MECHANISM OF ACTION

### Local Anesthetics

- Membrane-stabilizing agents cause a reversible conduction block along nerve fibers.
- Smaller nerve fibers are more sensitive, allowing inhibition of pain signals while sparing pressure and proprioceptive fibers.
- Maximum plasma concentrations of local anesthetic are achieved within 10–25 minutes.
- Avoid preparations containing epinephrine for intra-articular or soft tissue injections; epinephrine causes vasoconstriction, which prolongs the anesthetic effect when used in the skin.

### Corticosteroids

- Interfere with inflammatory cell-to-cell adhesion and migration through the vascular endothelium.
- Inhibit the synthesis of cyclooxygenase-2 (COX-2) and various proinflammatory cytokines.

**TABLE 62.1 SUGGESTED DOSES FOR CORTICOSTEROID PREPARATIONS**

| Corticosteroid Agent | Large Joint | Medium Joint | Small Joint | Soft Tissue |
|---|---|---|---|---|
| Methylprednisolone acetate (Depo-Medrol) | 20–80 mg | 10–40 mg | 4–10 mg | 4–30 mg |
| Betamethasone sodium phosphate (Celestone) | 1.0 mg | 0.5–1.0 mg | 0.25–0.5 mg | 0.5–1.0 mg |

- Stimulate gluconeogenesis and catabolic activity in muscle, skin, lymphoid, adipose, and connective tissue
- Solubility determines duration of action and extent of systemic effects.
  - **Insoluble:** Average duration of action is longer for less soluble agents; primarily display local effects
  - **Soluble:** Diffuse more readily from the injected region and may exert greater systemic effects; may be more useful in extra-articular or soft tissue injections
- Average duration of action is 1–3 weeks and is longer for less soluble agents.

## SIDE EFFECTS

### Local Anesthetics

**Anaphylaxis:** Occurs from acute mast cell degranulation and is characterized by laryngeal edema, bronchospasm, and hypotension; the cause of local toxicity is allergic reaction to para-aminobenzoic acid (PABA). PABA is a metabolic product of the degradation of the ester class of local anesthetics, such as procaine (Novocain), and to a lesser extent, amide class anesthetics such as lidocaine; it is also a metabolic by-product of methylparaben, a preservative in multidose vials of lidocaine.

**Toxicity:** Usually the result of inadvertent intravenous injection; primary target organ is central nervous system resulting in altered speech, muscle twitching, and seizures. Cardiovascular toxicity (e.g., ventricular arrhythmias) may also occur. Aspirate before injection to avoid inadvertent intravenous injection. Compared with other local anesthetics, bupivacaine is markedly cardiotoxic.

**Chondrotoxicity:** Emerging evidence suggests that both lidocaine and bupivacaine may have negative effects on articular cartilage health and chondrocyte viability.

### Corticosteroids

**Postinjection flare:** Most common side effect; related to steroid crystal synovitis; self-limiting; typically lasts for <12 hours; analgesic therapy and ice packs offer effective relief.

**Facial flushing:** Subjective sensation of warmth in the face and upper trunk

**Poor diabetic control:** May increase hepatic glucose production and antagonize insulin effects; close monitoring is suggested following a corticosteroid injection

**Subcutaneous fat atrophy and skin depigmentation:** May occur after a single injection; avoid repeated subcutaneous or superficial injections. Subcutaneous fat atrophy is particularly problematic in the plantar surface of the foot. Skin depigmentation occurs more commonly in dark-skinned individuals.

**Tendon rupture:** Dose-dependent decrease in tenocyte proliferation and reduced collagen production by tenocytes. Care should be taken to avoid direct intratendinous injection, which can result in this complication.

**Steroid-induced arthropathy and cartilage damage:** Destruction of articular cartilage and a decrease in cartilage matrix production has been shown in animal studies. Although not reported in humans, a theoretical risk remains.

**Infection:** Incidence of joint sepsis varies from 1 in 3,000 to 1 in 50,000 for corticosteroid injections. Informed consent should be obtained before any invasive procedure. Use of good sterile technique minimizes this risk.

**Anaphylaxis:** May occur even after previous uneventful injections

**Hypothalamic–pituitary–adrenal axis suppression:** Suppression is mild and transient. Avoid simultaneously injecting multiple large joints. Prolonged suppression has been reported.

### Other

**Syncope:** Patients who feel lightheaded or overly apprehensive should lie down. Protect the airway in the event of loss of consciousness. Reassure the patient.

## CONTRAINDICATIONS

**Infection:** Overlying cellulitis, bacteremia, or septic arthritis

**Fracture or unstable joint:** Risk of worsening injury

**Tendinopathy:** Risk of tendon rupture if injected directly into tendon (e.g., Achilles tendon)

**History of medication allergy or anaphylaxis following injection:** Previous uneventful injection does not eliminate possibility of future reactions

**Coagulopathy or anticoagulation therapy:** Risk of bleeding at the injection site

**Poorly controlled diabetes:** Corticosteroids may result in temporary exacerbation of diabetes.

**Prosthetic joint:** Risk of infection

## UPPER EXTREMITY INJECTIONS

### Shoulder

#### Subacromial Space

**Indications:** Subacromial bursitis and rotator cuff tendinitis

**Technique:**

1. Allow the arm to hang at the patient's side to open up the subacromial space.
2. Palpate the lateral border of acromion.
3. The subacromial bursa is deep to the acromion and superficial to the rotator cuff (Fig. 62.1A).
4. Insert the needle angled slightly superiorly at the midpoint of acromion and approximately 2–3 cm lateral to the lateral border of the acromion
5. Slowly withdraw the needle while injecting.

**Considerations:** Insertion of the needle too far medially may result in injection into the supraspinatus muscle belly. Up to 30% of subacromial injections may miss the subacromial space. Ultrasound guidance has been shown to improve the accuracy of subacromial injections.

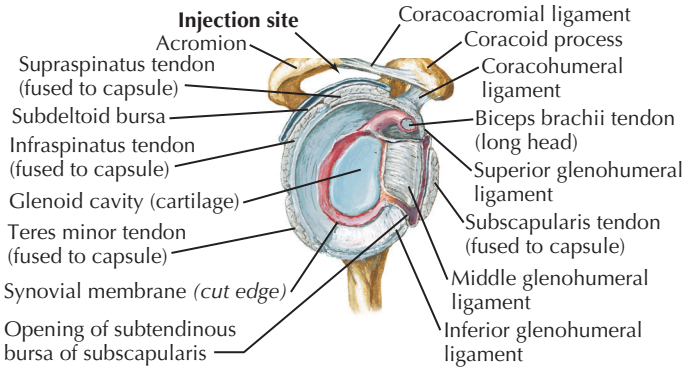
#### Glenohumeral Joint

**Indications:** Labral tears, chondral defects, bone contusions, diagnostic injections

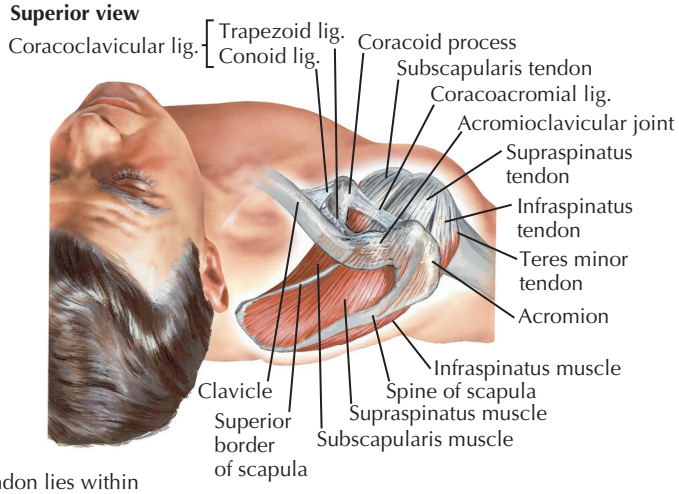
**Technique (anterior):**

1. Approach the glenohumeral joint through the rotator interval between the superior border of the subscapularis tendon

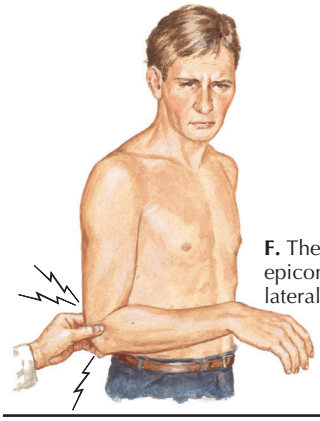
**A.** The subacromial bursa lies deep to the acromion and superficial to the rotator cuff. The glenohumeral joint is approached anteriorly through the interval between the supscapularis and supraspinatus tendons.



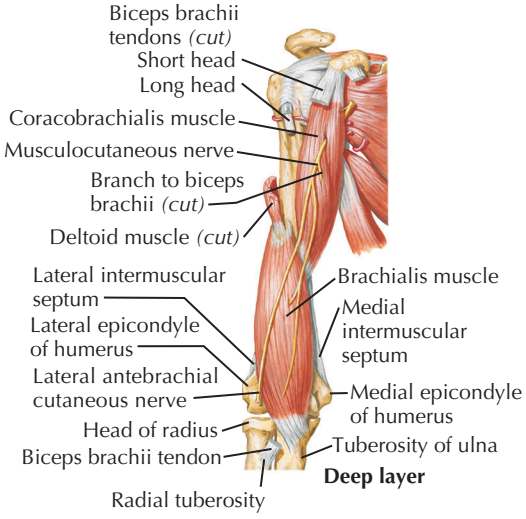

**B.** The acromioclavicular joint is identified by palpating medially from the anterolateral border of the acromion.



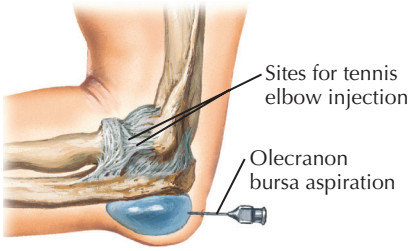

**C.** During an AC joint injection, the needle is directed at a 30° angle from the vertical to follow the normal anatomic orientation of the AC joint.

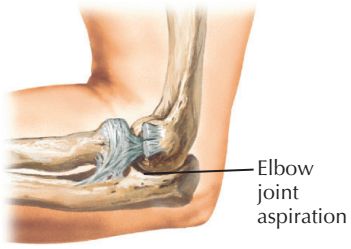


**D.** The long head of the biceps tendon lies within a sheath in the bicipital, or intertubercular, groove.




**E.** Approach the elbow joint through the soft spot between the olecranon, lateral epicondyle, and radial head. The area of maximum fluctuance is identifed for needle placement into the olecranon bursa.



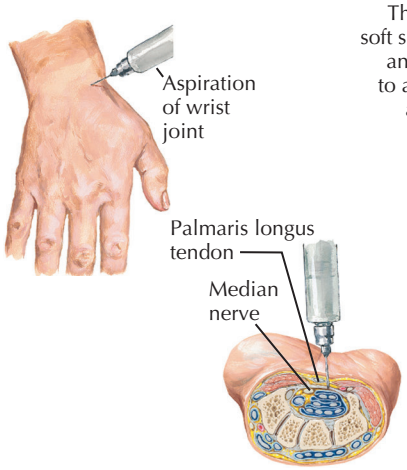




**F.** The point of maximum tenderness in lateral epicondylitis typically lies 1 cm distal to the lateral epicondyle.

**Epicondylitis** (tennis elbow)
Exquisite tenderness approximately 1 cm distal to the lateral epicondyle

F. Netter M.D.
C. Machado M.D.

**G.** The needle is inserted ulnar to the palmaris longus tendon to avoid direct injury to the median nerve.




The needle is inserted into the soft spot distal to Lister's tubercle and angled slightly proximally to account for the normal volar angulation of the wrist joint.

The injection is placed into the gap between the abductor pollicis longus and extensor pollicis brevis tendons at the base of the thumb metacarpal and directed proximally at a 30° angle. The superficial branch of the radial nerve lies in close proximity to the injection point.

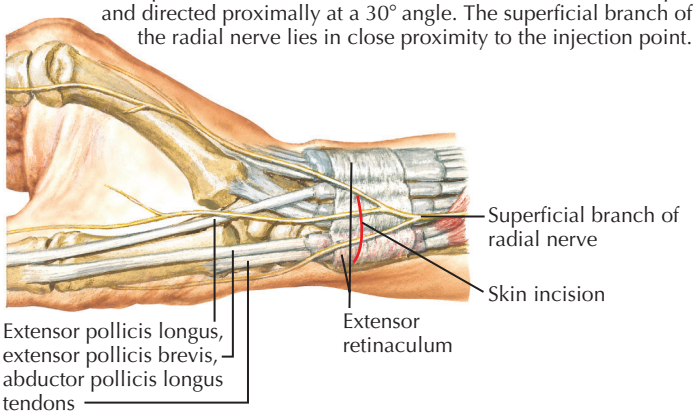



**Figure 62.1.** Upper extremity injections.

and the anterior border of the supraspinatus tendon (see Fig. 62.1A).
2. Insert the needle at the midpoint between the anterolateral border of the acromion and coracoid, directing the needle slightly medially. Typically, a soft spot can be palpated in this region.

**Technique (posterior):**
1. Palpate the posterolateral corner of acromion.
2. Palpate the soft spot of the glenohumeral joint approximately 1–2 cm medial and 2–3 cm inferior to the posterolateral corner of acromion.
3. Palpate the coracoid anteriorly with the opposite hand.
4. Insert the needle into the soft spot, directing the needle toward the coracoid.

**Considerations:** Use caution to avoid direct injury to the articular cartilage of the humeral head and glenoid. Anterior injections may offer a higher rate of accuracy than posterior injections.

### Acromioclavicular (AC) Joint

**Indications:** AC joint sprain, grades I and II AC joint separation, AC joint arthropathy, and distal clavicle osteolysis

**Technique:**
1. Identify the lateral border of the acromion anteriorly and palpate medially to identify the superior aspect of the AC joint (see Fig. 62.1B and C).
2. The AC joint articulation runs obliquely medially from superior to inferior.
3. Direct the needle medially at a 30-degree angle from the vertical (i.e., parallel to the joint line) (see Fig. 62.1B and C).
4. May inject lidocaine subcutaneously to locate the joint and switch syringe to inject steroid

**Considerations:** Use a smaller total volume for the AC joint, typically 1–2 mL.

### Long Head of the Biceps

**Indications:** Biceps tendinopathy

**Technique:**
1. The biceps tendon lies within a sheath in the bicipital groove (Fig. 62.1D).
2. Palpate the point of tenderness at the anterior aspect of the shoulder.
3. Insert the needle at the most superior point of tenderness, and direct the needle inferiorly, parallel with the tendon.

**Considerations:** Withdraw the needle slightly if resistance is encountered to prevent intratendinous injection. Ultrasound guidance may be useful to confirm needle placement in the tendon sheath.

## Elbow

### Elbow Joint

**Indications:** Chondral defects, bone contusions, posteromedial osteophytes, aspiration of effusion, and synovitis

**Technique:**
1. Position patient in slight elbow flexion with hand resting in lap.
2. Palpate the soft spot between the olecranon, lateral epicondyle, and radial head (see Fig. 62.1E).
3. Insert the needle into the center of the triangle formed by the aforementioned landmarks.
4. Direct the needle toward the medial epicondyle.

**Considerations:** The joint is relatively superficial—repeated injections may result in subcutaneous fat atrophy or skin depigmentation. Avoid overpenetration of the needle to prevent any direct injury to the articular cartilage of the elbow.

### Olecranon Bursa

**Indications:** Olecranon bursitis

**Technique:**
1. Position the elbow in slight flexion with the hand resting in the lap.
2. Insert the needle into the area of maximum fluctuance (see Fig. 62.1E).

**Considerations:** Attempted aspiration before injection may be helpful in determining whether an infection is present. Avoid injection in the setting of infection.

### Common Flexor and Extensor Origin

**Indications:** Medial epicondylitis, lateral epicondylitis

**Technique:**
1. Position elbow in slight flexion with hand resting in lap.
2. Supinate the forearm for common flexor origin injection. Pronate the forearm for common extensor origin injection.
3. Insert the needle at the point of maximum tenderness, typically 1 cm distal to the epicondyle (see Fig. 62.1F).

**Considerations:** Radial tunnel syndrome is a rare compression neuropathy that may be confused with lateral epicondylitis.

## Wrist

### Wrist Joint

**Indications:** Triangular fibrocartilage complex (TFCC) tear, scapholunate ligament sprain, bone contusion

**Technique:**
1. Position the wrist in slight flexion.
2. Palpate Lister's tubercle on the dorsal surface of the distal radius.
3. Identify the soft spot approximately 1 cm distal to Lister's tubercle.
4. Direct the needle slightly proximally to account for the 11 degrees of volar angulation at the distal radius (see Fig. 62.1G).

**Considerations:** Scaphoid fracture must be ruled out definitively before proceeding with wrist joint injection.

### Carpal Tunnel

**Indications:** Carpal tunnel syndrome

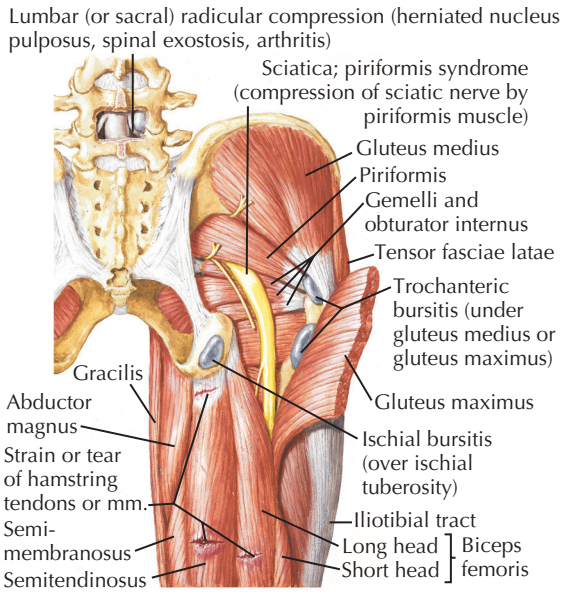
**Technique:**
1. Position wrist in 30 degrees of dorsiflexion.
2. The proximal border of the carpal tunnel lies at the distal wrist crease. The median nerve is ulnar to the flexor carpi radialis and deep to the palmaris longus (see Fig. 62.1G).
3. Palpate the palmaris longus tendon at the proximal wrist crease. Use the wrist midline if the palmaris longus tendon is absent.
4. Direct the needle distally toward the middle/ring finger at a 30- to 45-degree angle.
5. Insert the needle 1–2 cm until no resistance.

**Considerations:** Paresthesias indicate that the needle is in the median nerve—withdraw the needle and redirect more ulnarly. Direct injection into the median nerve can cause irreversible damage. Avoid injecting large volumes into the carpal tunnel because this may exacerbate compression of the median nerve.

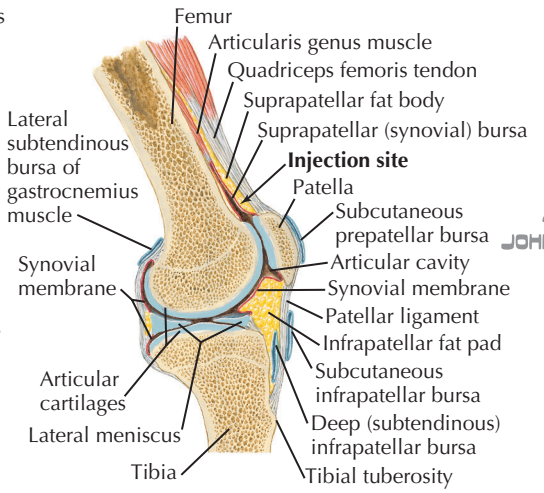
### First Dorsal Compartment

**Indications:** De Quervain's tenosynovitis

**Technique:**
1. Position the thumb in abduction and extension to facilitate identification of the radial wrist tendons.
2. Palpate the abductor pollicis longus and extensor pollicis brevis tendons in the first dorsal compartment (see Fig. 62.1G).

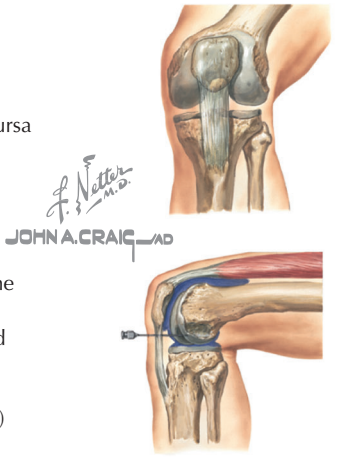



The trochanteric bursa lies deep to the iliotibial band over the greater trochanter. The injection is administered at the point of maximum tenderness.

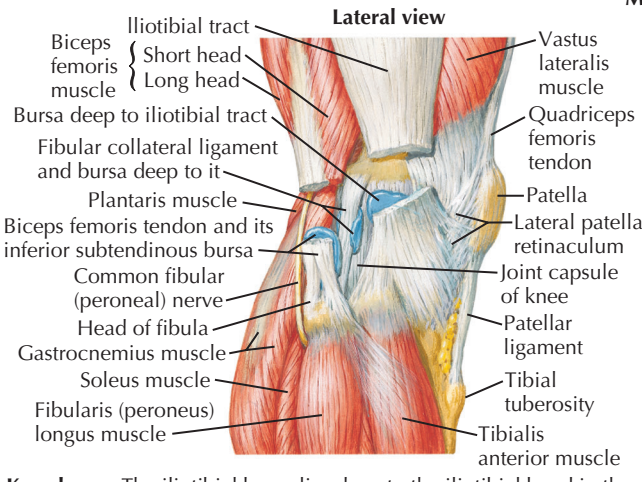



**Sagittal section (lateral to midline of knee)**

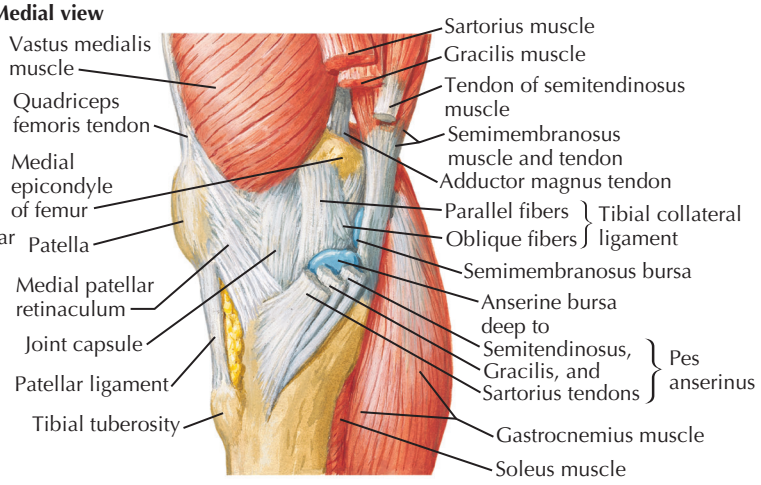
Knee injection using the lateral suprapatellar approach enters the joint parallel to the superior pole of the patella, just lateral to the vastus lateralis tendon. The prepatellar bursa is a subcutaneous bursa superficial to the patella.




Knee injection using the anterolateral approach enters the joint in the soft spot at the midpoint between the infero-lateral border of the patella and the lateral joint line.




**Knee bursa.** The iliotibial bursa lies deep to the iliotibial band in the region of the lateral epicondyle.




The pes anserinus bursa is deep to the sartorius, gracilis, and semitendinosus tendons and superficial to the medial collateral ligament.

**Figure 62.2.** Knee injections.

3. Insert the needle in the gap between the two tendons at the base of the thumb metacarpal.
4. Direct the needle at a 30-degree angle proximally toward the radial styloid.

**Considerations:** Redirect the needle slightly if resistance is encountered to prevent intratendinous injection. Paresthesias may indicate that the needle is in the superficial branch of the radial nerve.

### Flexor Tendon Sheath

**Indications:** Trigger finger

**Technique:**

1. Position patient with forearm supinated and fingers extended.
2. Palpate the nodule in the flexor tendon.
3. Insert the needle at the distal palmar crease.
4. Direct the needle distally at a 30-degree angle into the flexor tendon sheath.

**Considerations:** Withdraw needle slightly if resistance is encountered to avoid intratendinous injection. Digital nerve anesthesia occurs with injection of local anesthetic.

## LOWER EXTREMITY INJECTIONS

### Hip

#### Trochanteric Bursa

**Indications:** Trochanteric bursitis, bone contusion

**Technique:**

1. Place patient in lateral decubitus position with hip and knee flexed.
2. Insert the needle perpendicular to the skin at the point of maximal tenderness over the greater trochanter.
3. Inject deep to the iliotibial band into the trochanteric bursa (Fig. 62.2).

**Considerations:** If the needle is advanced into the bone, withdraw slightly and inject. In larger patients, consider using a spinal needle.

### Knee

#### Knee Joint

**Indications:** Bone contusion, meniscus tear, chondromalacia patellae, plica syndrome

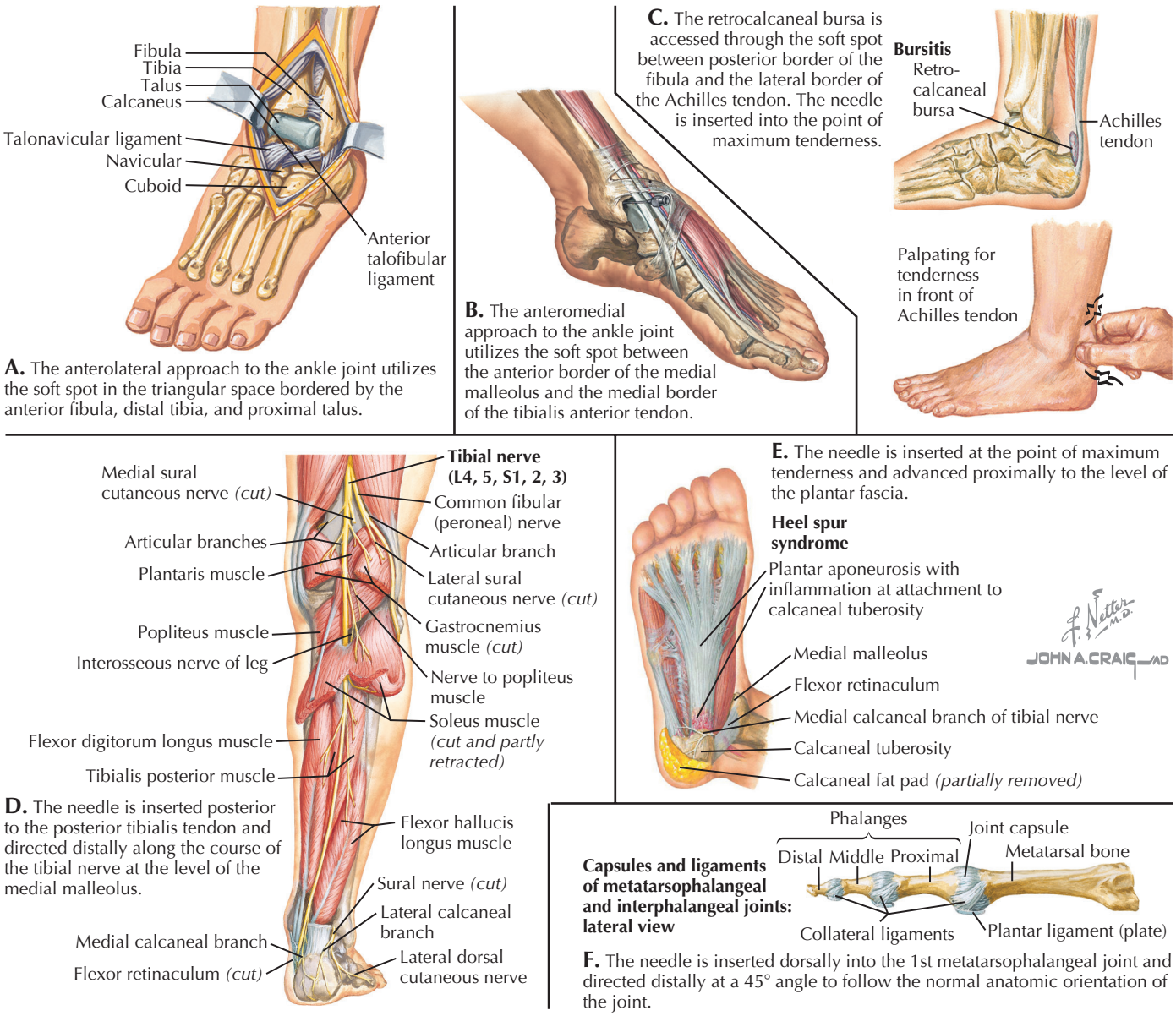



**Figure 62.3.** Ankle and foot injections.

**Technique (lateral suprapatellar):**

1. Position patient with the knee in full extension.
2. Palpate the superior border of the patella.
3. Push the patella laterally to tent the capsule.
4. Insert the needle inferior to the vastus lateralis tendon and parallel to the superior border of the patella (see Fig. 62.2).

**Technique (anterolateral):**

1. Position patient with the knee flexed to 90 degrees and the leg hanging off the examination table to distract the joint.
2. Palpate the lateral border of patellar tendon.
3. Palpate the soft spot at the midpoint between the inferolateral border of the patella and the lateral joint line (see Fig. 62.2).
4. Direct the needle toward the notch approximately 0.5–1 cm lateral to the patellar tendon.

**Considerations:** The lateral suprapatellar approach may allow for more accurate intra-articular placement of the needle. With an anterolateral approach, use caution to avoid direct injury to the articular cartilage of the lateral condyle.

## Prepatellar Bursa

**Indications:** Prepatellar bursitis

**Technique:**

1. Position patient with the knee extended.
2. Prepatellar bursa is superficial to the patella (see Fig. 62.2).
3. Insert the needle into area of maximum fluctuance.

**Considerations:** Attempted aspiration before injection may be helpful in determining whether an infection is present. Avoid injection in the setting of infection.

## Pes Anserine Bursa

**Indications:** Pes anserine bursitis

**Technique:**

1. Position patient with the knee in 90 degrees of flexion.
2. The pes anserine is the insertion of the sartorius, gracilis, and semitendinosus tendons. The bursa is deep to the tendons and superficial to the medial collateral ligament (see Fig. 62.2).

3. Insert the needle perpendicular to the skin at the point of maximum tenderness deep to the pes anserine tendons.

**Considerations:** Advance needle until no resistance is met to avoid intratendinous injection. If the needle is advanced into the bone, withdraw 2–3 mm and inject.

### Iliotibial Bursa

**Indications:** Iliotibial band syndrome
**Technique:**

1. Place patient in lateral decubitus position with hip and knee slightly flexed.
2. Palpate the lateral epicondyle of the femur. The bursa lies deep to the iliotibial band in the region of the lateral epicondyle (see Fig. 62.2).
3. Insert the needle perpendicular to the skin at the point of maximum tenderness and inject.

**Considerations:** Inflammation of the iliotibial band may occur more distally near its attachment at the Gerdy's tubercle.

## Ankle

### Ankle Joint

**Indications:** Osteochondral defect, bone contusion, ankle sprain
**Technique (lateral):**

1. Position the ankle in slight plantar flexion.
2. Identify the soft spot in the triangular space bordered by the anterior fibula, distal tibia, and proximal talus (Fig. 62.3A).
3. Direct the needle slightly proximally and medially.

**Technique (medial):**

1. Position the ankle in slight plantar flexion.
2. Palpate the soft spot between the anterior border of the medial malleolus and the medial border of the tibialis anterior tendon (see Fig. 62.3B).
3. Direct the needle slightly proximally and laterally.

**Considerations:** Use caution to avoid direct injury to the articular cartilage of the tibial plafond or talar dome.

### Retrocalcaneal Bursa

**Indications:** Retrocalcaneal bursitis
**Technique:**

1. Place the patient in the prone position with the foot in slight dorsiflexion.
2. Palpate the soft spot between the posterior border of the fibula and the lateral border of the Achilles tendon (see Fig. 62.3C).
3. Insert the needle at the point of maximum tenderness anterior to the Achilles tendon.

**Considerations:** Withdraw the needle and redirect if resistance is encountered to prevent intratendinous injection. Avoid overpenetration to prevent inadvertent injection into the medial neurovascular structures. Retrocalcaneal bursitis may be difficult to distinguish from Achilles tendinitis.

### Tarsal Tunnel

**Indications:** Tarsal tunnel syndrome
**Technique:**

1. Palpate the posterior tibialis tendon.
2. Insert the needle posterior to the posterior tibialis tendon at the level of the medial malleolus (see Fig. 62.3D).
3. Direct the needle distally at a 30-degree angle from the skin surface, parallel to the posterior tibialis tendon.

**Considerations:** Aspirate before injecting to avoid inadvertent intravascular injection. Paresthesias indicate that the needle is in the tibial nerve. Withdraw the needle slightly and redirect if resistance is encountered to prevent intratendinous injection.

## Foot

### Plantar Fascia

**Indications:** Plantar fasciitis
**Technique:**

1. Place the patient in the prone position with the foot in slight dorsiflexion.
2. The area of inflammation typically occurs at the medial aspect of the plantar fascia near the medial process of the calcaneal tuberosity, just distal to the heel pad (see Fig. 62.3E).
3. Insert the needle at the point of maximum tenderness and advance proximally at a 45-degree angle to the level of the plantar fascia.

**Considerations:** Avoid injection into the heel pad to prevent subcutaneous fat pad atrophy. If the needle is advanced into the bone, withdraw slightly and inject.

### First Metatarsophalangeal (MTP) Joint

**Indications:** Turf toe, chondral defects
**Technique:**

1. Position the great toe in slight plantar flexion. Passively flex and extend the great toe to aid in identifying the joint space.
2. Insert the needle on the dorsal surface of the MTP joint (see Fig. 62.3F).
3. Direct the needle distally at a 45-degree angle.

**Considerations:** Ultrasound can be particularly useful to visualize and avoid dorsal osteophytes. The joint is relatively superficial—repeated injections may result in subcutaneous fat atrophy or skin depigmentation. Avoid deep penetration with the needle to prevent direct injury to the articular cartilage.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 63 DIAGNOSTIC IMAGING IN SPORTS MEDICINE

Colin D. Strickland

## GENERAL PRINCIPLES

- Imaging in sports medicine plays an increasingly important role in the diagnosis of injury and in decision making regarding return to play.
- Increasingly sophisticated imaging modalities allow for the precise diagnosis of various patterns of injury but also present the care provider with a bewildering range of tests.
- Continued participation of older athletes in sporting activities also presents a challenge as degenerative findings become intermingled with sports-related injuries detected by imaging studies.

## INDICATIONS FOR IMAGING

- The approach to imaging in sports medicine mirrors the philosophy applied in clinical medicine as a whole. Imaging should primarily be pursued only when it is likely to alter the management of the patient and when the benefits outweigh the risks. The cost and availability of imaging studies are also important factors to consider (Box 63.1).
- It is important to consider the risks of exposure to ionizing radiation, particularly in young or pregnant patients. In addition, discomfort, expense, and inconvenience of imaging to the patient are valid concerns.

## IMAGING TECHNIQUES

- Musculoskeletal imaging techniques used in sports medicine provide an excellent depiction of anatomic structures and often facilitate a precise diagnostic accuracy. Imaging modalities differ in their mode of image generation and in terms of cost and radiation exposure.
- Radiographs (plain radiographs) create two-dimensional images of anatomy and typically serve as the first-line imaging study. Computed tomography (CT) also uses ionizing radiation to create cross-sectional images. Nuclear medicine examinations (including bone scintigraphy) detect gamma rays emitted from a radiopharmaceutical administered to the patient. Each of these modalities requires exposure of the patient to ionizing radiation.
- Magnetic resonance imaging (MRI) does not use ionizing radiation but is contraindicated for certain patients with metallic implanted medical devices. Ultrasound is generally safe, although tissue heating may be a concern.
- The use of the modern picture archiving and communication system (PACS) software allows diagnostic images to be stored and shared widely, which has greatly increased the portability of imaging information.
- The judicious use of ionizing radiation is critical in protecting the safety of sporting patients, particularly the young who face greater theoretical lifetime risks from exposure to ionizing radiation. Some common examinations and typical dose levels are listed in Table 63.1.
- The diagnosis and management of injuries in sports medicine greatly relies on accurate history and physical examination findings as well as an understanding of the mechanism of injury. Communicating this information at the time of an imaging request allows the radiologist to interpret studies in the appropriate clinical context and provide the most accurate report.
- A close working relationship among sports medicine providers, radiologic technologists, and diagnostic radiologists helps foster an efficient system and ensure that appropriate care is delivered to the patient.

## RADIOGRAPHS

- Radiographs (plain radiographs) use ionizing radiation and provide excellent depiction of bony anatomy with very high spatial resolution. The PACS software allows radiographic images to be windowed and leveled, which reveals additional detail in the soft tissues.
- **Advantages:** The benefits of radiographic evaluation are the high spatial resolution and high sensitivity in detecting subtle fractures or dislocations. Radiographs are widely available, and a multitude of standardized views have been developed to evaluate each bone and joint.
- **Disadvantages:** Radiographs generate images through the use of ionizing radiation, and thus, care must be exercised in their use, particularly in young athletes. However, the overall dose is small, particularly when imaging extremities (see Table 63.1). Dose is more substantial when imaging the hip, pelvis, or spine because these structures are deeper in the patient and require a greater radiographic exposure to penetrate surrounding soft tissues and to provide images of diagnostic quality.
- While the spatial resolution is high, soft tissue contrast is limited; thus, radiographs are less useful in assessment of soft tissue injuries. However, in several cases, the exclusion of a bony component to an injury is sufficient to direct appropriate management.
- **Common indications:** When an imaging study is required in the evaluation of an injured athlete, radiographs are almost always the starting point. Even if no fracture or dislocation is present, other features such as a joint effusion may direct the provider to suspect an occult intra-articular injury.
- Fractures are well depicted by radiographs, and at least two orthogonal views are required to depict any displacement or angulation that may be present. When the patient requires additional imaging with another modality such as MRI, the radiographs still play an important role in depicting the bony anatomy and may improve the accuracy of MRI interpretation.
- Certain diagnoses such as an anterior shoulder dislocation may be obvious by clinical examination, but radiographs confirm the diagnosis and are critical for demonstrating significant fractures that may accompany the dislocation (Fig. 63.1A).
- Scaphoid fractures may be radiographically occult, and repeat radiographs in 10–14 days may better show a nondisplaced fracture once bony resorption around the fracture and early callus formation are evident (Fig. 63.1B).
- Chronic injuries associated with sports may also be demonstrated by radiographs such as cortical thickening in a patient with a tibial stress fracture. It is important to recognize that certain features such as a thickened tibial cortex may persist beyond the period of injury and subsequent healing; thus,

correlation between imaging findings and the clinical picture remains critical as always.

## COMPUTED TOMOGRAPHY (CT)

- CT scans also use radiographs but generate cross-sectional images from which multiplane reformatted images can be created. Surface rendered images can also be created, which are often useful in surgical planning.
- **Advantages:** CT scans show anatomy and injury patterns in cross section, which allows greater detail than is possible with planar imaging with radiographs alone. Complex fractures that involve an articular surface or have impacted fragments are often better depicted by CT because the cross-sectional capabilities facilitate a more complete characterization of the injury and guide appropriate management.

### BOX 63.1 INDICATIONS FOR MEDICAL IMAGING IN SPORTS MEDICINE

- Imaging likely to change clinical management
- When diagnosis is established but extent of injury and associated injuries must be defined
- When conservative management has failed
- When atypical or systemic symptoms are present that cast doubt on a common diagnosis
- When additional information is needed for surgical planning

### TABLE 63.1 COMMON EXAMINATIONS AND TYPICAL DOSE LEVELS

| Examination | Average Effective Dose (mSv) |
|---|---|
| Chest (Posteroanterior and Lateral) | 0.05 |
| Extremity Radiograph | 0.005 |
| Abdomen, Hip or Pelvis Radiograph | 0.7 |
| Thoracic or Lumbar Spine Radiographs | 1.25 |
| CT Thoracic or Lumbar Spine | 6 |
| CT Abdomen/Pelvis | 8–14 |
| Bone scan | 6.3 |

- **Disadvantages:** The use of CT is limited by expense and more restricted availability compared with radiography. In addition, the dose of ionizing radiation to the patient may be significantly higher with CT, and thus, judicious use of the modality is important. Imaging of the hip, pelvis, and spine in particular results in exposure of a significant radiation dose and should only be performed when necessary.
- **Common indications:** CT plays a role in sports imaging in the characterization of complex fractures where radiographs alone are insufficient to fully depict the fracture features necessary to guide treatment. The depiction of calcific density by CT may also be useful in confirming certain diagnoses such as myositis ossificans or in the characterization of certain bone tumors and infections.
- A vast majority of fractures in the setting of sports medicine are adequately characterized by radiographs; however, complex fractures of joints such as the knee, ankle, and wrist may require CT (Fig. 63.2). In addition, CT is occasionally useful in the monitoring of fracture healing in the scaphoid and in regions of hardware fixation.
- Surgical planning may require CT to fully characterize a bony fragment, as in the case of a bony Bankart fracture of the glenoid suffered in the setting of an anterior shoulder dislocation. In such a patient with ongoing instability for whom a stabilizing procedure is being considered, a CT scan may depict the degree of glenoid insufficiency and whether the distracted fragment is of sufficient size that reduction by surgical means is possible (Fig. 63.3).

## MAGNETIC RESONANCE IMAGING (MRI)

- MRI generates images by exposing the patient to a powerful magnetic field and using radio waves to excite tissues and extensive computer processing to generate cross-sectional images. The technique provides a high degree of soft tissue contrast and is thus unparalleled in its ability to depict ligament, tendon, and cartilage abnormalities. Numerous diagnoses can be made with MRI that previously required arthroscopic or open surgical inspection.
- **Advantages:** MRI images can be generated in any plane, and numerous sequences have been developed to characterize soft tissues. The technique does not use ionizing radiation, which is also an attractive feature, particularly when working with young athletes. Contrast agents exist and may be useful in evaluating infections and tumors.

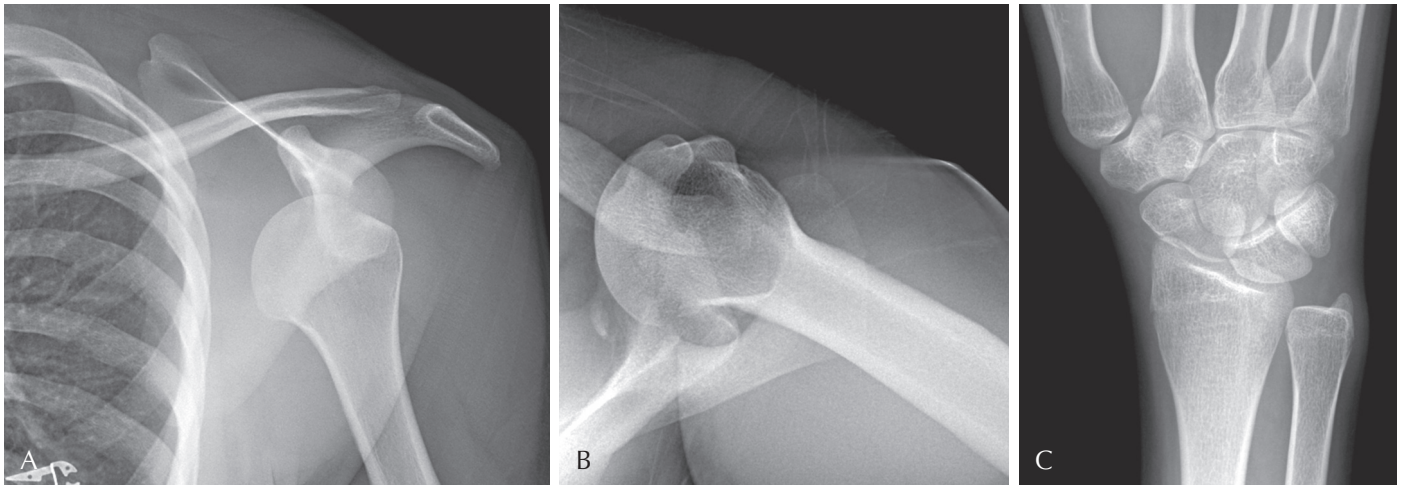


**Figure 63.1.** Anteroposterior **(A)** and axillary **(B)** radiographs of an anterior glenohumeral shoulder dislocation. Note the subtle fracture fragment on the axillary view arising from the anterior rim of the glenoid. An oblique radiograph of the wrist **(C)** shows a very subtle nondisplaced scaphoid waist fracture.

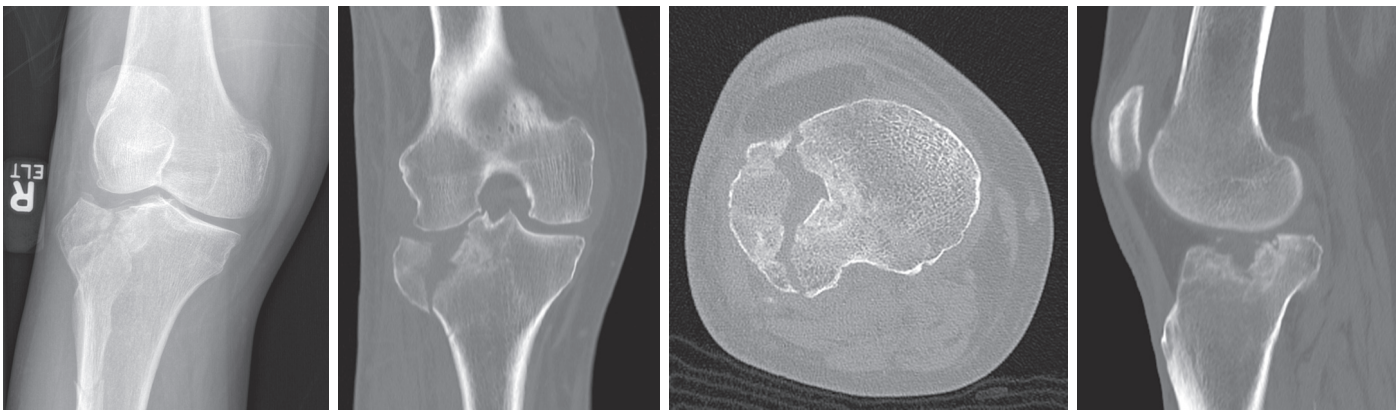

**Figure 63.2.** A knee radiograph shows a lateral tibial plateau fracture. Computed tomography (CT) images in the coronal, axial, and sagittal planes more completely characterize the injury, facilitating appropriate treatment.

**Figure 63.3.** A coronal computed tomography (CT) image and 3D surface rendered reconstruction of bone (with the humerus subtracted) shows the small glenoid rim fracture (bony Bankart lesion) related to a prior anterior shoulder dislocation.

- **Disadvantages:** Disadvantages of MRI include a more limited availability and high expenses. Certain implanted medical devices (such as cochlear implants) are contraindicated in the MRI environment, which can prevent the use of MRI in certain cases. Certain patients are unable to hold still for the required examination, which may be for >30 minutes. The MRI scanner itself has an enclosed patient compartment, and numerous patients find the scanning experience claustrophobic and uncomfortable.
- **Common indications:** The role of MRI in modern sports imaging is central. Injuries of soft tissues such as the cruciate ligaments or meniscus of the knee or labrum of the shoulder are well demonstrated by MRI. Intra-articular injection of contrast agents (a technique called *arthrography*) is occasionally used to diagnose injuries of cartilage or of the labrum at the hip and the shoulder.
- An acutely injured knee with suspected cruciate ligament and meniscus tearing is a common indication for MRI, and the examination can clearly depict internal derangements (Fig. 63.4), which is useful for planning conservative or surgical intervention. Commonly, a diagnosis or anterior cruciate ligament tear may be confirmed from physical examination findings, but an MRI may reveal associated meniscus or cartilage injuries that will alter planned surgical management.
- Large tendons such as the biceps tendon (Fig. 63.5) are also well depicted by MRI, and the degree of tendon retraction can be clearly seen. Soft tissue injuries such as muscle tears are also well demonstrated and easily differentiated from a fracture that might otherwise be clinically suspected.

**Figure 63.4.** Sagittal proton density-weighted image of the knee showing a complete anterior cruciate ligament (ACL) rupture with an associated effusion.

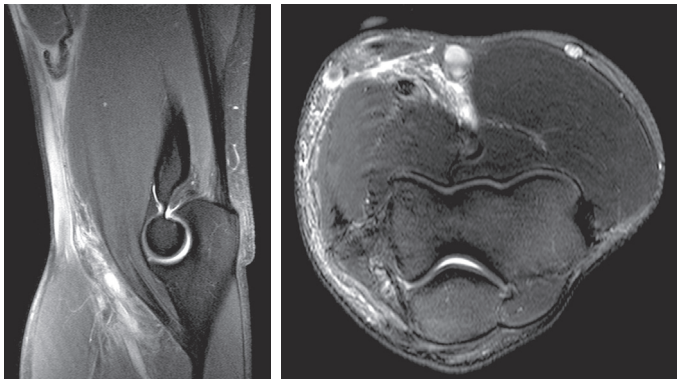

**Figure 63.5.** Sagittal and axial proton density fat-saturated images of the elbow show complete rupture of the biceps tendon with significant retraction. Note the fluid and swelling in the antecubital fossa.

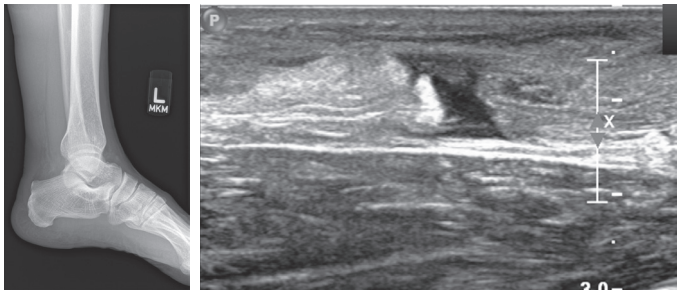

Figure 63.6. A lateral ankle radiograph shows no fracture, but there is soft tissue swelling in the region of the Achilles tendon. A longitudinally oriented ultrasound image in the same region shows a retracted brightly echogenic Achilles tendon with dark anechoic fluid in the tendon gap.

## ULTRASOUND

- Ultrasound is an increasingly used modality in sports medicine. The development of high-frequency linear transducers and greater portability of ultrasound units has led to widespread adoption of musculoskeletal ultrasound imaging.
- **Advantages:** Unlike the imaging modalities previously discussed, ultrasound images are generated in real time, allowing a provider to directly examine the site of injury and discuss the injury with the patient while the study is performed. This unique aspect allows a dynamic assessment of painful maneuvers and comparison with the unaffected ipsilateral structure when necessary. Tendons and ligaments tend to be echogenic and bright on ultrasound images while the fluid is anechoic and dark. Muscle and fat are more intermediate in echogenicity and appear toward to center of the gray scale. However, the appearance of each of these structures is partially dependent on the scanning parameters and the probe used to acquire images.
- **Disadvantages:** While ultrasound depicts superficial structures with high spatial resolution, deeper structures are not well demonstrated. The ultrasound beam cannot penetrate bone and thus only the surface of bone can be seen. Given the small portion of the body shown on any ultrasound image, it is also critical to understand bony landmarks and to review radiographs and other imaging studies, if available, before an ultrasound examination. In addition, appropriate documentation of findings and labeling of images is necessary to maintain continuity of care.
- **Common indications:** Ultrasound is well suited for evaluation of superficial tendons and ligaments. Small portable ultrasound units have seen use in acute settings for evaluating sports injuries and may show the presence of an effusion or torn structure. An acutely ruptured patellar tendon or Achilles tendon (Fig. 63.6) is an example of an injury well demonstrated by ultrasound.
- Ultrasound is also uniquely suited to guide procedures such as a subacromial subdeltoid bursa injection at the shoulder (Fig. 63.7). Depiction of muscle planes and tendons allows the operator to precisely guide a needle into a thin bursa or joint for a selective injection.

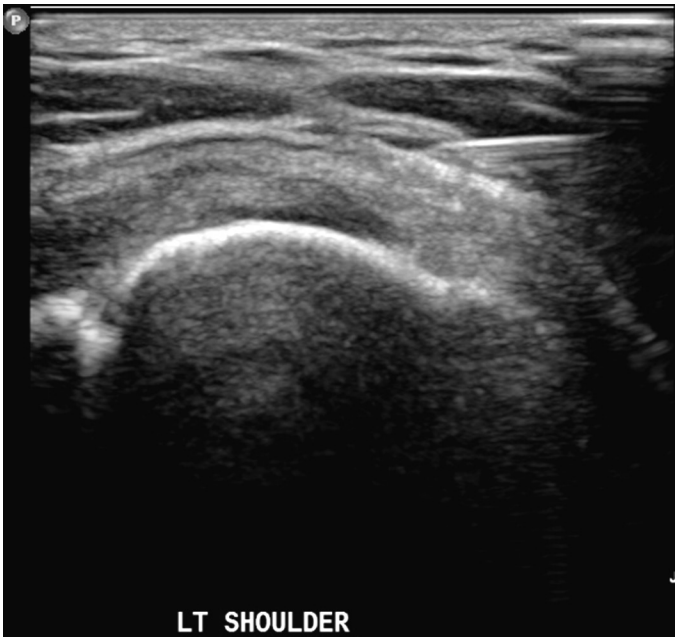


Figure 63.7. An ultrasound-guided subacromial subdeltoid bursal injection is demonstrated with placement of the needle tip (the bright linear structure at the upper right of the image) between the deltoid muscle and rotator cuff. This image is of the shoulder in the coronal plane, and the echogenic bright surface of the superior humeral head is visible at the bottom of the image.

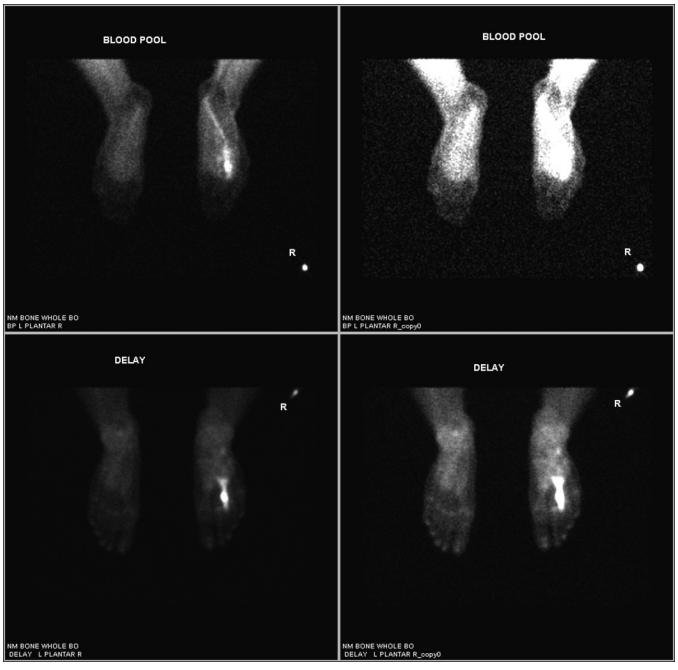


Figure 63.8. A bone scintigraphy scan shows increased activity on blood pool and delayed images in the region of the left third metatarsal, consistent with a stress fracture.

## NUCLEAR MEDICINE

- Nuclear medicine techniques are also used in sports medicine to depict stress fractures and localize infections. MRI has increasingly supplanted the use of nuclear medicine in sports medicine, but knowledge of the modality and potential uses remains important.
- The nuclear medicine technique used most commonly in the evaluation of sports injuries is the bone scan, which involves the intravenous administration of a radiopharmaceutical (typically Technetium-99m tagged to hydroxymethylene diphosphonate or methylene diphosphonate), followed by imaging with a gamma camera. The radiopharmaceutical is taken up in bone and detected by the camera as it decays, emitting gamma rays.

- Imaging the body part in question may begin in seconds, minutes, and hours after the administration of the radiopharmaceutical as a "3-phase" bone scan, which provides information about blood flow, inflammation, and bony abnormalities. A cross-sectional technique (single-photon emission computed tomography [SPECT]) can also be performed.
- **Advantages:** Bone scan examinations are highly sensitive to any injury or abnormality of bone. However, they are relatively nonspecific as an infection, tumor, or fracture all result in uptake of the radiopharmaceutical in regions of bone turnover.
- **Disadvantages:** The lack of specificity limits the utility of bone scan techniques and the dose to the patient can be substantial. For this reason, most diagnoses of stress fractures or suspected infections are currently made using MRI.
- **Common indications:** Bone scans are highly sensitive in the detection of stress fractures (Fig. 63.8) and become positive soon after the onset of injury when no abnormality may be apparent on radiographs.

## SUMMARY

- Imaging of an injured athlete typically begins with radiographs, although several diagnoses may be made without imaging of any kind.
- Imaging findings must be interpreted in the appropriate clinical setting because certain injuries may have persistent findings after symptoms or dysfunction have resolved.
- Each imaging modality has unique features that are well suited to answer certain clinical questions, and an understanding of their utility is crucial to the sports medicine provider.
- Open communication between radiologists and the sports medicine team is critical in determining accurate diagnosis and treatment.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SPORTS ULTRASOUND

# 64

Mederic M. Hall

## GENERAL PRINCIPLES

### Physics

- Electric **voltage from the base unit is converted to sound waves by the reverse piezoelectric effect**. Sound waves are reflected at tissue boundaries, producing echoes. Sound wave echoes returning to the transducer are converted to volts via the piezoelectric effect, and these volts are assigned a gray-scale color and location on the screen to produce an image.
- **A medium** is required for sound waves to pass through (acoustic coupling gel, water, etc.).
- Degree of reflection dependent upon **acoustic impedance**; similar acoustic impedance results in less reflection. Greater difference in acoustic impedance results in more reflection.
- The angle at which a sound wave strikes a tissue interface is referred to as the **angle of incidence**. **Perpendicular incidence** refers to a sound wave traveling perpendicular to the boundary between two media; this maximizes the amount of echoes reflected back to the transducer and optimizes the image. **Oblique incidence** occurs when the sound wave is not traveling perpendicular to the boundary between two media, and some echoes are reflected away from the transducer; this can result in the artifact of **anisotropy** (discussed later).

### Terminology

**Anechoic:** Black (i.e., no reflection)
**Hypoechoic:** Dark, relative to the surrounding structures (i.e., less reflection)
**Isoechoic:** Same shade of gray as another structure
**Hyperechoic:** Bright, relative to the surrounding structures (i.e., high reflection)

### Transducers

- Higher frequency results in better resolution but lower penetration; recommended for imaging superficial structures
- Lower frequency results in lower resolution but improved penetration; recommended for imaging deep structures
- Linear array transducers: Flat transducer surface; sound waves exit perpendicular; minimizes anisotropy but produces a smaller field of view; recommended for superficial structures
- Curvilinear array transducers: Transducer surface curved with sound waves exiting in fan shape; increases the risk of anisotropy, but produces a larger field of view; recommended for imaging deep structures and guiding procedures at steep angles

### Artifacts

#### Anisotropy (Fig. 64.1)

- Sound waves reflected away from the transducer, result in an artifactual hypoechoic/anechoic appearance; risk of false-positive interpretation
- Results from oblique angle of incidence
- Tendons are most susceptible, followed by ligaments and then nerves
- Most common artifact in musculoskeletal imaging

#### Posterior Acoustic Shadowing

- Hypoechoic/anechoic region deep to the area of high reflectivity or high attenuation
- Commonly deep to a bone or calcium (may also be seen deep to scar tissue or normal fibrous septae)

#### Edge Shadowing

- Refraction artifact secondary to velocity change deep to a curved interface
- Helpful in identifying torn tendon edges
- Can obscure imaging deep to edges of both normal and abnormal structures

#### Acoustic Enhancement

- Also referred to as increased **through transmission**
- Hyperechoic region deep to an area of low attenuation
- Commonly deep to fluid collections

#### Reverberation

- Multiple internal reflections from highly reflective surface
- Commonly seen with metal from needles or orthopedic hardware

## DIAGNOSTIC SPORTS ULTRASOUND

### Musculoskeletal

- Provides detailed evaluation of musculoskeletal structures and offers several advantages compared with other imaging modalities, including high-resolution imaging (submillimeter), ability to image in real time and interact with a patient during the examination, contralateral comparison examination, minimal metal artifacts, portable, relatively inexpensive, and no radiation or known contraindications
- Inherent limitations include limited field of view, limited penetration, incomplete evaluation of bones and joints, variability in equipment, and operator dependence.
- Diagnostic musculoskeletal ultrasound examinations are either a complete assessment of a joint or anatomic region (e.g., anterior knee) or a focused, limited assessment of a specific structure (e.g., Achilles tendon). Complete examinations should include all relevant structures within the anatomic region, including muscles, tendons, joints, other soft tissue structures, and any identifiable abnormality. Although most joint regions are divided into quadrants, a complete examination of the shoulder should include all relevant structures.

#### Tendon

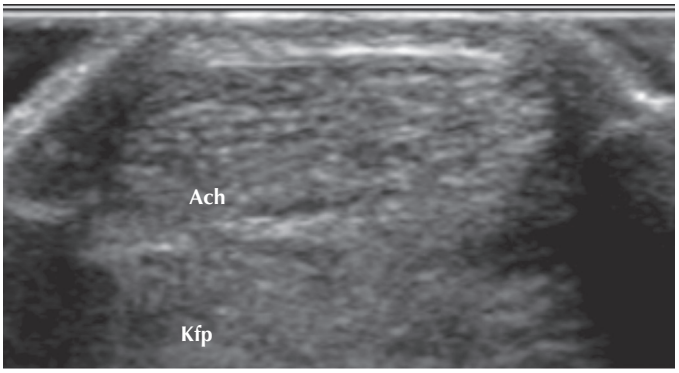
##### NORMAL SONOGRAPHIC APPEARANCE

- High-frequency linear array transducer recommended
- Exceptions are in certain deep tendons in patients with large body habitus (iliopsoas, gluteals, etc.)
- Long axis (LAX): Fibrillar pattern of hyperechoic tightly packed linear echoes
- Short axis (SAX): Stippled clusters dots resembling a "broom end"

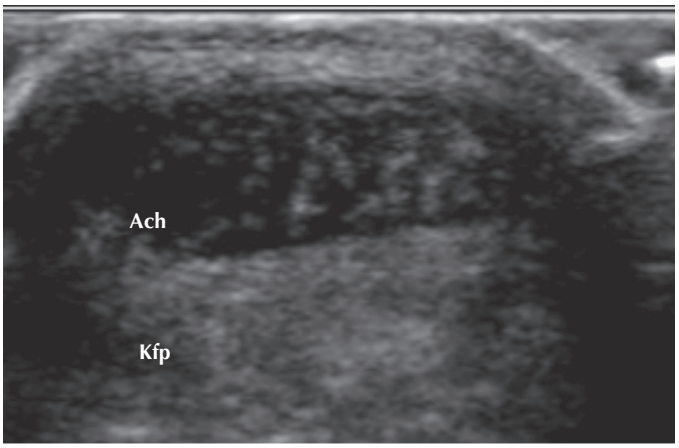
##### GENERAL PATHOLOGIC FEATURES

Pathologic findings are similar across tendons. Location of injury within specific tendons will vary and often affected by zones of relative hypovascularity, biomechanical factors, and repetitive trauma.

**Tendinosis:** Features include thickening/swelling of the tendon, hypoechogenicity, and heterogeneity with loss of usual fibrillar




**A.** Sound waves exiting transducer are perpendicular to the short axis of the Achilles tendon resulting in optimal image represented as hyperechoic uniform echotexture.




**B.** Slight angulation of the transducer with oblique angle of incidence resulting in artifactual hypoechogenicity of the Achilles tendon.

**Figure 64.1.** Anisotropy. *Ach*, Achilles tendon; *Kfp*, Kaeger's fat pad.

echotexture. In chronic cases, the tendon may appear atrophic instead of thickened. Intratendinous calcifications may be identified; power or color Doppler imaging may demonstrate intra/peritendinous hyperemia, termed neovascularity.

**Tendon tear:** Classified as partial, full thickness, and complete
- **Partial thickness tear:** Well-defined focal defect in tendon confirmed in two planes; often seen as progression of tendinosis
- **Full thickness tear:** Tear extends across full thickness of the tendon (deep to superficial) but may not involve full width of tendon
- **Complete tear:** Full thickness and full width of tendon; retraction of torn tendon fibers; this will appear as a gap within the tendon or complete nonvisualization of the tendon

**Calcific tendinopathy:** Calcium hydroxyapatite deposition within tendon; appearance is hyperechoic with variable amounts of posterior acoustic shadowing dependent upon type/phase of calcification; symptoms typically related to mass effect/impingement or inflammatory response in resorptive phase; may be asymptomatic

**Tenosynovitis:** Inflammation of tenosynovial lining of tendon sheath represented as complex fluid and hyperemic tissue adjacent to tendon within sheath; pain with transducer pressure helpful in distinguishing symptomatic tenosynovitis from other secondary causes of tendon sheath fluid (e.g., communicating joint fluid)

PEARLS AND PITFALLS
- Anisotropy can mimic tendinosis or tear and is common, particularly in the rotator cuff; confirmation in two planes and careful scanning technique is critical
- Edge shadowing artifact can be useful in identifying torn tendon ends.
- Dynamic imaging is helpful in identifying complete tears.
- Clinical significance of neovascularity is controversial.

SPECIFIC PATHOLOGIES
- Rotator cuff tendinopathy and tears (Fig. 64.2)
- Proximal biceps tendinopathy (Fig. 64.3)
- Common extensor tendinopathy (Fig. 64.4)
- DeQuervain's tenosynovitis (Fig. 64.5)
- Proximal hamstring tendinopathy (Fig. 64.6)
- Patellar tendinopathy (Fig. 64.7)
- Fibular (peroneal) tendinopathy (Fig. 64.8)
- Achilles tendinopathy and tears (Fig. 64.9)
- Plantar fasciopathy (Fig. 64.10)

## Muscle

NORMAL SONOGRAPHIC APPEARANCE
- Hypoechoic muscle fibers/fascicles and hyperechoic septa of peri/epimysium form a pennate pattern on LAX and "starry night" pattern on SAX.

GENERAL PATHOLOGIC FEATURES

**Strain:** Focal disruption of normal fiber pattern/structure; in an acute injury, there will be swelling of muscle with possible focal defect in higher grade injuries. If fibers are torn, hemorrhage may be identified as hypo/anechoic compressible fluid collection at the site of injury; will begin to organize into mixed echogenic hematoma and eventually form hyperechoic scar tissue. Hyperemia on power and color Doppler imaging is common in acute injury.
- Hamstring muscle strain (Fig. 64.11)

**Contusion:** Typically involves deep muscle fibers adjacent to bone (e.g., vastus intermedius in the thigh); myositis ossificans will appear as a hyperechoic linear calcific density within the zone of muscle injury; demonstrated earlier on ultrasound than radiograph
- Quadriceps contusion and myositis ossificans (Fig. 64.12)

## Ligament

NORMAL SONOGRAPHIC APPEARANCE
- Identify bony landmarks and align transducer with ligament; LAX most useful imaging plane
- Fibrillar echotexture pattern is less compact than tendon
- Typically hyperechoic, but depending upon surrounding tissue, may appear hypoechoic

GENERAL PATHOLOGIC FEATURES

**Grade I:** Swollen and hypoechoic without fiber disruption or laxity on dynamic stress imaging

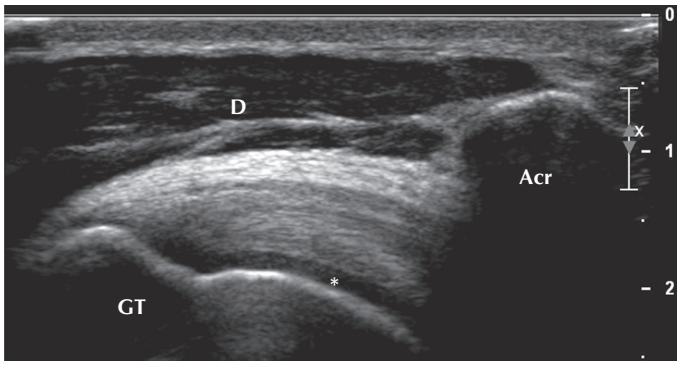
**Grade II:** Partial tear/evidence of fiber disruption; may have some laxity on dynamic stress imaging, but end-point appreciated

**Grade III:** Complete tear; dynamic stress imaging demonstrates laxity and absence of bridging fibers.
- High ankle sprain (Fig. 64.13)

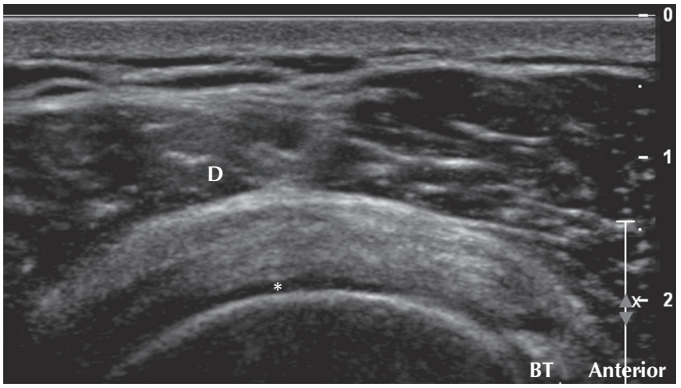
## Nerve

NORMAL SONOGRAPHIC APPEARANCE
- Hypoechoic nerve fascicles and hyperechoic intra/extra neural epineurium form a "honeycomb" appearance on SAX; fascicular appearance on LAX (less tightly packed than ligament); typically adjacent to vessels, which may be easier to identify

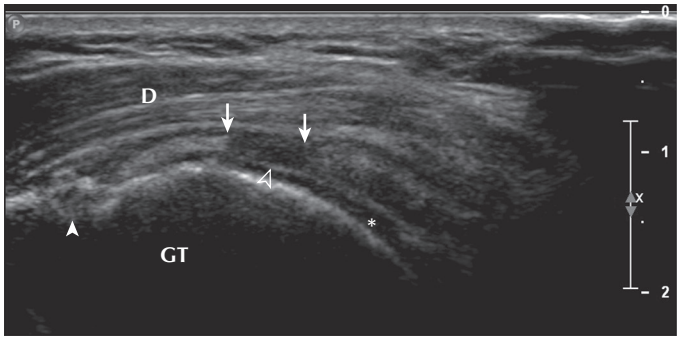
*Text continued on p. 522*



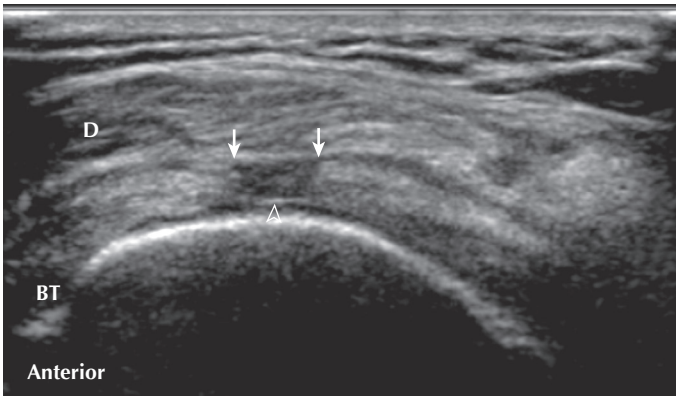

**A.** Long axis view of normal supraspinatus tendon. Often referred to as "bird's beak" appearance. Note homogenous echotexture and smooth cortical margin.



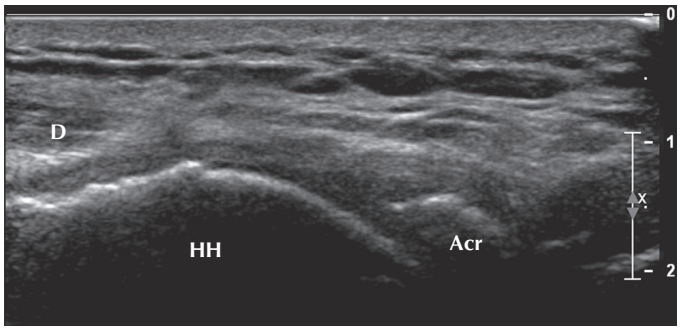

**B.** Short axis view of normal supraspinatus tendon. Note uniform thickness of tendon overlying anechoic rim of cartilage *(asterisks)*. Often referred to as "tire on a wheel." Identification of the long head of the biceps tendon (BT) is key to ensuring adequate evaluation of the leading anterior edge of the supraspinatus.




**C.** Full thickness tear *(arrows)* of supraspinatus tendon in long axis. Cortical irregularities *(solid arrowhead)* at the greater tuberosity foot print and cartilage interface sign *(open arrowhead)*, both secondary signs of rotator cuff tear, are present.

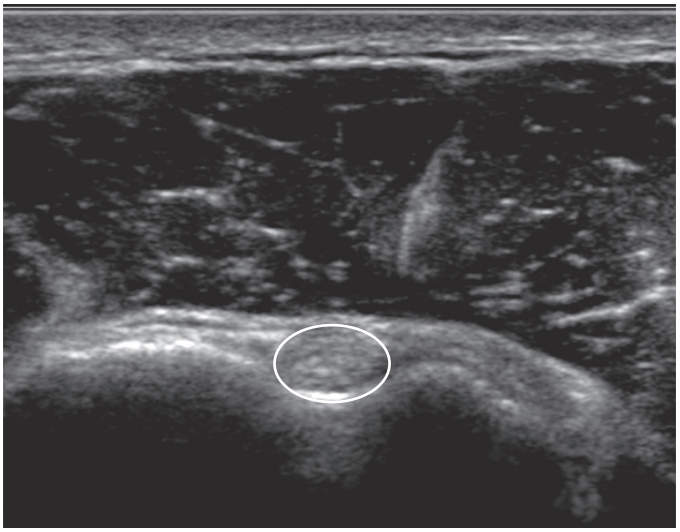



**D.** Corresponding short axis view of full thickness supraspinatus tear. Note volume loss at site of tear *(arrows)* and cartilage interface sign *(open arrowhead)*.

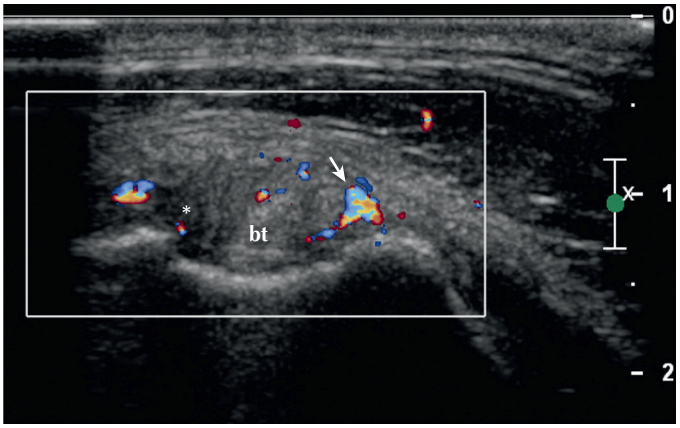



**E.** Chronic complete supraspinatus tear in long axis. There is non-visualization of the rotator cuff with the humeral head (HH) high riding in relation to the acromion (Acr).

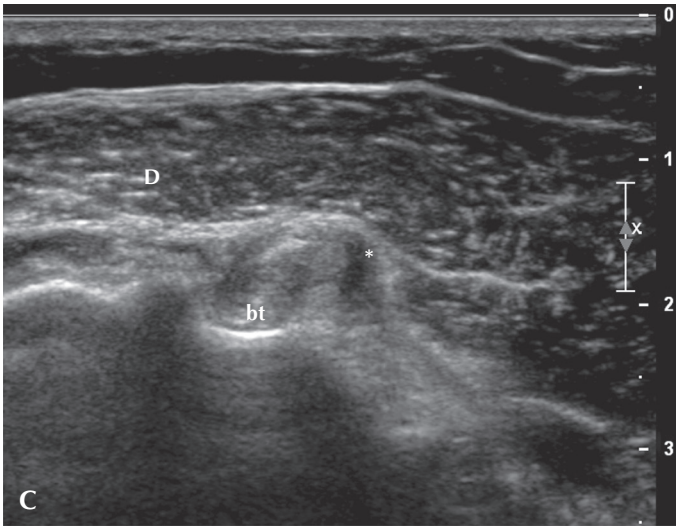
**Figure 64.2.** Rotator cuff tendinopathy and tears, *asterisk* indicates articular cartilage. *Acr*, Acromion; *BT*, long head biceps tendon; *D*, deltoid; *GT*, greater tuberosity; *HH*, humeral head.

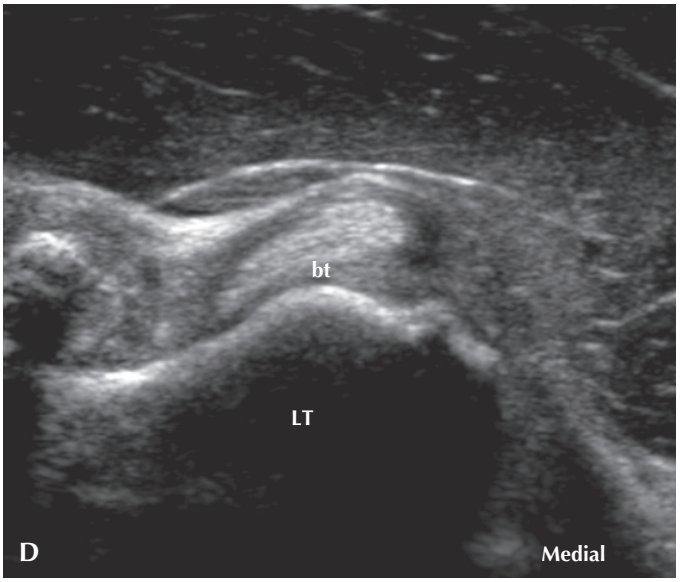


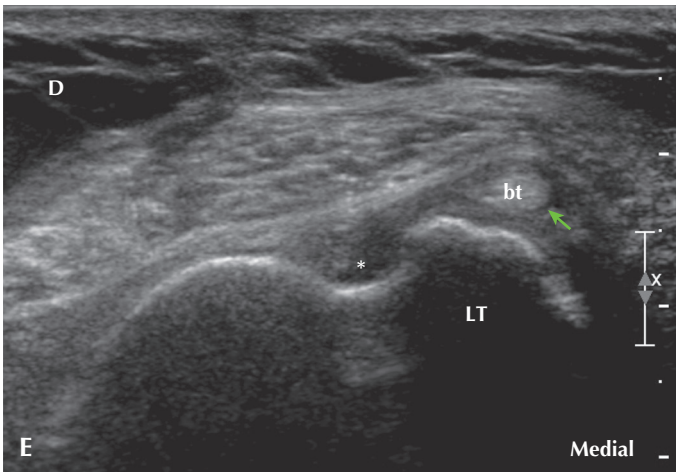
**A.** Short axis view of normal long head of the biceps tendon *(open oval)* at intertubercular groove.




**B.** Biceps tenosynovitis is identified as hyperemic tissue *(arrow)* and complex fluid *(asterisks)* within tendon sheath on color Doppler evaluation.

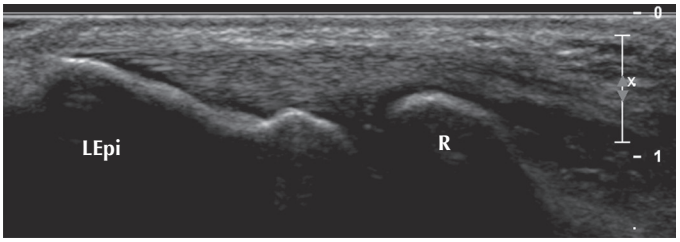


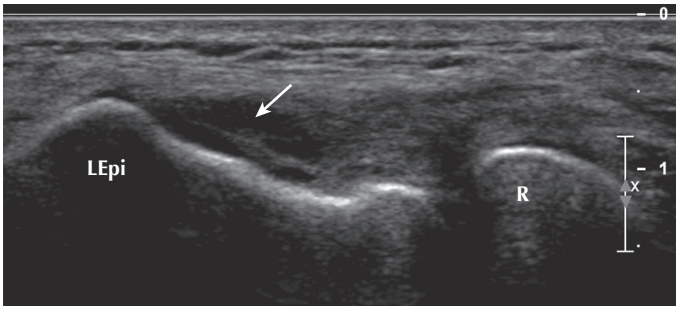




**C–E.** The biceps tendon (bt) is enlarged and heterogenous with hypoechoic fissurations representing tendinosis. Instability of the biceps tendon can be seen as subluxation onto the lesser tuberosity (LT) as seen in **D**, or frank dislocation medially as seen in **E** with empty groove *(asterisks)*.

**Figure 64.3.** Long head of the biceps tendinopathy. *Bt*, Long head biceps tendon; *D*, deltoid; *LT*, lesser tuberosity.

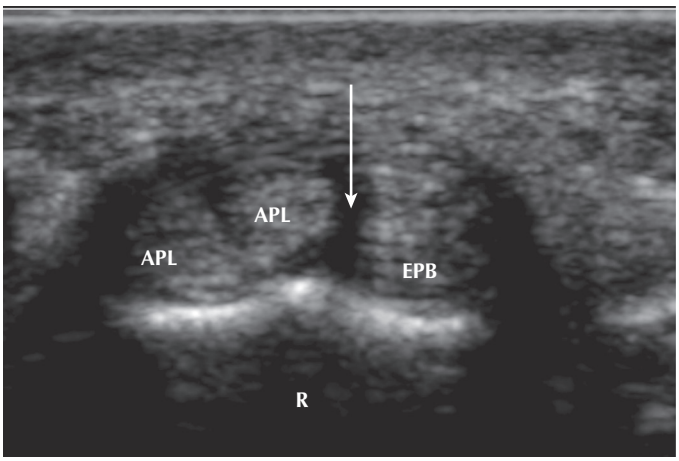



**A.** Long axis view of normal common extensor tendon.

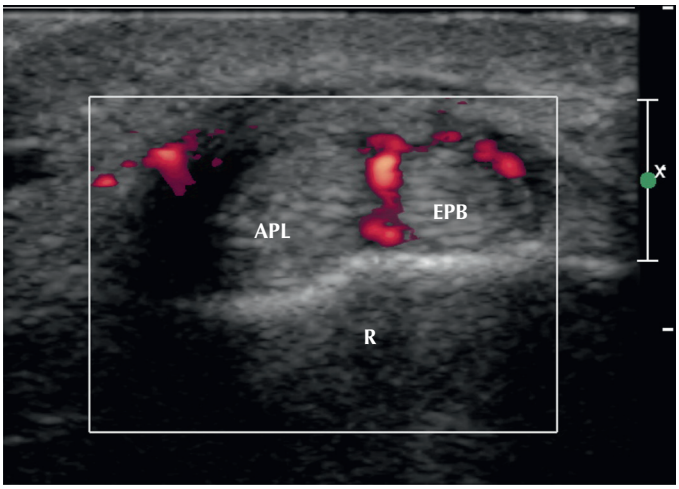



**B.** Severe tendinosis of the common extensor tendon origin in long axis. Note the complete loss of fibrillar echotexture and hypoechogenicity *(arrow)*.

**Figure 64.4.** Common extensor tendinopathy. *LEpi*, Lateral epicondyle; *R*, radius.

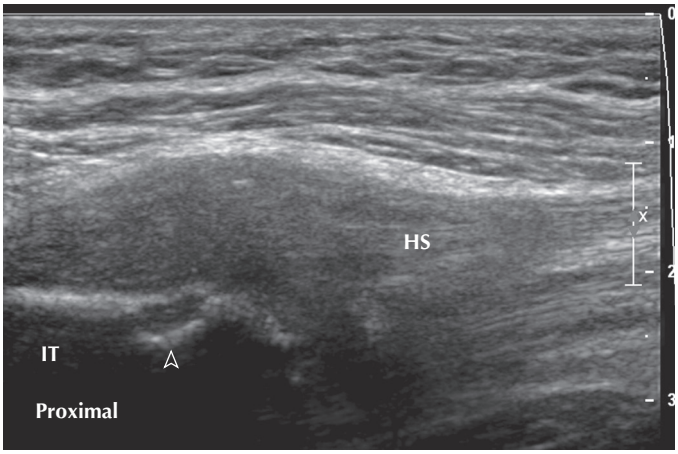



**A.** Short axis view of normal first dorsal compartment tendons. Note the multiple slips of the abductor pollicis longus (APL) which is a normal variant as well as the hypoechoic vertical septum *(arrow)*, which sub-compartmentalizes the sheath.

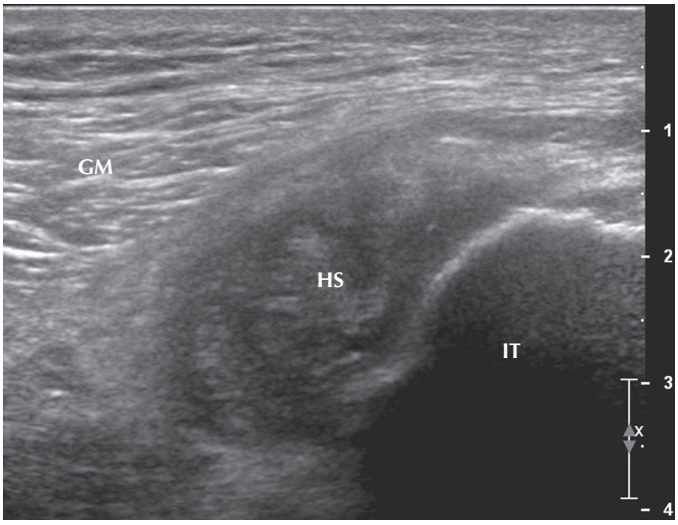



**B.** Power Doppler image of DeQuervain's tenosynovitis demonstrating hyperemia within the tendon sheath.

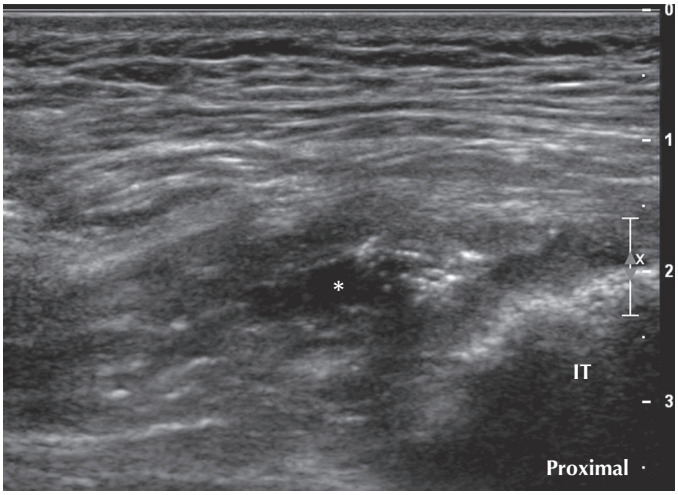
**Figure 64.5.** DeQuervain's tenosynovitis. *APL*, Abductor pollicis longus; *EPB*, extensor pollicis brevis; *R*, radius.




**A.** Long axis view of proximal hamstring tendon demonstrating changes of tendinosis including thickening, hypoechogenicity, and cortical irregularities *(open arrowhead)* at the ischial tuberosity.

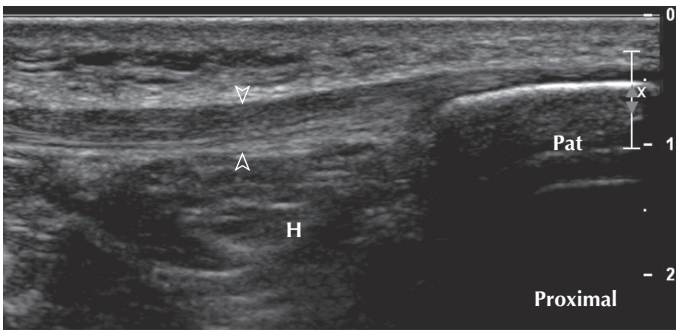



**B.** Corresponding short axis image of tendinosis.

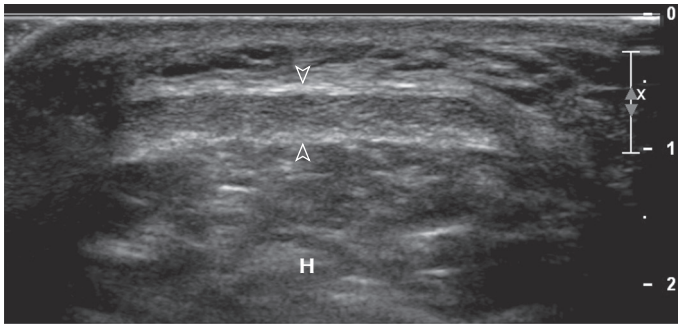



**C.** Severe tendinopathic changes are noted with focal partial thickness tear represented as anechoic region of fiber disruption *(asterisks)*.

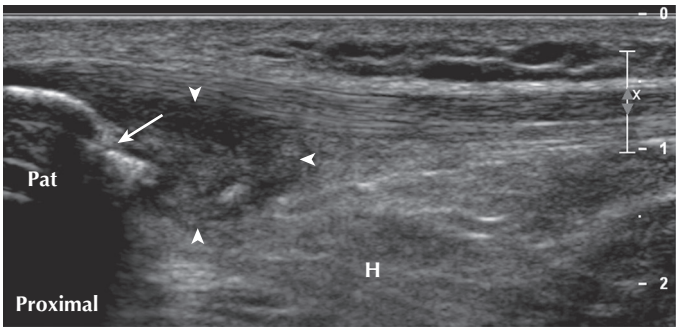
**Figure 64.6.** Proximal hamstring tendinopathy. *GM*, Gluteus maximus; *HS*, proximal hamstring tendon; *IT*, ischial tuberosity.



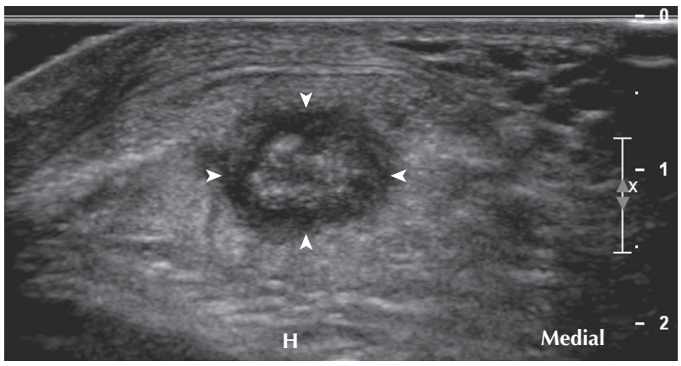

**A.** Long axis view of normal patellar tendon *(open arrowheads)*.



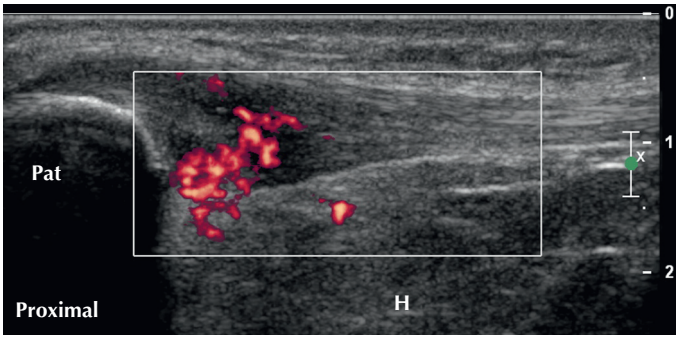

**B.** Short axis view of normal patellar tendon *(open arrowheads)*.




**C.** Severe tendinosis of the proximal patellar tendon *(arrowheads)* demonstrated in long axis. Cortical irregularity is present at the patella *(arrow)*. Note the superficial fibers are spared.

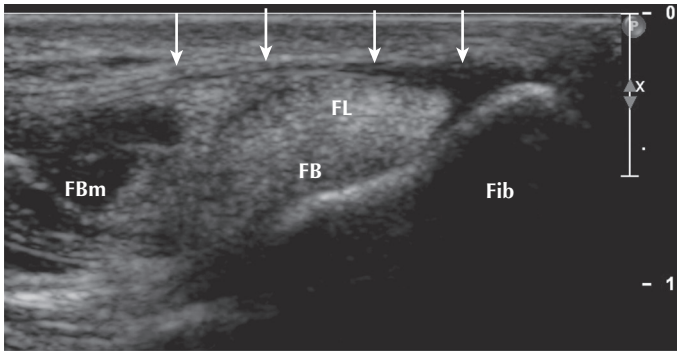



**D.** Corresponding short axis view of severe tendinosis *(arrowheads)*. Note the nodular involvement of the deep central aspect of the tendon. This is the classic pattern of involvement in clinical "Jumper's Knee."

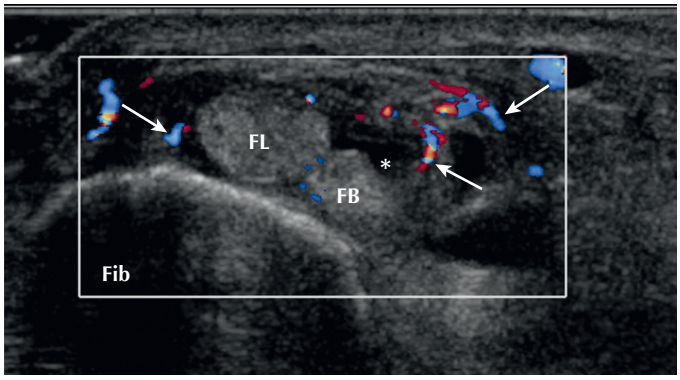



**E.** Power Doppler image demonstrating neovessels invading the patellar tendon from the posterior Hoffa's fat pad.

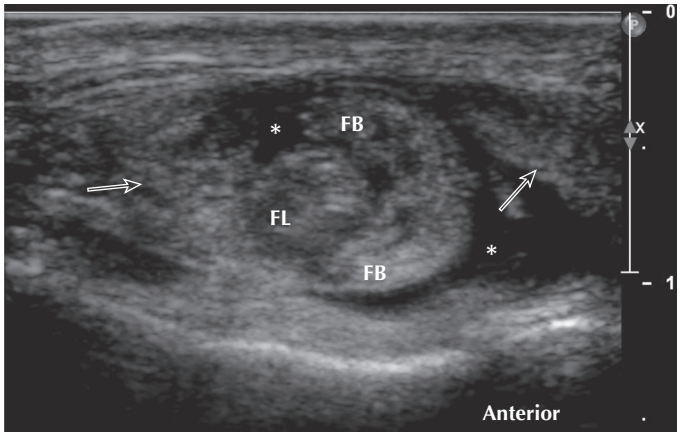
**Figure 64.7.** Patellar tendinopathy. *H*, Hoffa's fat pad; *Pat*, patella.




**A.** Short axis image of normal fibular tendons at the retro-malleolar groove. Arrows identify the superior fibular retinaculum.

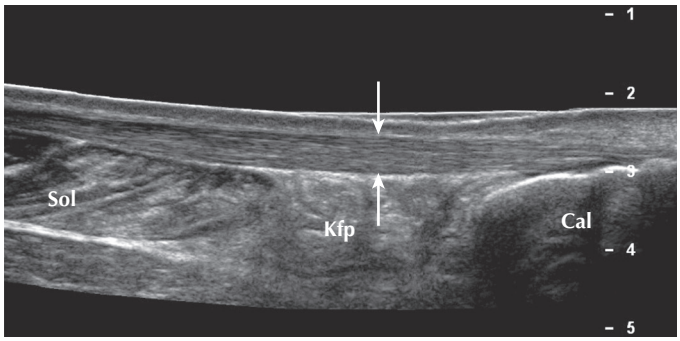



**B.** Fibular tenosynovitis is identified as hyperemic tissue *(arrows)* on color Doppler within the tendon sheath. Note small amount of anechoic free fluid adjacent to tendons *(asterisks)*.

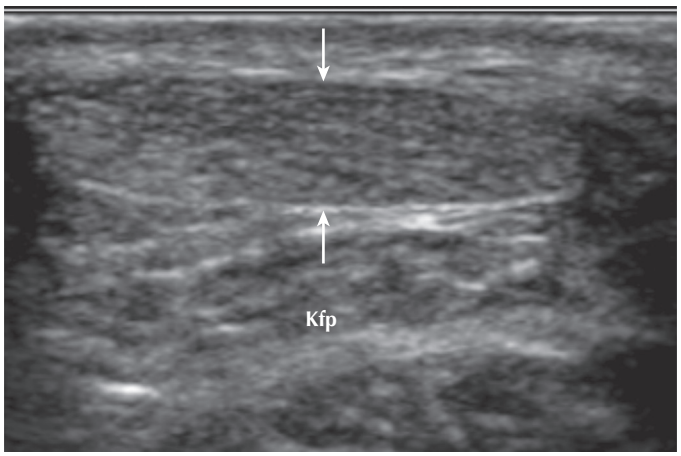



**C.** Short axis image of fibularis brevis longitudinal split tear in the infra-malleolar region. The fibularis brevis has taken a "boomerang" shape as the fibularis longus has invaginated into the tendon. Note the hypoechoic regions of tendinosis within the fibularis brevis. Free fluid *(asterisks)* and thickened tenosynovial tissue *(open arrows)* are seen within the tendon sheath.

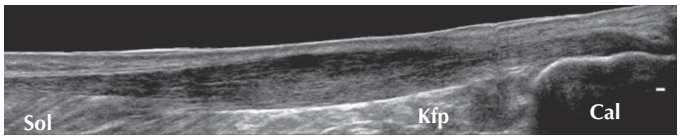
**Figure 64.8.** Fibular (peroneal) tendinopathy. *FB*, Fibularis brevis; *FBm*, fibularis brevis muscle. *Fib*, fibula; *FL*, fibularis longus.



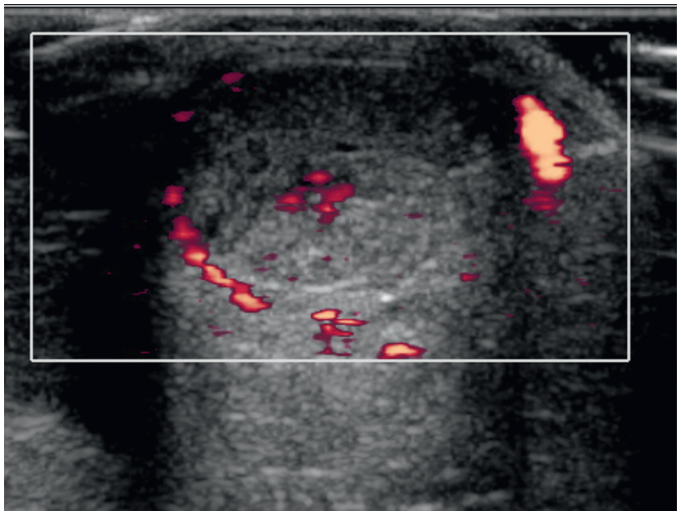

**A.** Long axis extended field of view image of normal Achilles tendon *(arrows)*.



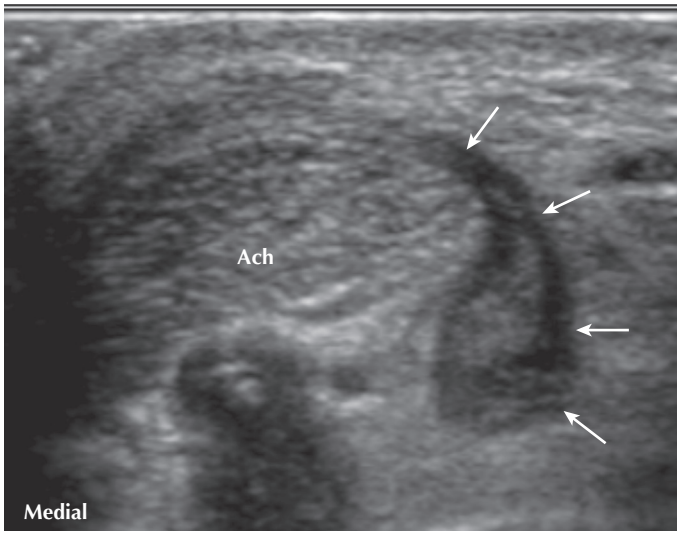

**B.** Short axis image of normal Achilles tendon *(arrows)*.



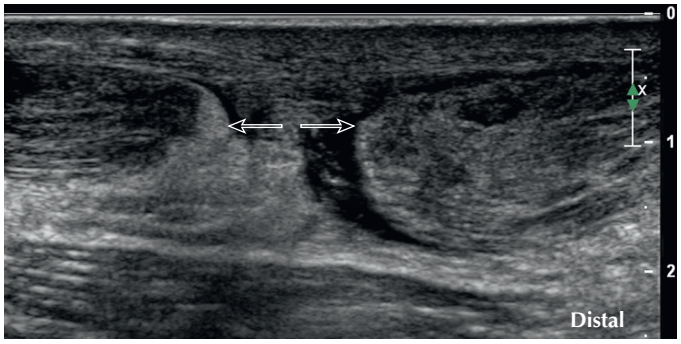

**C.** Long axis extended field of view image demonstrating tendinosis of the midsubstance of the Achilles tendon.



**D.** Power Doppler image of the Achilles tendon in short axis with intra- and peri-tendinous hyperemia referred to as *neovascularity*.

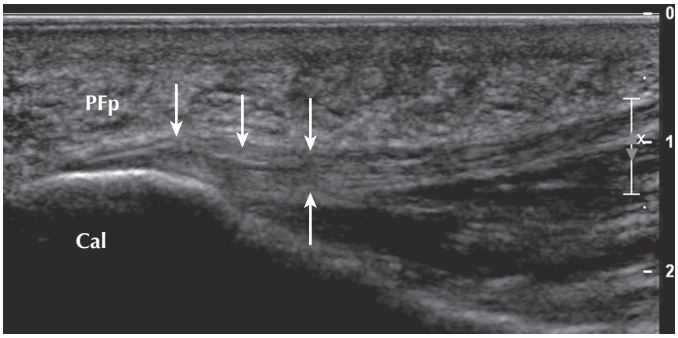



**E.** Complex fluid collection *(arrows)* along the lateral border of the Achilles tendon representing acute paratenonitis. Note the normal appearance of the Achilles tendon in short axis.

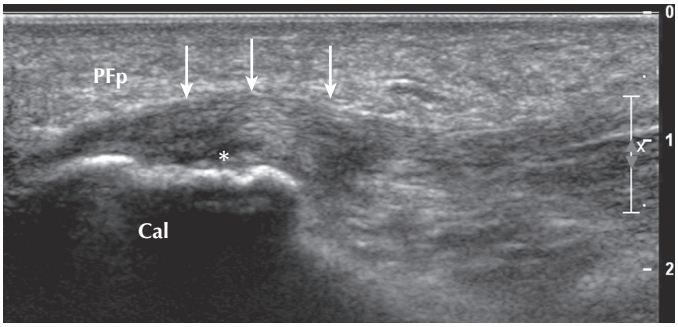



**F.** Acute rupture of the Achilles tendon in long axis. *Open arrows* identify the torn tendon edges.

**Figure 64.9.** Achilles tendinopathy *(asterisk)* indicates retrocalcaneal bursa. *Cal*, Calcaneus; *Kfp*, Kaeger's fat pad; *Sol*, soleus.

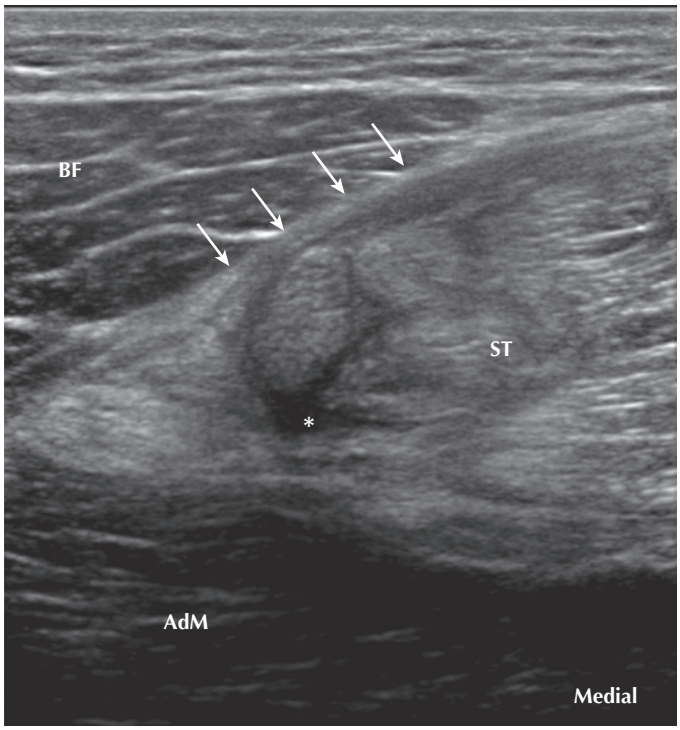



**A.** Long axis image of normal plantar fascia origin *(arrows)*.

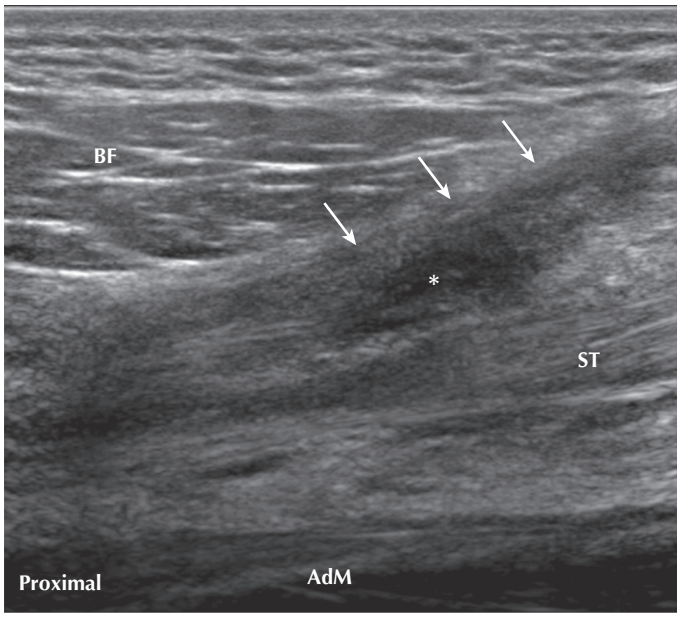



**B.** The plantar fascia origin *(arrows)* is thickened and hypoechoic consistent with degenerative fasciosis. A focal anechoic region devoid of fibers *(asterisks)* is consistent with a partial thickness tear.

**Figure 64.10.** Plantar fasciopathy. *Cal*, Calcaneus; *PFp*, plantar fat pad.

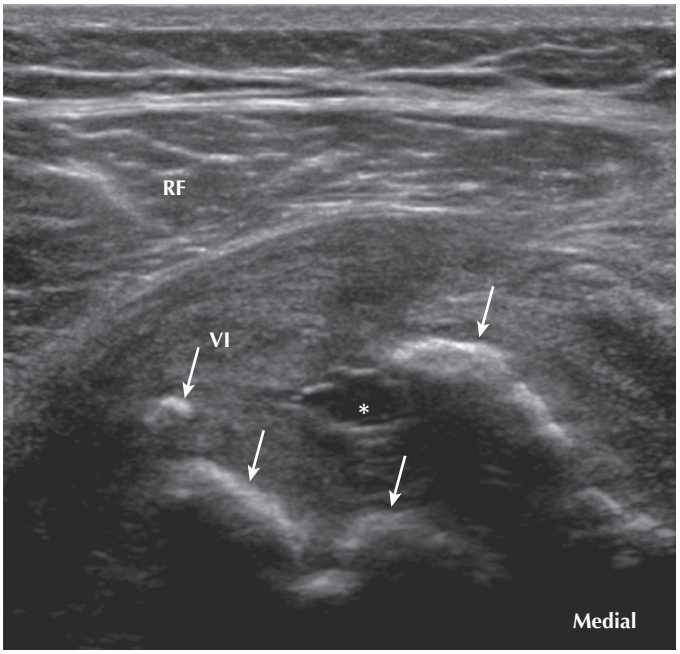



**A.** Short axis image of an acute partial muscle tear of the semitendinosus at the aponeurosis with the biceps femoris *(arrows)*. Note the anechoic free fluid *(asterisks)* at site of tear and disruption of normal echotexture at site of injury. The entire semitendinosus muscle is hyperechoic in relation to the surrounding biceps femoris and adductor magnus representing muscular edema.

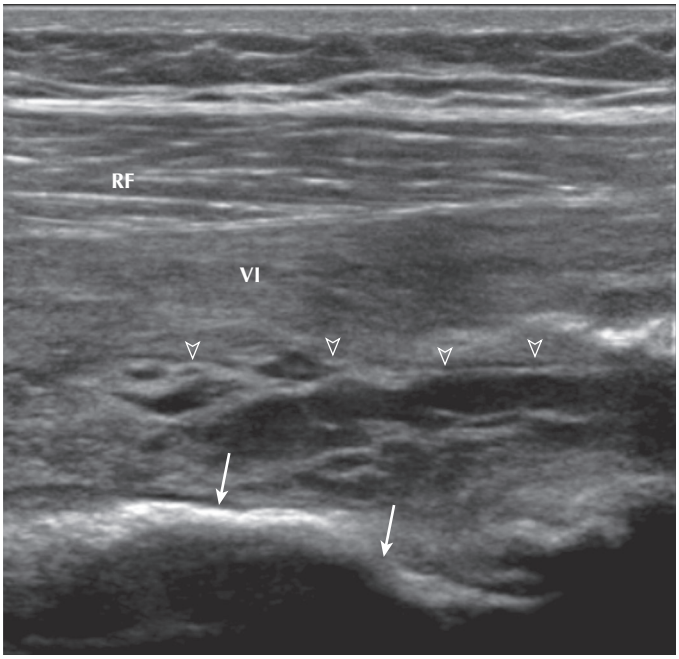



**B.** Corresponding long axis view of the same injury.

**Figure 64.11.** Hamstring strain. *AdM*, Adductor magnus; *BF*, biceps femoris; *ST*, semitendinosus.

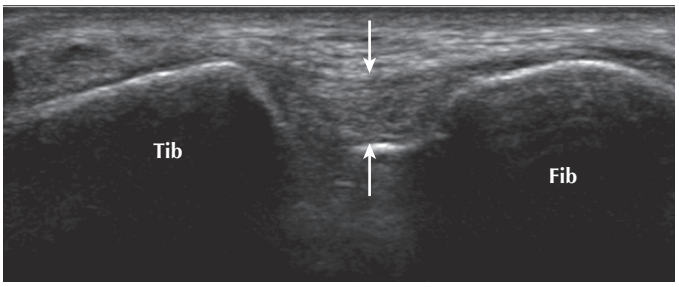



**A.** Short axis image of subacute quadriceps contusion with early myositis ossificans formation. The vastus intermedius is significantly swollen in comparison to overlying rectus femoris, which is normal in appearance. A central region of anechoic free fluid *(asterisks)* is noted with disruption of muscle fibers. Hyperechoic calcific tissue *(arrows)* representing myositis ossificans is seen throughout the vastus intermedius. Note posterior acoustic shadowing deep to the regions of calcification.

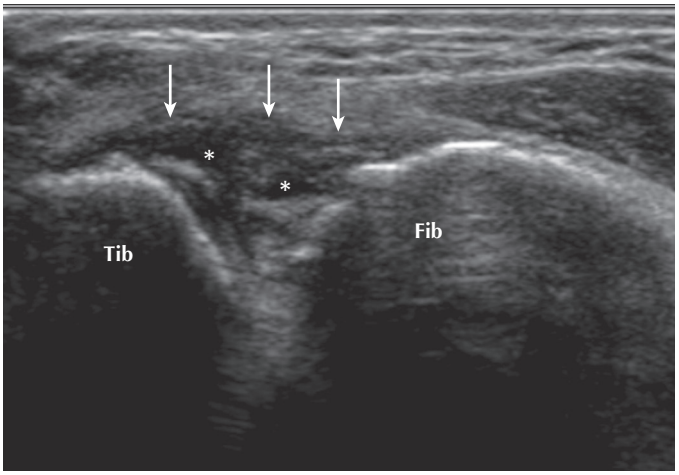



**B.** Corresponding long axis image of the same injury. The extent of the muscle fiber disruption is more apparent *(arrowheads)*. A large calcific lesion *(arrows)* shadows the underlying femur.

**Figure 64.12.** Quadriceps contusion. *RF*, Rectus femoris; *VI*, vastus intermedius.

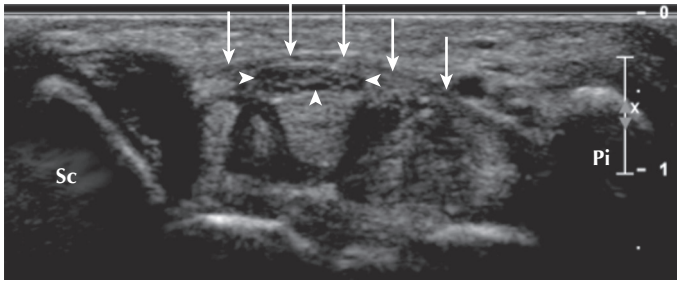



**A.** Normal image of the AITFL *(arrows)*

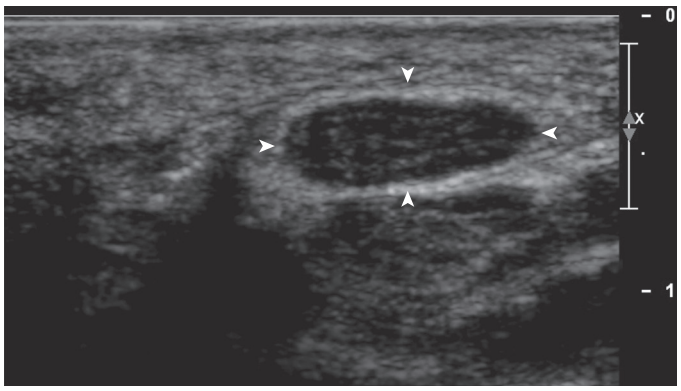



**B.** High-grade sprain of the AITFL *(arrows)* in collegiate football running back. Note disruption of the fibers, swelling, and anechoic regions of acute fluid *(asterisks)*. Dynamic stress imaging demonstrated laxity with external rotation stress.

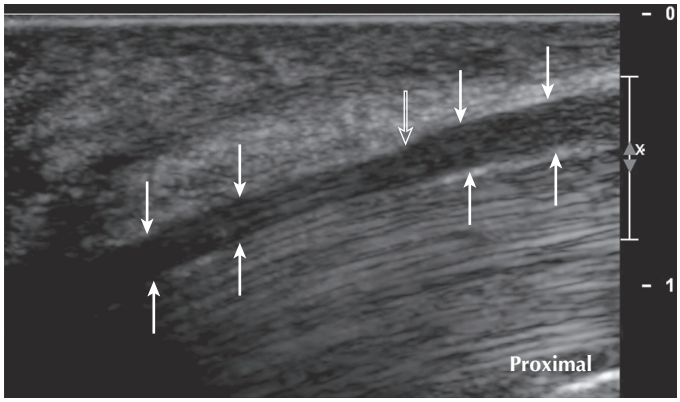
**Figure 64.13.** Anterior inferior tibiofibular ligament (AITFL) tear. *Fib*, Fibula; *Tib*, tibia.




**A.** Short axis image of normal median nerve *(arrowheads)* under the transverse carpal ligament *(arrows)* at the carpal tunnel inlet.




**B.** Short axis image of enlarged and hypoechoic median nerve *(arrowheads)* at carpal tunnel inlet in patient with carpal tunnel syndrome. Note fascicular loss.




**C.** Long axis image of median nerve *(arrows)* at carpal tunnel inlet demonstrating "notch sign" *(open arrow)* where the nerve is compressed at the transverse carpal ligament.

**Figure 64.14.** Carpal tunnel syndrome. *Pi*, Pisiform; *Sc*, scaphoid.

GENERAL PATHOLOGIC FEATURES

- Swelling and hypoechogenicity often accompanied by loss of normal fascicular pattern
- May demonstrate focal swelling proximal to entrapment site or focal narrowing ("notch sign") at the entrapment site
- Dynamic testing may demonstrate subluxation/dislocation or hypomobility and adhesion to surrounding structures.

SPECIFIC PATHOLOGIES

- Carpal tunnel syndrome (Fig. 64.14)
- Ulnar neuropathy and instability at the elbow (Fig. 64.15)

## Bone

NORMAL SONOGRAPHIC APPEARANCE

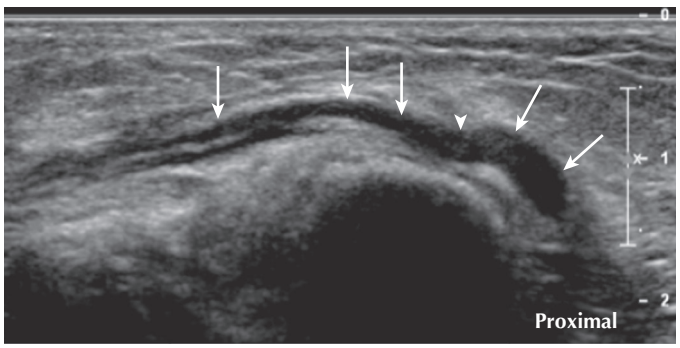
- Evaluation limited to superficial portion due to inability of sound waves to pass through bone
- Surface of the bone should appear hyperechoic, smooth, and linear.

GENERAL PATHOLOGIC FEATURES

**Fracture:** Disruption of smooth cortical surface; hypoechoic hematoma at the fracture site; hyperemia common; pain with transducer pressure

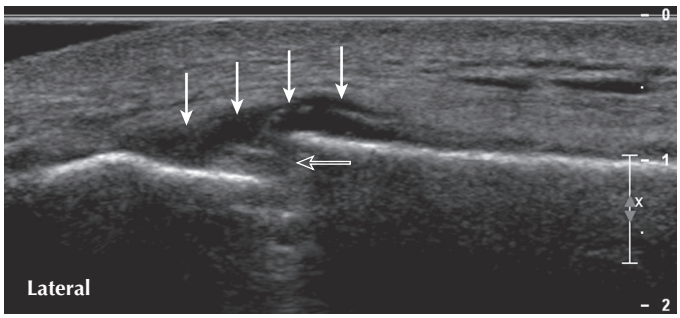
- Ultrasound appearance of fractures (Fig. 64.16)

**Stress fracture/reaction:** Periosteal thickening with (fracture) or without (reaction) cortical discontinuity

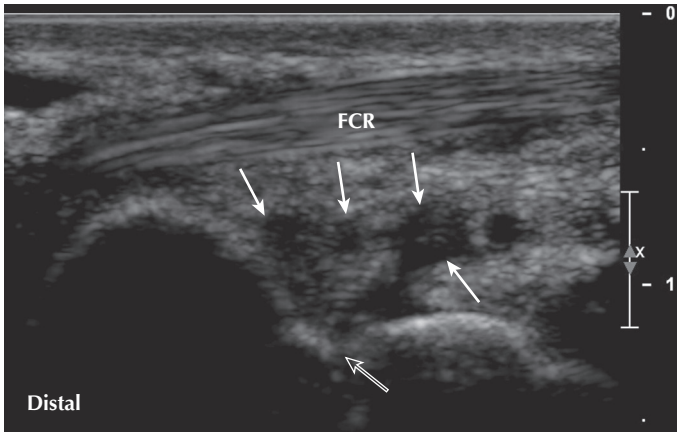



Long axis image of the ulnar nerve *(arrows)* at the elbow status post ulnar transposition performed at time of ulnar collateral ligament reconstruction in a collegiate baseball pitcher. Patient presented with complaints of ulnar distribution paresthesia and grip weakness during his postoperative rehabilitation. Kinking of the nerve *(arrowhead)* was demonstrated distal to transposition site with proximal swelling consistent with ongoing compression.

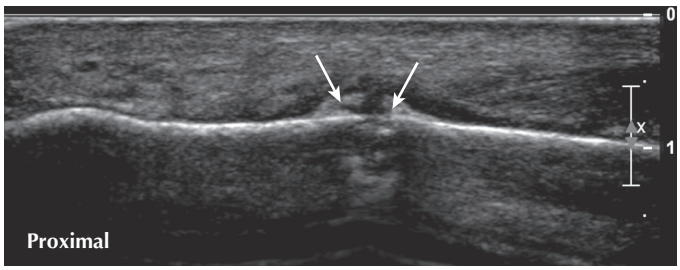
**Figure 64.15.** Ulnar neuropathy at the elbow.




**A.** Acute acromial fracture after mountain bike crash. Note the cortical step-off *(open arrow)* and the peri-fracture hematoma *(arrows)*.

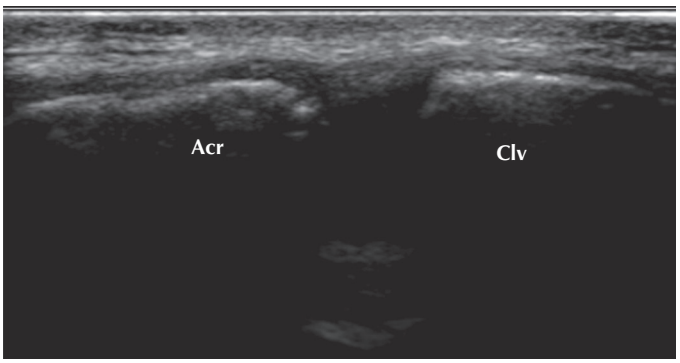



**B.** Radiographically occult acute scaphoid fracture *(open arrow)* demonstrated on volar wrist sonogram. Peri-fracture hematoma *(arrows)* is a helpful secondary sign of fracture.

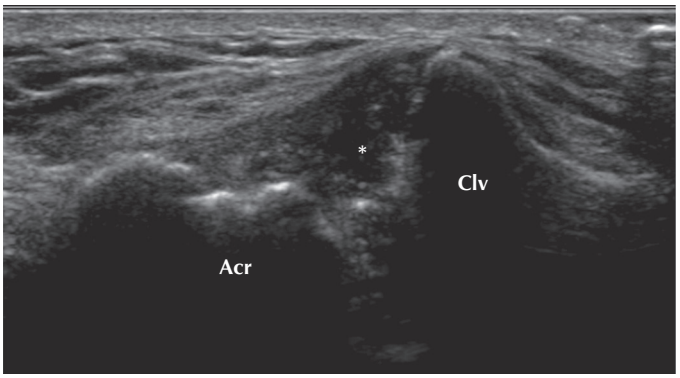



**C.** Long axis image of subacute 5th metatarsal fracture with early callus formation *(arrows)* at fracture site.

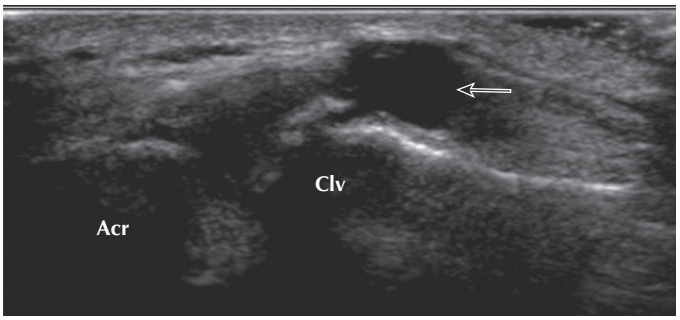
**Figure 64.16.** Acute acromial fracture. *FCR,* Flexor carpi radialis.




**A.** Normal image of acromioclavicular joint.




**B.** Osteoarthritis of the acromioclavicular joint with hypoechoic effusion *(asterisks)* and cortical irregularity.




**C.** Distal clavicular osteolysis with associated ganglion cyst *(open arrow)* presenting in a competitive bodybuilder. Note the fragmentation at the distal clavicle.

**Figure 64.17.** Acromioclavicular joint disorders. *Acr*, Acromion; *Clv*, clavicle.

## Joint and Cartilage

### NORMAL SONOGRAPHIC APPEARANCE

**Joint:** Incomplete evaluation due to same limitations as bone; can identify hypo/anechoic joint effusions, certain synovial disorders, and loose bodies within the visualized portion of a joint

**Hyaline cartilage:** Hypo/anechoic and noncompressible (helpful distinction from fluid collection); uniform thickness

**Fibrocartilage:** Hyperechoic or mixed echogenicity with uniform echotexture ("salt and pepper" appearance)

### GENERAL PATHOLOGIC FEATURES

- Joint effusion, nonspecific sign of intra-articular dysfunction
- Hyperemia within synovium consistent with synovitis
- Nonuniformity of hyaline cartilage can be seen with chondral injuries.
- Focal hypoechoic defect in fibrocartilage with or without secondary signs of parameniscal cyst, joint effusion, etc.

### SPECIFIC PATHOLOGIES

- Acromioclavicular (AC) joint disorders (Fig. 64.17)
- Knee joint effusion (Fig. 64.18)

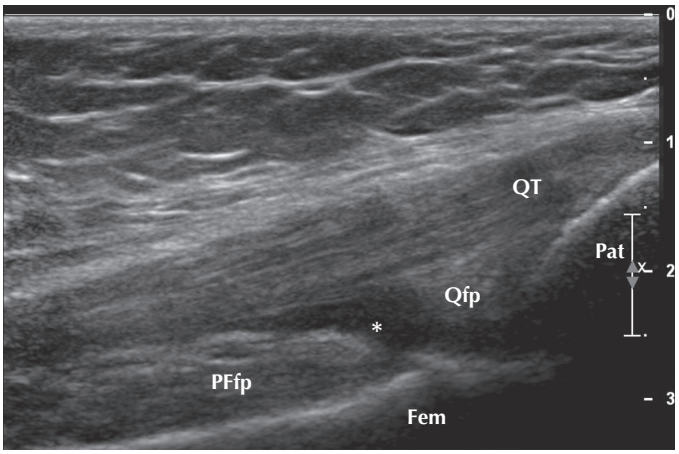
## Vessels

### NORMAL SONOGRAPHIC APPEARANCE

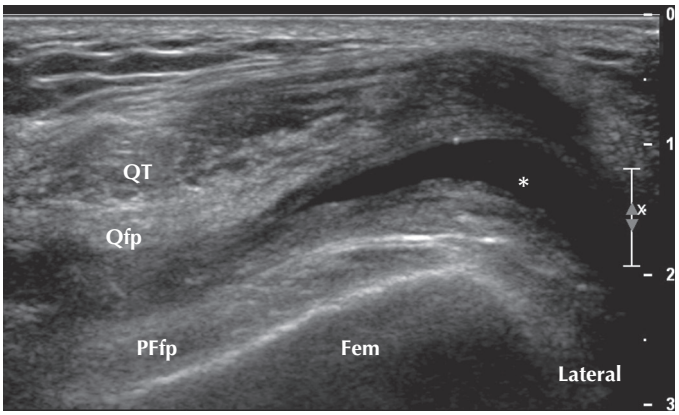
- Hypo/anechoic tubular structures
- **Arteries:** Pulsatile, thicker walls, less compressible
- **Veins:** Thinner walled, easily compressible, nonpulsatile

### GENERAL PATHOLOGIC FEATURES

- Typically limited evaluation with sports ultrasound and referred for formal vascular study as needed




**A.** Longitudinal image of the knee demonstrating small effusion in the suprapatellar recess *(asterisks)*. The locations of the pre-femoral and quadriceps fat pads are key to identifying the suprapatellar recess. The quadriceps tendon is seen in long axis inserting onto the patella.




**B.** Corresponding transverse image of the knee demonstrating effusion within the superolateral recess *(asterisks)*.

**Figure 64.18.** Knee joint effusion. *Fem*, Femur; *Pat*, patella; *PFfp*, pre-femoral fat pad; *Qfp*, quadriceps fat pad; *QT*, quadriceps tendon.

- Focal enlargement concerning for aneurysm; noncompressibility concerning for thromboembolism

## Nonmusculoskeletal

While clinical applicability is well established, role in sports medicine remains to be clearly defined; several potential uses are evident, but training and expertise may vary among sports medicine physicians and currently outside of scope of usual practice; potential for initial screening at point of care and triage for more definitive evaluation and treatment by an appropriate subspecialist

### Chest/Abdominal Trauma

FOCUSED ASSESSMENT WITH SONOGRAPHY FOR TRAUMA (FAST)

- Goal is identifying any free fluid (blood) in peritoneum to expedite the time to more definitive care; low sensitivity but high specificity; negative study does not exclude the potential for serious injury.
- Four standard views: hepatorenal recess (Morison's pouch), splenorenal recess, bladder, and pericardial

SOLID ORGAN INJURY

- Used to identify injury to the liver, spleen, and kidney; low sensitivity; any positive finding requires urgent referral
- **Laceration:** Hypo/anechoic fluid collection perpendicular to organ capsule
- **Contusion:** Inhomogeneous hypoechoic fluid collection

PNEUMOTHORAX

- Sensitivity and specificity better than chest radiograph
- **Criteria for diagnosis:** (i) absence of lung sliding (identification = 100% negative predictive value), (ii) disappearance of "B-lines" (visualization = 100% negative predictive value), and (iii) M-mode transition from "sandy beach" in normal lung to "barcode" in pneumothorax

### Cardiology

- Identification of pericardial effusion/tamponade
- Evaluation of central venous volume status (inferior vena cava collapse)
- Limited echocardiography may identify athletes at a risk of sudden cardiac death during pre-participation physical examination.

### Ocular Trauma

- Evaluation of posterior chamber trauma (retinal and vitreous detachments)
- Anterior chamber difficult to assess
- Normal posterior chamber is anechoic. Any echogenic material suggestive of injury and requires immediate referral

# INTERVENTIONAL SPORTS ULTRASOUND

## Injections—General Considerations

### Indications for Ultrasound Guidance

- No universally accepted indications
- Common indications may include the following:
  - Failed palpation-guided injection
  - Diagnostic aspiration without clinically present effusion
  - Diagnostic injections wherein accurate placement is required for diagnosis
  - Inability to identify surface/palpation landmarks due to body habitus, deformity, or deep location of target structure
  - Therapeutic injection where benefit requires accurate placement (i.e., no regional or systemic effects)
- Reduce risk of complication (proximity to neurovascular structures or organs at risk, anticoagulation, and bleeding diathesis)

### Contraindications for Ultrasound Guidance

- General procedural contraindications apply. There are no known contraindications specific to ultrasound.

### Accuracy, Efficacy, and Cost Effectiveness

- Ultrasound-guided injections (USGIs) are more accurate than landmark-guided injections (LMGIs). Strength-of-Recommendation Taxonomy (SORT) evidence rating A: site- and practitioner-specific variability
- USGIs are more efficacious than LMGIs. SORT evidence rating B: numerous study limitations, including lack of specific target and diagnosis; inherent limitations related to corticosteroid injection, which predominates the literature; obvious potential to reduce complications and provide more reliable information in setting of diagnostic injections
- USGIs are more cost-effective than LMGIs. SORT evidence rating B: currently a limited number of studies that evaluate comparative cost

### Technique

- Author's preferred technique is presented, but alternative techniques may be preferred in certain clinical situations.
- Sterile technique is preferred, including sterile transducer cover, sterile acoustic coupling gel, and sterile gloves. However, this is

an individual practice decision. The patient's skin should always be cleansed with an antiseptic cleanser before puncture.

- Needle size is determined by location of target, patient body habitus, and clinician preference. In general, smaller-gauge needles are more comfortable for patients and are readily identified with appropriate scanning technique. In cases of aspiration, a ≥20-gauge needle is typically required.
- Injectate volume will depend on clinical scenario and practitioner preference. One should always consider the goals of the injection and proceed accordingly (e.g., diagnostic vs. therapeutic injection). Certain injection sites, such as the AC joint, will accept a very limited volume, and overdistension can be uncomfortable and limit the diagnostic utility of injection.

## Specific Injection Techniques

### Subacromial–Subdeltoid Bursa Injection

**Patient position:** Supine, head of the bed elevated 30–45 degrees
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer placed in the LAX of the supraspinatus tendon with the acromion in view to identify the subacromial bursa in the tissue plane between the rotator cuff and the deltoid. Needle is advanced in-plane with transducer from anterolateral to posteromedial.

### Glenohumeral Joint Injection

**Patient position:** Side lying
**Preferred transducer:** Curvilinear array transducer will allow better needle visualization secondary to steep angle required
**Technique:** Transducer placed in the anatomic transverse oblique plane over the posterior glenohumeral joint in LAX of the infraspinatus tendon. Needle is advanced in-plane posterolateral to anteromedial as needle tip is guided between the humeral head and the glenoid labrum (Fig. 64.19).

### Acromioclavicular (AC) Joint Injection

**Patient position:** Supine, head of the bed elevated 30–45 degrees
**Preferred transducer:** High-frequency linear array

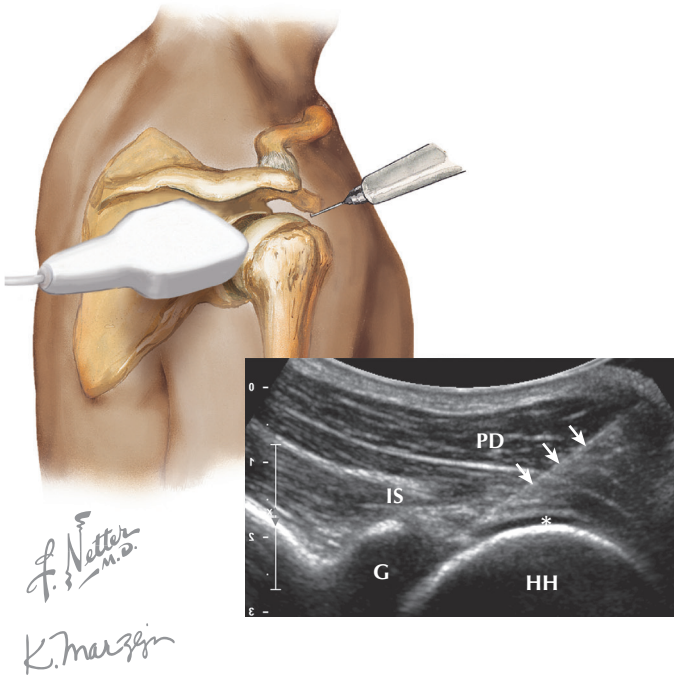



**Figure 64.19.** Glenohumeral joint injection: posterior approach, *arrows* indicate needle and *asterisks* indicate articular cartilage. *G*, Glenoid; *HH*, humeral head; *IS*, infraspinatus; *PD*, posterior deltoid.

**Technique:** Transducer is placed in an anatomic sagittal plane over the AC joint. It may be helpful to identify the clavicle and translate the transducer laterally until the joint space is identified. Needle is advanced in-plane anterior to posterior (Fig. 64.20).

### Bicipital Tendon Sheath Injection

**Patient position:** Supine, head of the bed elevated 30–45 degrees, forearm in supination
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer is placed in an anatomic transverse plane to visualize long head of the biceps tendon in SAX within bicipital groove. Ascending branch of the anterior humeral circumflex artery should be identified with Doppler and avoided. Needle is advanced in-plane from lateral to medial as the needle tip is guided into the tendon sheath and care is taken to avoid intratendinous injection.

### Elbow Joint Injection

**Patient position:** Supine, elbow flexed 30–45 degrees with palm resting on lower abdomen
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer is placed transverse to posterolateral aspect of radiocapitellar joint. Needle is advanced out-of-plane using a walk-down technique into the joint.

### Carpal Tunnel Injection

**Patient position:** Seated or supine with forearm supinated (palm up)
**Preferred transducer:** High-frequency linear array transducer; a small-footprint transducer (i.e., "hockey stick") is preferred, if available.
**Technique:** Transducer is placed in an anatomic transverse plane over proximal carpal tunnel inlet, and the median nerve is identified in SAX. Needle is advanced in-plane from ulnar to medial superficial to the ulnar nerve and artery into the carpal tunnel adjacent to the median nerve. An "oblique stand-off" technique, where ample sterile acoustic coupling gel is used to identify the needle before skin puncture, should be considered. Depending on physician preference and skill level, hydrodissection of the median nerve may be performed as the injectate is placed both above and below the nerve, and any adhesions to the transverse carpal ligament are freed.

### Wrist Joint Injection

**Patient position:** Seated, wrist flexed 20–40 degrees with forearm pronated and supported on a pillow

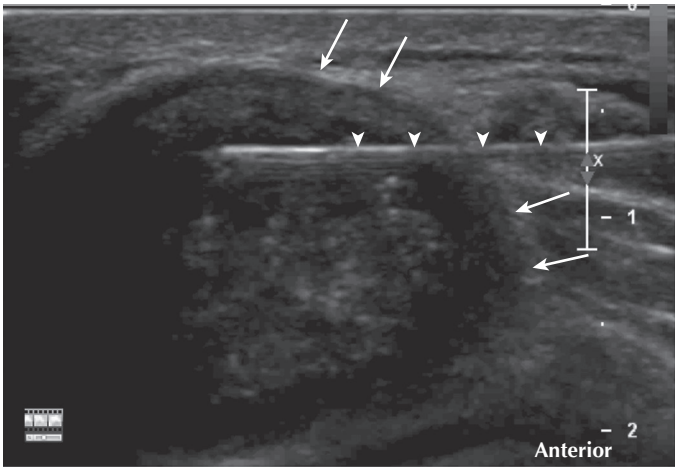



**Figure 64.20.** Acromioclavicular joint injection: anterior approach. *Arrowheads* indicate needle, *arrows* indicate hip joint capsule.

**Preferred transducer:** High-frequency linear array transducer; a small-footprint transducer (i.e., "hockey stick") is preferred, if available.
**Technique:** Transducer is placed in anatomic sagittal plane over the wrist (radio-lunate). Needle is advanced in-plane from distal to proximal into radiocarpal joint. An "oblique stand-off" technique, where ample sterile acoustic coupling gel is used to identify needle before skin puncture, should be considered.

### First Dorsal Compartment Tendon Sheath Injection

**Patient position:** Seated or supine with forearm in neutral position
**Preferred transducer:** High-frequency linear array transducer; a small-footprint transducer (i.e., "hockey stick") is preferred, if available
**Technique:** Transducer is placed in an anatomic transverse plane over distal radius and tendons of first dorsal compartment identified in SAX. Superficial radial nerve and radial artery should be identified during preprocedural planning. Needle is advanced in-plane from volar to dorsal as needle tip is guided into tendon sheath. Note should be made of any subcompartmentalization within the first dorsal compartment, and injectate spread around both tendons should be documented.

### Hip Joint Injection

**Patient position:** Supine, hip in neutral rotation
**Preferred transducer:** Curvilinear array transducer
**Technique:** Transducer is placed in anatomic transverse oblique plane over anterior hip (axis of femoral neck), and the femoral head–neck junction is identified. Position of femoral neurovasculature should be noted medial to the target. Needle is advanced in-plane from inferolateral to superomedial as the needle tip is guided into the anterior joint recess near the femoral head–neck junction (Fig. 64.21).

### Greater Trochanteric Bursa Injection

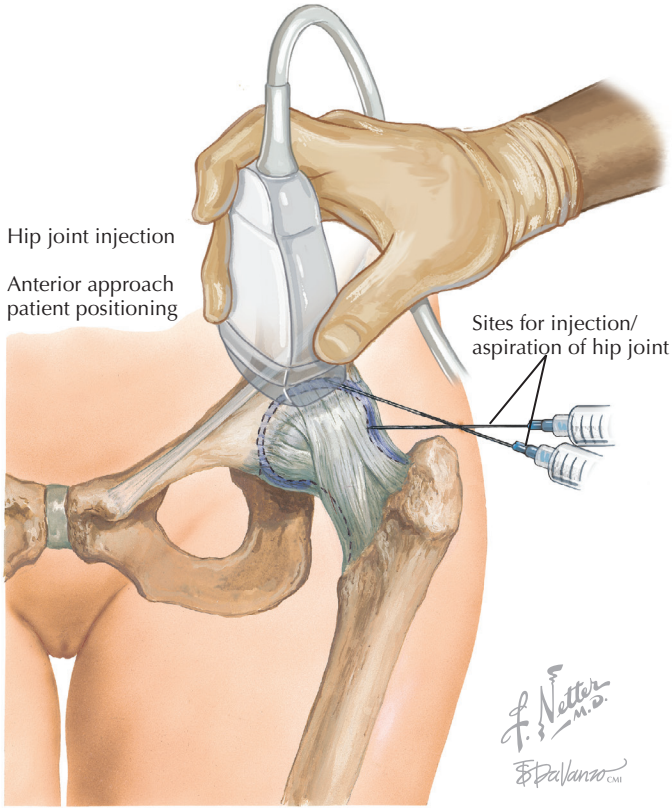
**Patient position:** Side lying
**Preferred transducer:** High-frequency linear array transducer is typically preferred; however, body habitus may necessitate a lower-frequency curvilinear array transducer in certain patients.
**Technique:** Transducer is placed in an anatomic transverse plane over the greater trochanter and the lateral and posterior facets of greater trochanter are identified. Gluteus medius tendon lies on the lateral facet, and gluteus maximus muscle overlies the posterior facet. Needle is advanced in-plane from posterior to anterior as the needle tip is guided into the subgluteus maximus bursa (the tissue plane between the gluteus maximus/iliotibial band superficially and the deep gluteus medius tendon).

### Knee Joint Injection

**Patient position:** Supine, knee flexed 20 degrees and supported on pillow
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer is placed in an anatomic transverse plane over the distal femur, and the suprapatellar recess is identified deep to quadriceps tendon (or quadriceps fat pad if transducer is closer to patella) and superficial to the prefemoral fat pad. Needle is advanced in-plane lateral to medial as the needle tip is guided into the joint recess (Fig. 64.22).

### Iliotibial Band Bursa Injection

**Patient position:** Side lying, knee flexed 20 degrees
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer is placed in an anatomic transverse plane over the lateral femoral condyle, and the iliotibial band is identified in SAX. Needle is advanced in-plane posterior to anterior as the needle tip is guided just deep to the iliotibial band into the region of the bursa. Common fibular nerve should be identified during preprocedural planning to ensure typical location posterior to the needle entry site.



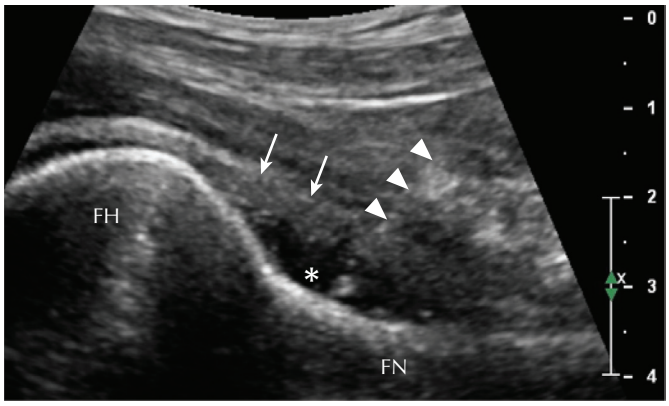




**Figure 64.21.** Hip joint injection: anterior approach. *Arrowheads* indicate needle, *arrows* indicate hip joint capsule, and *asterisks* indicate injectate within anterior hip recess. *FH*, Femoral head; *FN*, femoral neck.

### Tibiotalar Joint Injection

**Patient position:** Side lying, ankle in slight inversion supported by pillow
**Preferred transducer:** High-frequency linear array
**Technique:** Transducer is placed in an anatomic transverse oblique plane over the fibular tip, and the lateral gutter of the tibiotalar joint is identified deep to the anterior talofibular ligament (ATFL). Needle is advanced out-of-plane anterolateral to posteromedial into the lateral joint recess.

### Fibular (Peroneal) Tendon Sheath Injection

**Patient position:** Side lying, ankle in slight inversion supported by pillow

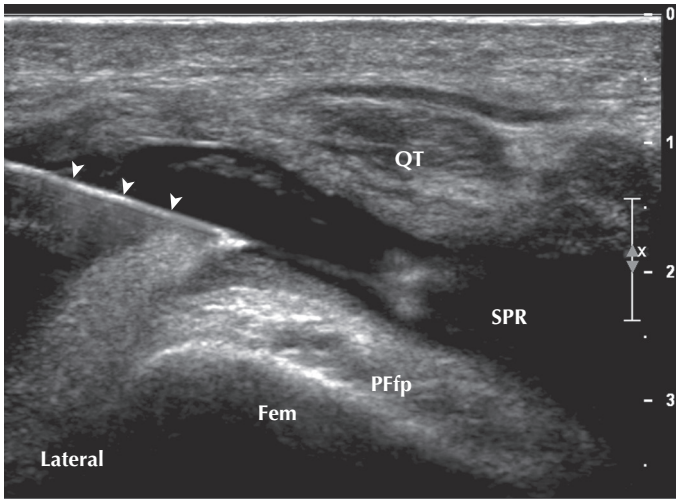



**Figure 64.22.** Knee joint injection: superolateral approach, *arrowheads* indicate needle. *Fem*, Femur; *PFfp*, prefemoral fat pad; *QT*, quadriceps tendon; *SPR*, suprapatellar recess.

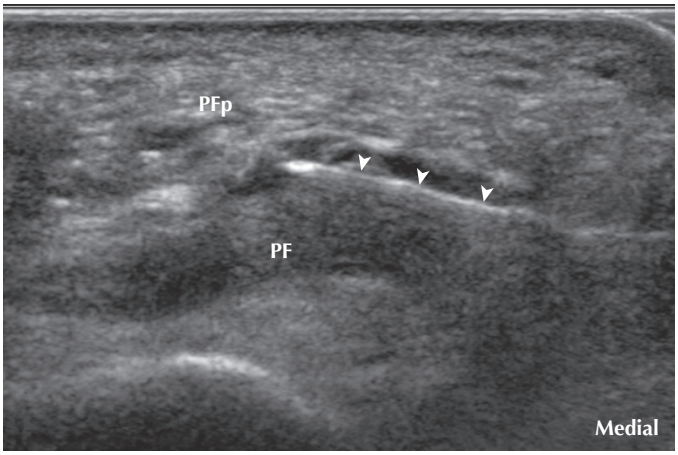



**Figure 64.23.** Plantar fascia injection, *arrowheads* indicate needle. *PF*, Plantar fascia; *PFp*, plantar fat pad.

**Preferred transducer:** High-frequency linear array transducer; a small-footprint transducer (i.e., "hockey stick") is preferred, if available

**Technique:** Transducer is placed in an anatomic transverse plane just inferior to tip of the lateral malleolus, and the fibular tendons are identified in SAX. Needle is advanced in-plane anterior to posterior into the tendon sheath.

### Plantar Fascia Injection

**Patient position:** Prone, foot hanging free off the end of examination table

**Preferred transducer:** High-frequency linear array

**Technique:** Transducer is placed in an anatomic transverse plane over the plantar heel, and the plantar fascia origin is identified in SAX at calcaneus. Needle is advanced in-plane medial to lateral as the needle tip is guided to the superficial/plantar surface of the plantar fascia. Care should be taken to avoid injection into the plantar fat pad (Fig. 64.23).

## Advanced Procedures—General Considerations

- Earlier advanced procedures such as calcific tendinopathy barbotage and percutaneous needle tenotomy have proven safe and effective means of treating certain tendinopathies in an outpatient clinical setting.
- Techniques and more specialized equipment continue to evolve with general principle of taking proven surgical concepts and making these less invasive via a percutaneous approach and live ultrasound guidance.
- Examples include percutaneous tenotomy with specialized tools, tendon scraping, carpal tunnel release, trigger finger release, peripheral nerve hydroneurolysis, fascial release for chronic exertional compartment syndrome, and various regenerative medicine techniques.
- Potential for decreased morbidity, improved patient outcomes, and overall cost savings, but definitive evidence for these claims is currently lacking

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 65 INJURY PREVENTION PROTOCOLS

Kevin E. Wilk • Robert A. Williams Jr.

## GENERAL PRINCIPLES

Sport-specific injury prevention training programs are utilized across the country as an aid to increase performance and decrease injury rates. Two common areas of athletic injuries include the shoulder and knee. Among the athletic population, injuries to the throwing shoulder are becoming more apparent as a result of overuse and poor conditioning. Similarly, the number of anterior cruciate ligament (ACL) knee injuries continues to rise each year among the athletic population. This chapter focuses on specific prevention protocols for reducing the likelihood of athletic knee injuries and specific to ACL pathology and injuries to the throwing shoulder of young and elite athletes.

## ACL INJURY PREVENTION

### Epidemiology and Injury Statistics

- Injury to the ACL can be functionally debilitating and often requires surgical intervention.
- ACL injuries are common in sports and strenuous work activities.
- Approximately 200,000 ACL injuries occur annually in the United States.
- Over a 20-year period, ACL surgery procedures have increased by 58%, with 148,714 ACL surgeries performed in 2013 (PearlDiver Technologies. PearlDiver supercomputer database. Available at: http://www.pearl-diverinc.com/).
- One in 3,500 people will sustain an ACL injury.
- In total, 62%–66% of ACL injuries are sports related, often in a noncontact manner (i.e., running, cutting, or landing from a jump).
- Females are 4–6 times more likely to sustain an ACL injury than their male counterparts.
- There is a 10-times greater risk of developing osteoarthritis in an injured ACL knee.
- In total, 67% of ACL injuries occur in individuals aged 15–29 years.
- Athletes are at an increased risk of contralateral ACL injury following a first-time ACL injury.

### Risk Factors for ACL Injury

- Increased knee valgus with running, cutting, and jump landing
- Decreased knee flexion angles with jump landing; this is often present in a quadriceps-dominant knee
- Decreased hamstring-to-quadriceps strength ratios: Females rely more on their quadriceps to stabilize their knee joints and tend to have lower hamstring-to-quadriceps muscle strength ratios. Hamstring-to-quadriceps ratios of <75% and a bilateral hamstring difference of >15% have been correlated to a higher incidence of ACL injuries in female athletes (Knapik et al: *AJSM* 1991).
- Neuromuscular control deficits can create a difficulty in generating muscular force. This limits the ability to resist displacing loads through dynamic stabilization of the knee.
- A smaller intercondylar notch has been found to be associated with the risk of ACL rupture in a skeletally immature population.
- Lateral trunk displacement with jump landing
- Poor hip strength and control
- Ligamentous laxity: Athletes with greater generalized laxity demonstrate increased midfoot loading and valgus collapse. This valgus force can be most apparent during running, cutting, or landing from a jump.
- Hormonal changes

### Review of Literature

- A 6-week prevention program focusing on plyometrics, neuromuscular and strength training, flexibility, and appropriate body mechanics reported that subjects of an untrained group had a 2.4–3.6 higher ACL injury rate.
- A study of trained soccer players aged 14–18 years who performed preventative stretching, strengthening, plyometrics, and agility drills reported an 88% reduction in the rate of ACL injuries over 1 year and a 74% reduction in ACL injuries over 2 years. Those in the untrained group were at a 10 times greater risk of ACL injury.
- A study of the Norwegian team handball players analyzed over 3 years reported significantly fewer noncontact injuries in participants of the intervention group. Subjects performed proprioceptive, agility, plyometric, and balance drills 3 times a week for 5–7 weeks before the season and then once a week during the season.
- In a program of 4,700 ski instructors and patrollers that consisted of educational training on avoiding high-risk behaviors, recognizing potentially dangerous situations, and how to respond to situations, the injury rate was reduced by 62% over a span of 3 years.
- A study of 600 Italian semiprofessional and amateur soccer players participating in a proprioception and balance-training program reported reduced ACL injury rates by 87% over a duration of 3 seasons.
- An 8-week preseason program for German female handball players reported significantly lower rates of ankle and knee injuries. It emphasized the role of educational training with regard to injury mechanisms. Subjects participated in balance-board training drills that consisted of double- and single-leg stance, catch with a partner, and throws at a goal. Jump training exercises were also performed, including forward, backward, and lateral jumps, drop jumps onto a mat, and jump landing with eyes closed.

### Prevention Training

- ACL prevention training should focus on the following:
  - Hip control
    - Aimed to prevent valgus knee collapse
    - Target muscle groups should be those that control hip abduction, hip external rotation (ER), and hip extension.
    - Example exercises include hip external/internal rotation (ER/IR) with resistance band (Theraband CLX; Performance Health, Akron, OH), single-leg front step downs, lateral slides with resistance bands (Theraband CLX; Performance Health, Akron, OH) (Fig. 65.1), unilateral Romanian deadlifts (RDLs), side-lying manual resistance clams, and stability ball bridging.

**Figure 65.1.** Lateral slides with resistance bands (Theraband CLX; Performance Health, Akron, OH) around the distal femur to strengthen gluteus medius musculature of the hip.

**Figure 65.2.** Manual perturbations with ball catches.

- Neuromuscular control and proprioception
  - Athletes should be aware of where their knee is relative to their body in space.
  - Dynamic joint stability is dependent on neuromuscular response time.
  - Improving neuromuscular co-activation enhances knee stability.
  - Manual perturbations can be implemented into drills to create a postural disturbance, thus further enhancing neuromuscular control.
  - Neuromuscular training can also help improve an athlete's confidence.
  - Example exercises include balance board drills with double- or single-leg stance. The difficulty of this activity can be progressed by having the athlete play catch with a ball. This drill can be further enhanced by having the athlete maintain balance while stabilizing against manual perturbations, while also playing catch (Fig. 65.2).
- Trunk control and core strengthening
  - Important in preventing lateral displacement
  - Females tend to land from jumping with greater lateral trunk motion than males.
  - Example exercises include prone or side planks and single-leg medicine ball cross-patterns on a foam disc (Fig. 65.3). Cross patterns are performed by having the athlete move extended arms side-to-side and then up/down.
- Hamstring strengthening
  - An athlete is dependent on muscular co-activation to maintain appropriate knee joint dynamic stabilization.
  - The goal is to eliminate a quadriceps-dominant knee and establish an appropriate hamstring-to-quadriceps muscle strength ratio.
  - Examples exercises include physioball bridging with concomitant hamstring curls (Fig. 65.4), prone machine hamstring curls, and eccentric Nordic hamstring drills.
- Appropriate running, cutting, and landing mechanics
  - Fatigue is a risk factor for musculoskeletal injury; therefore, endurance training plays a key role in injury prevention.

**Figure 65.3.** Single-leg cross pattern on a foam disc to alter the patient's center of gravity.

  - Lower extremity muscle fatigue alters knee kinematics and kinetics during landing from a jump stop. This creates an increase in peak tibial anterior shear forces, increase in valgus knee moment, and decreased knee flexion.
  - Encourage a balanced positioning of the hip and knee in a flexed position.
  - Example exercises include agility ladders, line jumps, and plyometric box jumps, with emphasis on appropriate body mechanics during landing.

## PREVENTION OF THROWING INJURIES

### Epidemiology and Injury Statistics

- Shoulder and elbow injuries are common in baseball and appear to be increasing every year.
- In major league baseball (MLB):

**Figure 65.4.** Stability ball hamstring curl to enhance recruitment of gluteus maximus and hamstring musculature.

- 67% of all injuries in pitchers occur in upper extremities, with the shoulder being the most commonly injured joint.
- Pitchers are 2.5 times more likely to sustain an upper extremity injury compared with position players.
- 31% of all injuries in pitchers involve the shoulder joint, while 26% of injuries involve the elbow joint.
- 72% of all disabled list days are accounted to shoulder and/or elbow injuries.
- 61% of all disabled list days are taken by pitchers.

- In youth baseball:
  - Over 17 million kids play youth baseball in the United States.
  - 50% of players (aged 9–14 years) complained of shoulder or elbow pain.
  - Surgeries appear to be increasing in youth baseball players.

## Risk Factors to Throwing Injuries

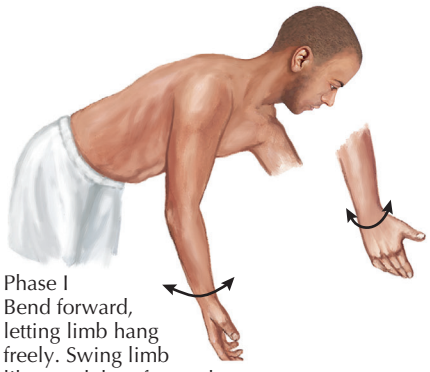
- Pitching at maximum effort can overload the stabilizing structures and lead to an accelerated rate of degradation of soft tissue intrinsic stabilizers.
- Pitching too many innings throughout the season can also result in overutilization of static and dynamic stabilizing structures.
- Pitching while fatigued can result in alteration of throwing mechanics, directly affecting the performance. Effects of fatigue can result in an increase of superior humeral head migration with arm elevation, diminished proprioception, and altered scapular position. Injury often can prelude as a result of these alterations.
- Pitching year round: It is encouraged that no overhead throwing be performed for at least 4 months throughout the year. Furthermore, 2–3 months of rest from throwing is encouraged at the end of a baseball season.
- Inappropriate throwing mechanics can lead to excessive strains in the upper extremity. Appropriate mechanics should be emphasized throughout the entirety of the throwing motion.
- Usage of breaking ball pitches in immature throwers alters throwing kinetics, particularly in the presence of poor throwing mechanics.
- Participating as a pitcher and catcher throughout the season drastically increases throwing volumes. A Little League baseball rule since 2010 prohibits a pitcher from going to a catcher, and vice versa, during the same game.
- Playing on multiple teams in various leagues increases the volume of throws on the upper extremity.
- Poor conditioning as a result of inadequate shoulder mobility and strength, neuromuscular control, dynamic stability, core and lower extremity strength, and posture

## Review of Literature

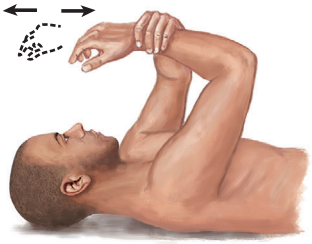
- Prevention programs addressing posture, mobility, flexibility, strength, endurance, and power have been proficient at reducing injury rates while also enhancing performance.
- In adolescent baseball players (aged 11–15 years), performance of the Thrower's Ten program over a span of 4 weeks demonstrated a 2% increase in throwing velocity.
- A 2% increase in throwing velocity was also demonstrated in adolescent baseball players by performing a 6-week plyometric training program.
- Stretching and flexibility programs have demonstrated improvements in total rotation motion (TROM). TROM is a total motion concept that combines ER and IR. A TROM of >5 degrees, comparing dominant to nondominant shoulders, can place throwers at a 2.5 times greater risk of upper extremity injury.

## Prevention Training

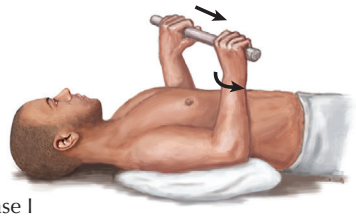
- Pitch counts
  - Pitch count charts can be utilized (game, season, and year) to maximize the longevity of the thrower.
  - The Little League pitch count rule limits the amount of throws a pitcher is allowed per game: pitchers aged 7–8 years are allowed 50 pitches, 9–10 years (75 pitches), 11–12 years (85 pitches), 13–16 years (95 pitches), and 17–18 years (105 pitches).
- Rest
  - Rest allows appropriate recovery in an athlete.
  - The Little League pitch count rule requires 4 days of rest when a pitcher throws >66 pitches in a game, 3 days of rest with 51–65 pitches, 2 days of rest with 36–50 pitches, and 1 day of rest with 21–35 pitches.
- Physical characteristics
  - Range of motion and flexibility exercises should target motions of shoulder flexion, shoulder ER, shoulder IR, and horizontal adduction (Fig. 65.5).
  - Emphasis to pectoralis minor stretching should be included as well. A wall corner stretch is an effective technique to maintain and improve pectoralis minor flexibility.
  - The modified cross body stretch (Fig. 65.6) is effective at improving flexibility of the posterior rotator cuff following episodes of throwing.
  - Stretching exercises should be held for a minimum of 30 seconds to promote plastic deformation and should be repeated thrice.
  - Strengthening exercises should target the posterior rotator cuff and scapular stabilizer musculature.
  - Standing ER with resistance bands (CLX), side-lying ER, standing full cans, and prone rows with ER are all excellent exercises that target the rotator cuff musculature.
  - Standing low and high rows with resistance bands (CLX), prone horizontal abduction, and the modified robbery with resistance bands (CLX) (Fig. 65.7) are great exercises that target the scapula musculature.
- Neuromuscular control
  - Neuromuscular control is an efferent motor output in response to afferent sensory stimuli.
  - Neuromuscular control is fundamental in the generation of dynamic shoulder stability to minimize the risk of injury during the act of throwing a baseball.
  - The rotator cuff muscles aid in dynamic stability of the shoulder through continuous interplay of synergistic



Phase I
Bend forward, letting limb hang freely. Swing limb like pendulum forward and backward and side to side. Rotate hand inward and outward.

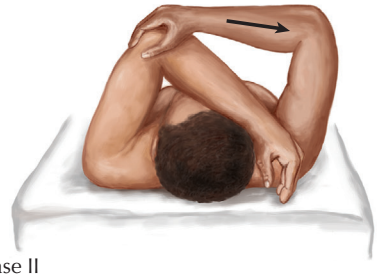


Phase I
Raise hand over hand, using opposite arm for power.

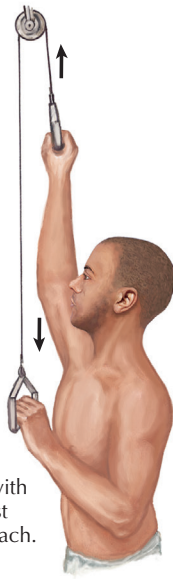


Phase I
External rotatation arm using a broomstick. The passive good arm is used for power to move the affected shoulder.

Phase II
Internally rotate arm by pulling wrist backward and upward behind back with good arm.



Phase II
Supine cross-body adduction to stretch the posterior capsule



Phase I
Raise affected arm with pulley placed at least 2 feet higher than reach.

**Figure 65.5.** Basic, passive, and active assisted range-of-motion exercises.

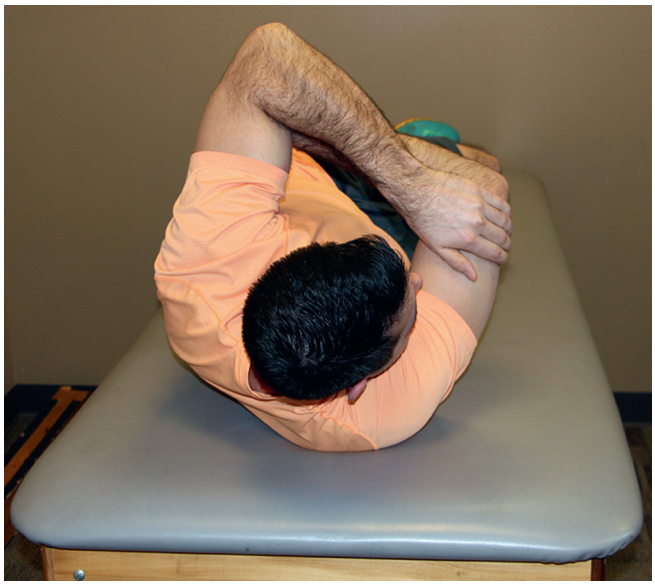


**Figure 65.6.** Modified cross body stretch. The athlete passively and horizontally adducts the shoulder as the scapula is stabilized against the table, while IR is passively performed with counter-pressure of the opposite forearm.

Modified robbery with resistance band (Theraband CLX; Performance Health, Akron, OH) targets the lower trapezius musculature.

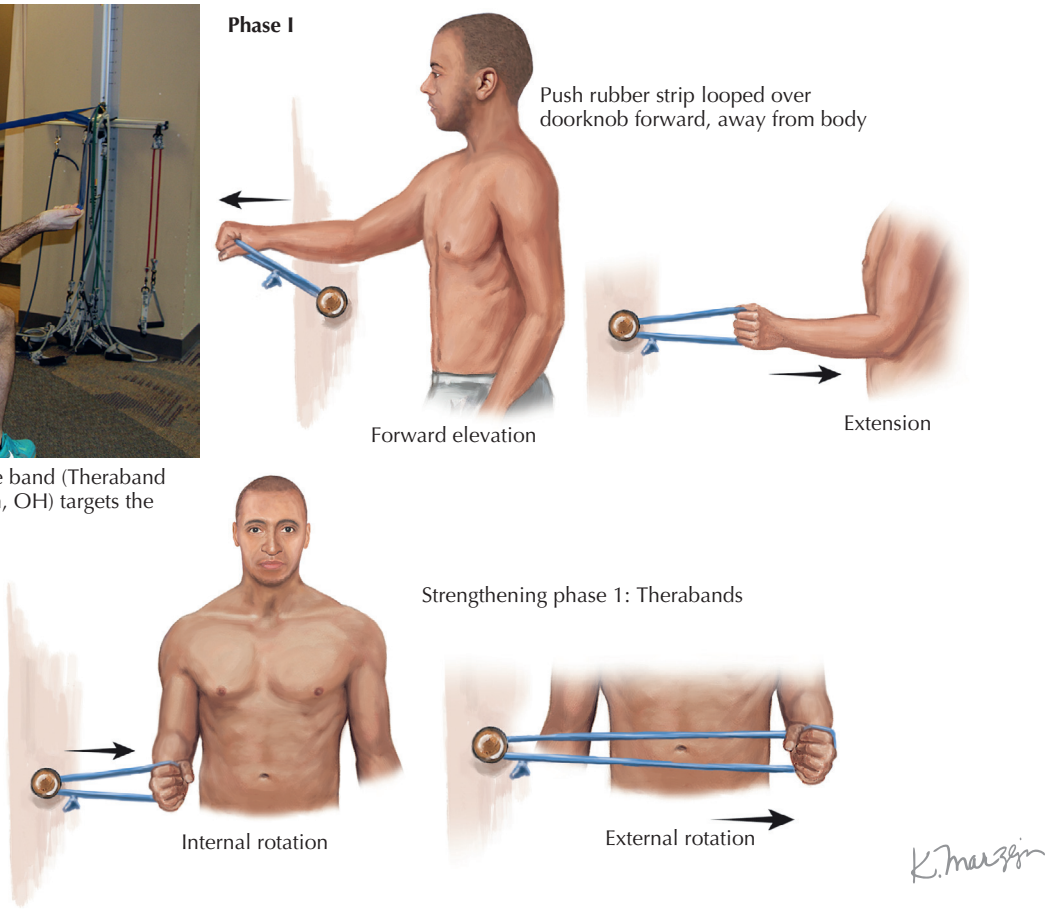



**Figure 65.7.** Shoulder and lower trapezius musculature strengthening.

muscles, providing compression and congruency to the glenohumeral joint.

- Effective neuromuscular control drills for the shoulder include rhythmic stabilizations, closed kinetic chain exercises, and plyometric training.
- Closed kinetic chain exercises stress the joint in a load-bearing position, resulting in joint approximation, thus stimulating receptors and facilitating in co-contraction of the shoulder force couples.
- Plyometric exercises provide quick, powerful movements by using the elastic and reactive properties of the muscle to generate maximum force production. These exercises increase the speed of the stretch reflex, desensitize the Golgi tendon organ, and increase neuromuscular coordination.
- Plyometric training should begin with two-handed drills into a plyoback. Exercise examples include chest pass (Fig. 65.8A), side-to-side throws (Fig. 65.8B), side throws (Fig. 65.8C), and overhead soccer throws (Fig. 65.8D).
- Once athletes are comfortable with two-handed drills, they can progress to one-handed drills. Exercise examples include standing one-handed throws (Fig. 65.9A) into a plyoback, wall dribbles (Fig. 65.9B), and throwing simulation, which can enhance synchronicity of throwing and improve mechanics. An athlete throws a 1-lb plyoball from 20 feet at a plyoback (Fig. 65.9C).
- Total body conditioning
  - The core and lower extremities are of utmost importance in the development and transfer of power in the act of throwing.
  - Adolescent and preadolescent aged athletes often present with poor core, hip, and leg strength.
  - Poor strength, endurance, and/or neuromuscular control of the lower body can have a significant impact on the forces experienced by the upper extremity and influence pitching mechanics.
  - Exercises should focus on linking the shoulder and lower extremities because of the energy transfer that occurs with overhead throwing.
  - Exercises such as front step downs (Fig. 65.10), lateral slides with resistance bands (Theraband CLX; Performance Health, Akron, OH), hip ER/IR with resistance bands (Theraband CLX; Performance Health, Akron, OH), side-lying clams, and single-leg RDLs are all excellent exercises to enhance lower extremity and core strength.
- Endurance
  - According to a motion analysis study, shoulder ER and ball velocity decreased once a thrower became fatigued.
  - Muscle fatigue has also been attributed to superior humeral head migration upon initiation of arm elevation, which can contribute to subacromial impingement.

**A.** Chest pass

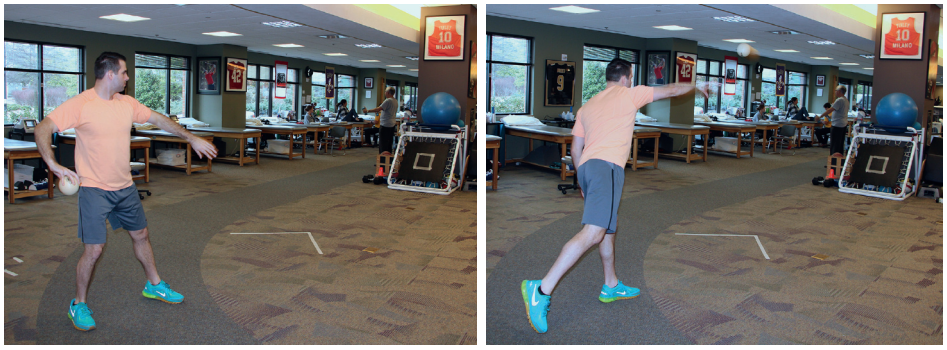
**B.** Side-to-side throws

**C.** Side throws

**D.** Overhead soccer throws

**Figure 65.8.** Two-handed plyometric drills.

**A.** Overhead throws **B.** Wall dribbles



**C.** Throwing simulation

**Figure 65.9.** One-handed plyometric drills. **A,** Overhead throws. **B,** Wall dribbles. **C,** Throwing simulation.

**Figure 65.10.** Front step down. The athlete is instructed to maintain proper alignment of the lower extremity to prevent valgus knee collapse or pelvic drop during the lowering phase of the exercise.

- Training activities that can help improve endurance of the shoulder joint complex include wall dribbles with a plyoball, side-lying ER ball flips with a #2 plyoball (Fig. 65.11), upper body cycling, and the Advanced Throwers Ten exercise program.

**Figure 65.11.** Side-lying ER ball flips with #2 plyoball for endurance training of shoulder external rotators.

- Throwing mechanics
  - Throwing with inappropriate or faulty mechanics can lead to shoulder pain or injury, or both, because of the abnormal stresses placed on various soft tissues.
  - Learn good throwing mechanics as soon as possible. It is encouraged that athletes learn in the following order: basic throwing, fastball pitching, and change-up pitching.

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Available at www.ExpertConsult.com.

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# FOOTBALL

# 66

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## GENERAL PRINCIPLES

### Overview

- Approximately 1.5 million athletes participate in American football in the United States.
- It has been competitively played at the college level for over 100 years. The "flying wedge" was a V-shaped formation used by Harvard in a game against Yale in 1892 and was thought to be associated with significant brutality and injuries; therefore, it was banned in 1894. Concerns regarding the safety issues of "flying wedge" led to the formation of the National Collegiate Athletic Association (NCAA).
- A total of 725 schools sponsor varsity college football, representing approximately 70,000 student-athletes playing college football.
- American football involves discontinuous sprint activity and requires strength; sport-specific skills vary depending on the position played.
- Various schemes and options are used to advance the team's objective: moving the ball down the field. There are four "downs" per possession, each with the goal of moving the ball forward by 10 yards, at which point four additional possessions are acquired. Teams score either by touchdown (6 points) or by kicking a field goal (3 points). Following a touchdown, the scoring team has the option of kicking for 1 point or attempting a play for 2 points. The team with the most points at the end of the game wins. Different rules regarding length of game, overtime, out of bounds, and possession are based on the level of play (high school, college, and professional).
- Football is a high-velocity contact/collision sport with an increased risk of injury. Physician coverage for games is optimal and often required, depending on the level played and the league and/or state regulations.
- Various medical as well as musculoskeletal issues must be considered in treating football players at all competitive levels.

### Injury Statistics

- At the college level, in a report reviewing sport-related injuries from 2009 to 2010 through 2013–2014, men's football accounted for the largest annual estimated number of injuries (47.2), the highest competition injury rate (30.0 per 1000 athlete-exposures [AEs]), and the third highest overall injury rate (9.2 per 1000 AEs) (Table 66.1). Football also accounts for the largest percentage of AEs, with 14.6% of all AEs and 31.2% of all male AEs. Football accounts for the largest proportion of injuries requiring >1 week before full participation (26.2%), surgery (40%), and emergency transport (31.9%).
- Between 1988–1989 and 2003–2004, the preseason injury rate was higher than both the regular season and postseason injury rates (7.05 vs. 2.02 and 7.05 vs. 1.70 per 1000 AEs, respectively). Preseason is a critical time to stress appropriate stretching, warm-up, fluid and nutritional replacements, recovery, technique, and supervision.
- For games, knee internal derangements, ankle ligament sprains, and concussions account for a majority of injuries (17.8%, 15.6%, and 6.8%, respectively). For fall practices, knee internal derangements, ankle ligament sprains, and upper leg muscle–tendon injuries account for a majority of injuries (12%, 11.8%, and 10.7%, respectively). Heat illness accounts for 3.9% of fall season injuries. Spring practice injuries are similar to those seen in the fall with regard to the most common body parts injured. Cervical spine injuries can have a catastrophic potential but fortunately have declined since rule modifications were made about tackling and blocking techniques, improved fitness, coaching, and equipment.
- Ankle sprains account for 13.6% of all injuries (0.83 injuries per 1000 AEs), anterior cruciate ligament (ACL) injuries for 3% of all injuries (0.18 injuries per 1000 AEs), and concussions for 6% (0.37 injuries per 1000 AEs). At the NCAA level, spring football ankle sprains account for 13.9% of all injuries (1.34 injuries per 1000 AEs), ACL injuries for 3.5% (0.33 injuries per 1000 AEs), and concussions for 5.6% of all injuries (0.54 per 1000 AEs).
- Player contact is the most common mechanism of injuries in football, and this is true for games as well as fall and spring practices.
- Player position plays a factor in the likelihood of injury, with game injuries by position being most common in a running back (19.6%), followed by quarterback (17.5%), linebacker (15.5%), flanker/wide receiver (14.4%), defensive back (11.7%), defensive lineman (11.3%), and offensive lineman (9.9%).

### Medical Problems

- Fatalities have been reported in both high school and collegiate football players, and from 1990 to 2010, 243 fatalities were reported to the National Center for Catastrophic Sports Injury Research, resulting in an average of 12.2 per year or 1 per 100,000 participants.
  - Of these, 164 were indirect (0.7 per 100,000 participants) and 79 direct (0.3 per 100,000 participants).
  - The most common causes of fatalities in this review were cardiac failure (41.2%), brain injury (25.5%), heat illness (15.6%), sickle cell trait (4.5%), asthma and commotio cordis (2.9%), embolism/blood clot (2.1%), cervical fracture (1.7%), and intra-abdominal injury, infection, and lightning (each 1.2%).
  - The risk of fatality was similar at high school and college levels for all causes with the exception of brain injuries, which were slightly more common at the college level.

#### Preparticipation Physical Examination

- Preparticipation physical examination is an important part of the initial evaluation of any athlete. The evaluation should be performed before the competitive season and with sport-specific objectives. Additional testing, such as the addition of electrocardiogram or sickle cell testing, has recently been debated (see Chapter 3, The Preparticipation Physical Evaluation).
- Three areas of importance in the preparticipation physical examination are cardiac, musculoskeletal, and neurologic. They are stressed not only in history but also on physical examination.
  - Family history of sudden cardiac death (SCD), including myocardial infarction before age of 50 years, Marfan syndrome, hypertrophic cardiomyopathy, and other abnormalities known to cause SCD
  - History of exertional syncope, chest pain, dizziness
  - History of prior injury or incomplete rehabilitation
  - History of previous head or neck injury, concussions, stingers or burners, as well as any work-up, in the past is important.

**TABLE 66.1 INJURY RATES (PER 1000 ATHLETIC EXPOSURES)**

| Sport | Practice | Game |
|---|---|---|
| Football | 5.8 | 39.9 |
| Wrestling | 9.0 | 38.5 |
| Men's Ice Hockey | 3.2 | 26.3 |
| Men's Soccer | 5.2 | 17.9 |
| Women's Soccer | 5.3 | 17.2 |
| Men's Basketball | 5.8 | 15.0 |
| Men's Lacrosse | 4.9 | 13.7 |
| Women's Gymnastics | 2.9 | 13.2 |
| Women's Ice Hockey | 3.2 | 11.2 |
| Women's Field Hockey | 5.1 | 10.5 |
| Women's Basketball | 4.9 | 10.4 |
| Men's Tennis | 2.9 | 10.0 |
| Women's Lacrosse | 4.2 | 9.7 |
| Women's Cross Country | 3.4 | 7.7 |
| Women's Tennis | 3.1 | 7.6 |
| Men's Baseball | 3.0 | 6.8 |
| Men's Indoor Track | 3.1 | 6.5 |
| Women's Softball | 3.5 | 5.9 |
| Women's Volleyball | 5.6 | 5.9 |
| Men's Outdoor Track | 2.3 | 5.4 |
| Men's Cross Country | 3.2 | 5.2 |
| Women's Outdoor Track | 2.7 | 5.1 |
| Women's Indoor Track | 3.8 | 4.4 |
| Men's Swimming and Diving | 2.1 | 2.0 |
| Women's Swimming and Diving | 2.1 | 1.9 |

Modified from NCAA Handbook 2014-2015. National Collegiate Athletics Association. 25th ed. Indianapolis, IN: August 2014; Kerr ZY, Marshall SW, Dompier TP, Corlette J, Klossner DA, Gilchrist J. College Sports-Related Injuries–United States, 2009-2010 through 2013-2014 Academic Years. *MMWR Morb Mortal Wkly Rep.* 2015;64(48):1330-1336.

  - Other areas of importance include exercise-induced asthma, single organs, heat intolerance, appliances (e.g., dental plates), medications, allergies, medical conditions, sickle cell disease, and immunizations.
- Historical questions to assess the risk of SCD, preexisting musculoskeletal injuries, or anatomic conditions that put athletes at risk of injury. **Thorough history is essential.**
- Physical examination
  - Emphasize cardiac, musculoskeletal, and neurologic systems.
  - Ensure adequate cervical spine protection, joint stability, absence of cardiac abnormalities, and neurologic integrity.
  - Screen for inflexibility, muscle imbalances, and inappropriate rehabilitation.
- **Nutrition issues:** Need for strength gaining often puts an athlete at risk of abuse of performance-enhancing drugs. Use of protein supplementation should be discussed. The athlete should understand basics about good nutritional balance and risks of excessive protein load as well as additional costs of protein supplements. A healthy, natural, and well-balanced diet (see Chapter 5, Sports Nutrition) should be emphasized. The use of unsupervised nutraceuticals should be discouraged.
- **Fluids:** A study in high school football players in 50-play scrimmage simulation demonstrated no difference in anaerobic performance with a 7% glucose polymer beverage containing electrolytes compared with water, but the beverage had a positive effect on maintaining plasma volume during recovery from anaerobic exercise.
- In 2003, as a response to heat-related deaths in preseason football, the NCAA instituted rules that addressed heat illness by requiring a 5-day acclimatization period during which the practice time is limited. American College of Sports Medicine published expert roundtable recommendations, "Youth Football: Heat Stress and Injury Risk," which expounded on these risks of youth football.
- Given the risk of heat-related illness with sickle cell disease, role of screening for all African-American athletes should be considered and counseling and modification of training regimens at altitude occur as needed.
- **Renal injuries:** A retrospective review of professional football players demonstrated 52 cases between 1986 and 2004, representing an average of 2.7 cases of renal injuries per season. Kidney injury rate was 10 times greater during games (0.55 per 10,000 exposure) than during practice (0.05 per 10,000 exposure), with the most common injury being renal contusion, followed by renal laceration and renal stones. In fact, 49 of 52 injuries were related to contact, and none required surgery. Renal laceration and renal contusion was associated with mean time loss of 59.8 and 15.1 days, respectively, and all athletes returned to play.

### Strength and Conditioning

- Important aspect at all levels of football
- Stress maintenance of good flexibility program in conjunction with core and overall strengthening
- Sport specificity in training; endurance work as baseline
- Interval training, stadium stair running, and hill training for explosive speed
- Supervision and appropriate technique in strengthening program, particularly if free weights are used

### Infections

- General guideline is to avoid exercise during acute infection; avoid participation if fever is >100° F. Avoid common-source water outbreaks. To avoid spread of infection, athletes should not share water bottles.
- **Myocarditis** reported as cause of SCD in athletes
- **Skin infections** (herpes gladiatorum, ringworm) often spread from person-to-person contact. Antiviral, antifungal, and antibacterial treatment necessary with protective covering to avoid transmission. Risk of methicillin-resistant *Staphylococcus aureus* (MRSA) of significant concern because of number of athletes participating. Important to avoid sharing towels, razors, toothbrushes, as well as ensuring appropriate hygiene. Rigorous cleaning of shared athletic training facilities and daily skin checks by athletic medicine staff to evaluate any suspicious skin lesions.
- **Infectious mononucleosis** is additional consideration for splenic rupture. Caused by Epstein–Barr virus (EBV). No activity for initial 3–4 weeks of symptoms, then dependent on athlete's clinical condition and presence or absence of splenomegaly, which is present acutely in a majority of cases of EBV. Abdominal ultrasound useful for accurate assessment but not generally useful unless serial assessments are performed. Position stand regarding mononucleosis in the athlete was written for the American Medical Society for Sports Medicine (AMSSM) and published in 2008. Recent systematic review of 81 cases of splenic rupture due to mononucleosis between 1984 and 2014 found that 84% occurred within 4 weeks of symptom onset, but cases reported up to 8 weeks

- **Human immunodeficiency virus (HIV)**
  - No reason to disallow competition. Study in professional football players found 3.7 bleeding injuries per game for each team involving 3.5 players and found that 88% of bleeding injuries were abrasions, with remainder lacerations. Risk of HIV transmission estimated to be <1 per 85 million game contacts.
  - Universal precautions should be used for all body fluids. More important to consider activities **off** field than on field regarding risk factors.
  - A more substantial risk of **hepatitis**; same precautions apply; hepatitis vaccination series recommended

## HEAD INJURIES

### Statistics

- The CDC estimates 2.6 million children aged <19 years, between 2001 and 2009 treated for sports-related injuries, and 6.5% or 173,285 of these were traumatic brain injuries (TBIs).
- Concussion, the most common type of TBI, is also the most common head injury occurring in sport, accounting for 8.9% and 5.8% of injuries at the high school and college level, respectively (see Chapter 45, Head Injuries).
- Team Physician Consensus Statement, AMSSM Statement, American Academy of Neurology Statement and National Athletic Trainers Association statements have all been updated; represent most recent consensus documents to address mild traumatic head injury (concussion) in athletes. Upcoming Fifth International Consensus Conference in Berlin Germany will provide additional update on evolving literature.

### NCAA Data on Head and Neck Injury

- Football has a high rate of head injury of sports monitored at the collegiate level (Table 66.2).
- Injury rates in sports with no head protection, such as men's soccer, women's soccer, and field hockey, are comparable to helmeted sports of ice hockey, football, and men's lacrosse (see Table 66.2). Helmets do not prevent concussion.

**TABLE 66.2 NCAA DATA ON CONCUSSION INJURIES: 2009-2010 THROUGH 2013-2014**

| Concussion* | | | |
|---|---|---|---|
| | **Game** | **Practice** | **Overall** |
| **Sports With No Head Protection** | | | |
| Field hockey | 11.10 | 1.77 | 4.02 |
| Women's lacrosse | 13.08 | 3.30 | 5.21 |
| Men's soccer | 9.69 | 1.75 | 3.44 |
| Women's soccer | 19.38 | 2.14 | 6.31 |
| Women's basketball | 10.92 | 4.43 | 5.95 |
| Men's basketball | 5.6 | 3.42 | 3.89 |
| Wrestling | 55.46 | 5.68 | 10.92 |
| **Sports With Head Protection** | | | |
| Men's ice hockey | 24.89 | 2.51 | 7.91 |
| Football | 30.07 | 4.20 | 6.71 |
| Women's ice hockey | 20.10 | 3.00 | 7.50 |
| Men's lacrosse | 9.31 | 1.95 | 3.18 |
| Women's softball | 5.61 | 1.75 | 3.28 |
| Baseball | 1.20 | 0.72 | 0.90 |

*Concussion as a subset of all head injuries; rates are per 10,000 athlete-exposures

From Zuckerman SL, Kerr ZY, Yengo-Kahn A, et al. Epidemiology of Sports-Related Concussion in NCAA Athletes from 2009-2010 to 2013-2014: Incidence, Recurrence and Mechanisms. *Am J Sports Med.* 2016;43(11):2654-2662.

- Player contact is primary injury mechanism in football.
- In football, spearing technique is associated with significant cervical injuries and head-to-head contact is associated with a significant risk of head injury. Rules to prevent these mechanisms of injury exist, although enforcement is often difficult; point of emphasis for officials in 2008 to call head-to-head contact fouls.
- Efforts to decrease head and neck injuries in football have come from the NCAA (Safety in in College Football Summit, 2014) as well as USA Football (HeadsUpFootball) and include having a concussion plan in place, decreasing contact exposures, education regarding appropriate tackling techniques, and signs and symptoms of head injury.

## CERVICAL INJURIES

Neck pain, radiating arm pain, paresthesias, weakness, and loss of cervical motion are criteria for removal from the game and further work-up.

### Myofascial Sprains

- Muscular or ligamentous injury to neck (most common form of neck injury)
- Presents with paravertebral spasm, decreased range of motion, usually without radicular or neurologic symptoms
- Radiographs to rule out fracture or ligamentous instability
- Treatment includes nonsteroidal anti-inflammatory drugs (NSAIDs), rest, and physical therapy.
- Return to sport when full active range of motion with little or no pain, absence of neurologic findings, and negative work-up for more severe injuries.

### Brachial Plexus Injuries ("Burner" or "Stinger")

- Usually occur with compression of vertebral foramina or distraction of neck and shoulder, causing pinching or stretching of brachial plexus
- Initial symptoms include one of knife-like searing pain radiating down from neck into arm. Typically, there is transient numbness or paralysis in the involved upper extremity; beware of bilateral symptoms, which may represent transient quadriparesis or spinal cord involvement.
- With first occurrence, consider cervical spine films to evaluate for congenital anomalies or spinal stenosis.
- Treatment includes NSAIDs, rest, and physical therapy.
- Return to sport with full clinical strength and resolution of neurologic symptoms, particularly in vertebrae C5 or C6 nerve distribution, on sidelines, check supraspinatus strength as this is often the last area of strength to return. Use protective equipment, including neck roll or a collar, which can help prevent some of the extension of the neck. These must be well fitted to tightly secured shoulder pads. Loose shoulder pads eliminate beneficial effect of neck roll. Be aware of delayed weakness. The athlete should be re-examined 24 hours later.

### Fractures and Dislocations

- Less frequent after installation of rules banning "spearing" and use of top of helmet to strike another player; injury still occurs with inadvertent hitting of crown of helmet on another player as players aggressively move to ball after initial contact is made.
- If neurologic injury present or fracture or dislocation suspected, spine must be immobilized until appropriately assessed, including radiographs
- Never remove helmet or shoulder pads until the spine is appropriately immobilized and protected; cut or remove face

guard for airway management when transporting the athlete. If removal of shoulder pads and helmet are necessary, remove both at same time to maintain neutral alignment of cervical vertebrae.
- Be aware of emergency personnel guidelines that may advocate helmet removal—protect your players! Pediatric head size is relatively larger than the shoulders—take this into consideration with young athletes.

### Return-to-Play Criteria After Cervical Injury

#### Absolute Contraindications to Return to Collision Sports

- **Congenital:** Odontoid hypoplasia, atlanto-occipital fusion, Klippel–Feil anomaly with occipital–cervical involvement or mass fusion
- **Developmental**
  - Spinal stenosis with episode of cervical cord neurapraxia and ligamentous instability or cord edema by magnetic resonance imaging (MRI) or more than one recurrence
  - "Spear tackler's spine" (stenosis with persistent straightening of cervical spine, often rigid in flexion and extension)
- **Traumatic conditions**
  - Any C1–C2 injury with ligamentous laxity or C1–C2 fusion
  - Unstable ligamentous injuries of C2–C7
  - Vertebral body fractures with sagittal component of displacement into the canal
  - Healed fractures with residual instability, neurologic finding, limitation of motion
  - Disk herniation with neurologic findings or limited motion
  - Status postfusion of more than three levels

#### Relative Contraindications

- **Developmental:** Episode of central cord neurapraxia (with resolution) and vertebral body-to-canal ratio of ≤0.8
- **Traumatic:** Full pain-free range of motion and neurologically intact with healed displaced body compression fracture or neural ring fracture; two- or three-level fusion

#### No Contraindication

- **Congenital:** Klippel–Feil anomaly with stable one- to two-level fusion below C2 and full range of motion
- **Developmental:** Stenosis ratio of ≤0.8 in otherwise asymptomatic patients
- **Traumatic:** Full, pain-free range of motion and neurologically intact with healed stable compression fracture or healed spinous process fracture; solid one-level fusion after disk excision at ≤C3
- Study of collegiate football players found that players with a Torg ratio <0.8 had three times the risk of incurring stingers.

## UPPER EXTREMITY

### Shoulder Girdle (Clavicle Fractures)

- Mechanism or injury: direct blow (most common) or impact onto point of shoulder
- Faster healing and return to sport with surgical fixation; however, conservative treatment still usually successful but with longer healing time and higher refracture rate. Surgery recommended if overlapping fragments of 1.5–2 cm
- Return to sport with resumption of strength and healed fracture

### Acromioclavicular Joint

- **Incomplete acromioclavicular separations** (coracoclavicular ligaments intact, grade I and II acromioclavicular sprains) (see Chapter 49, Shoulder Injuries)
  - Acromioclavicular injuries most commonly result from fall onto point of shoulder (Fig. 66.1).

Injury to acromioclavicular joint with direct mechanism of fall on to top of shoulder causing sprain of acromioclavicular ligaments.

Anesthetic injection during game for grade I acromioclavicular joint sprain with proper sterile technique.

**Figure 66.1.** Acromioclavicular injury.

  - Diagnosed by tenderness directly over acromioclavicular joint, pain with resisted adduction of arm across the chest; radiographs with weights sometimes helpful in determining severity between types III and V
  - Conservative treatment with rest, ice, compression, elevation (RICE), physical therapy; return to sport with acromioclavicular padding after restoration of strength. Injections with a local anesthetic may be used judiciously for return to sport with injury during game or to enable play in games with a sprain resolving in the semiacute phase (see Fig. 66.1).
- Complete acromioclavicular separations (grade III–VI acromioclavicular sprains)
  - Diagnosed by palpable step-off between clavicle and acromion; can also confirm with radiographs
  - Treatment controversial for grade III; mostly conservative unless extensive muscle stripping or penetration of the distal clavicle through trapezius is present
  - Strength and endurance comparable in surgical versus nonsurgical results for grade III injuries
  - Return to sport after restoration of strength, motion, and with acromioclavicular padding
  - Grades IV, V, and VI require surgical treatment

### Glenohumeral Instability

- Common football injury, typically seen as either anterior or posterior pattern

- Usually traumatic in nature, although multiligamentous laxity can contribute to the instability pattern

### Anterior

**Description:** Most common secondary to abduction–external rotation force; sometimes results from inappropriate tackling technique. Acute injury should be treated with gentle reduction, which can be performed on field by experienced physician; immobilization following acute injury is controversial. Early rehabilitation is important, and bracing can control abduction–external rotation moments (the position at risk) and allow early return to sport (Fig. 66.2). Immobilization and rehabilitation, however, have not been shown to affect the high re-dislocation rate in young people.

**Diagnosis:** Radiographs including apical oblique to evaluate for reduction, Hill–Sachs, or bony Bankart lesion; MRI recommended to evaluate extent of labral injury and any associated injuries

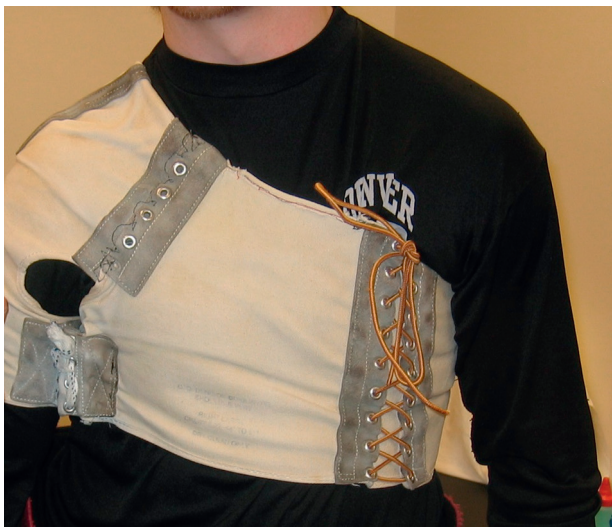
**Treatment:** Arthroscopy or acute stabilization in young people shown to significantly decrease the dislocation rate; decision making should be tailored to each case depending on risk factors, career goals, and timing. Surgical stabilization of choice is surgeon dependent; trend is for arthroscopic stabilization in the football player, although open stabilization has demonstrated less recurrence of instability in contact athletes.

### Posterior

**Description:** Instability/labral tears are common in offensive lineman; however, traumatic posterior dislocation is uncommon.

**Diagnosis** of posterior instability/labral tear often made with history of pain in posterior shoulder when arms are extended forward and are presented with a posterior directed force, thus causing pain and/or shifting of shoulder posteriorly. Examples include an offensive lineman jamming an opponent to block or an athlete performing a bench-press lift. On examination, a posterior jerk test will often reproduce the symptoms. In a traumatic posterior dislocation, examination features an anterior prominent coracoid, and the arm is adducted and internally rotated with minimal external rotation. Adequate radiographs are crucial to avoid missed diagnosis.

**Treatment:** Should consist of rehabilitation; however, no bracing can control posterior directed force on shoulder and if player remains symptomatic then surgical posterior stabilization is the treatment of choice. Typically, there is a labral tear, and players have been shown to have a successful return to football after surgery.



Example of shoulder harness to help assist player with anterior shoulder instability in preventing abduction and external rotation during competition.

**Figure 66.2.** Anterior shoulder instability.

### Rotator Cuff/Biceps

- Uncommon in younger population but may occur in quarterbacks; important to rule out subtle instability presenting as impingement syndrome
- Evaluate for supraspinatus weakness by testing resisted abduction with arm in 30 degrees of forward flexion from 90 degrees of straight abduction, with internal rotation; impingement test by forward flexing arm in internal rotation
- MRI if tear is suspected; MRI of suspected biceps rupture helpful to rule out large intra-articular tendon fragment
- Impingement syndrome with rotator cuff tendinopathy usually responsive to structured therapy program; surgical repair is usually indicated if full-thickness tear in young population.
- Biceps tendon tendinitis and proximal rupture are usually responsive to rest followed by strengthening program; surgical tenodesis indicated for select high-demand positions, to include arthroscopic debridement of symptomatic intra-articular tendon fragment; distal biceps rupture treated by surgical re-attachment

### Contusions/Exostosis

- Common, particularly in linebackers; occurs from direct blow to shoulder, resulting in injury similar to hip pointer; presentation will be weakness in deltoid and rotator cuff region; often will present with examination similar to that of someone with rotator cuff tear
- Periosteal stripping in region of deltoid insertion may lead to "dotted veil" appearance on radiograph and eventual formation of mature exostosis.
- Initial treatment for contusions similar to treatment for quadriceps contusion—ice, rest, aspiration of hematoma if present, avoidance of heat, and other treatment modalities that may exacerbate bleeding and subsequent hematoma formation

## ELBOW AND FOREARM

### Valgus Injuries (Overuse)

- Occurs occasionally in quarterbacks; diagnosed by pain and tenderness distal and posterior to medial humeral condyle with valgus stress and occasional ulnar nerve symptoms. MRI is becoming the study of choice to verify diagnosis.
- Conservative treatment includes rehabilitation, protective function; may require 4–6 months to return to sport.
- Surgery if patient failed adequate rehabilitation and desires to continue as high-demand thrower; arthroscopy to remove loose bodies and examine degree of medial laxity, followed by ulnar collateral ligament if needed

### Hyperextension/Dislocation Injuries

- Occurs with fall onto outstretched hand, often with forced extension during tackling
- Dislocations can be reduced with gentle traction with elbow in semiflexed position (for posterior dislocations—most common).
- Both hyperextension and dislocation injuries require radiographs to rule out fracture and neurovascular assessment of hand and forearm.
- Treat both injuries with early protected range of motion; return to sport with protected bracing after strength and motion restored (usually 3–6 weeks for dislocation, often sooner with hyperextension injury) (Fig. 66.3).

Hinged elbow brace utilized to protect player's elbow that has valgus laxity after elbow dislocation.

**Figure 66.3.** Valgus laxity.

## Olecranon Bursitis

- Traumatic bursitis common secondary to impact with tackling
- Examination of skin important to rule out open communication with bursa; radiograph to evaluate for bony spurs
- Aspiration for persistent or large collection; consider a sclerosing agent or excision with recurrence.
- Return to sport with protective pad

# WRIST AND HAND

## Wrist Sprains and Fractures

- Injuries of wrist must be evaluated carefully for location of tenderness and pain with movement; fractures and ligament injuries must be ruled out clinically and radiographically before diagnosis of wrist sprain.

### Scaphoid Fractures

- Acute nondisplaced fracture can be treated with long arm thumb spica cast for 4–6 weeks, followed by short arm thumb spica until healed. However, the current trend is to treat these fractures by intraosseous fixation in high-demand athletes who cannot afford prolonged healing period so that early return to sport is possible.
- **Return to sport** depends on position; for example, linemen can often return quickly with appropriate outer cast padding.
- **Displaced** or symptomatic nonunion needs open fixation, often with bone grafting; return to sport with protective splinting after adequate healing and strength restoration

### Distal Radius and Ulna Fractures

- **Conservative versus** surgical **treatment** depends on fracture pattern and stability.
- **Return to sport** depends on fracture stability and player position; may require 2–3 months before sufficient healing and strength.

### Carpal Fractures Other Than Scaphoid

- Avulsion fractures are most common; these can often be treated with immobilization, and early return to sport if position allows
- Important to rule out carpal dislocations (e.g., lunate/perilunate) or ligamentous injuries (e.g., scapholunate) with appropriate stress radiographs; MRI if plain films are inconclusive

## Injuries to Thumb

### Collateral Ligament Injuries

- Common injuries result from adduction (radial) or abduction (ulnar ligament) forces applied to metacarpophalangeal (MCP) joint. Diagnosis by reproduction of pain with stress testing and tenderness to palpation over radial or ulnar aspect of the MCP joint
- Radial ligament injuries and incomplete ulnar ligament injuries can be treated with protective splinting; early return to sport in most positions.
- Complete tear of ulnar collateral ligament (as confirmed by stress radiography) most commonly treated by open ligament repair because of high prevalence of Stener lesions; could be treated with casting and early return to sport after repair.

### Fractures and Dislocations of Thumb

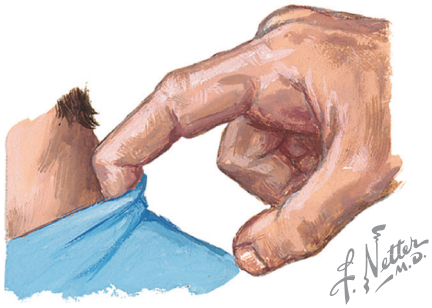
- Fractures and dislocations at MCP joint are also common because of precarious position of thumb.
- MCP dislocations are most commonly dorsal; important to differentiate simple (reducible) from complex (irreducible, requiring open reduction secondary to interposition of volar plate); can return to sport early with protective splint for 4–6 weeks
- Intra-articular fractures (Bennett's) are usually unstable and require fixation. Phalanx fractures, if stable, can be treated with protective splinting and early return to sport.

## Hand Fractures/Dislocations Excluding Thumb

- Occur frequently from direct impact; diagnosis confirmed by radiography
- Fractures of fourth and fifth metacarpals can usually be manually reduced to an acceptable position and treated with protective splinting. Less tolerance for displacement with second and third metacarpals; require surgical fixation more frequently
- Proximal phalanx fractures often require surgical fixation to maintain length, appropriate rotation.
- Middle phalanx fractures more commonly amenable to closed reduction, buddy taping.
- Distal tuft fractures should be treated as soft tissue injuries; if suspected, examination of nail bed and meticulous repair if damaged or removal from fracture site if incarcerated; time-honored hot paper-clip or electrocautery relieves painful subungual hematomas.
- Most hand fractures, if stable, can be treated with protective splinting, early return to sport.
- Dislocations are most commonly dorsal and can be treated by closed reduction, continuous buddy taping for 3–6 weeks, early return to sport
  - Radiographs after game important to rule out bony injury
  - If dislocation of proximal interphalangeal (PIP) joint is volar (rare), must treat it in same manner as rupture of central slip of an extensor tendon

## Tendon/Capsular Injuries

- Flexor tendon injuries usually occur with attempts to grab another player's jersey, resulting in pain and inability to flex distal interphalangeal (DIP) joint of involved finger (Fig. 66.4).



**"Jersey Finger,"** example of injury to flexor tendon of hand while player trying to tackle opponent and finger getting stuck in jersey.

**Figure 66.4.** Jersey finger.

  - Important to determine level of retraction of tendon; if it retracts into palm, blood supply is significantly compromised, and repair should be performed as soon as possible (within 7 days); less retracted tendon injuries can be repaired later (blood supply from vincula maintained, but best results with early diagnosis and repair).
  - Always obtain radiographs because bony avulsion injuries, which usually limit amount of retraction of tendon, can occur. Early repair of bony injury also yields best result.
- Extensor tendon avulsions of distal phalanx (mallet finger) usually occur by "jamming" finger, causing forced flexion of DIP joint.
  - Examination reveals inability to extend DIP joint.
  - Always obtain radiographs to determine if bony avulsion has occurred; subluxation of DIP joint (usually occurs with fragment involving more than one-third of the joint surface) requires more aggressive treatment.
  - Mallet fingers should be treated with continuous extension of DIP joint with splint, free range of motion of PIP joint, and early return to sport.
- Extensor tendon avulsions of central slip (insertion into proximal aspect of middle phalanx) also occur by "jamming" finger and are often misdiagnosed as capsular sprain.
  - With other fingers held in extension and MCP joint flexed on involved finger, test for ability to extend PIP joint. If ability is absent, suspect central slip rupture.
  - Radiographs important to rule out fracture
  - Treat with PIP joint held in continuous extension for at least 6 weeks with DIP joint free. Failure to recognize and treat these injuries results in boutonnière deformity.
- Capsular sprains and lateral collateral ligament injuries can be treated by continuous buddy taping for 6 weeks and return to sport.

## CHEST AND ABDOMEN

### Rib Fractures

- Due to direct blow to ribs with resultant pain, particularly when breathing
- Radiographs important to rule out presence of pneumothorax (collapsed lung)
- Return to sport with protective "flak jacket" when symptoms are tolerable

### Sternoclavicular Joint

- Dislocations or injuries caused either indirectly by blow to shoulder or directly by injury to upper chest
- Diagnosed by pain and swelling at sternoclavicular region with exacerbation during arm range of motion
- Computed tomography (CT) scan is important to rule out posterior dislocation, which requires emergent surgical reduction to avoid injury to major vessels.
- Anterior dislocations most common and usually treated conservatively with return to sport when strength and motion restored

### Pectoralis Major Ruptures

- May occur with forceful extension/external rotation of arm; pain and ecchymosis most commonly along anteromedial aspect of arm (most ruptures occur near insertion onto humerus) and inability to flex pectoralis with adduction and internal rotation
- Suspected incomplete and musculotendinous injuries can be treated with rest and ice, with return to sport upon restoration of motion and strength
- Complete ruptures are usually treated surgically because of significant loss of strength if treated conservatively.

### Rectus Abdominis Strain

- Forceful resisted contraction of abdominal musculature can lead to intramuscular strain or avulsion from iliac crest or pubis; diagnosed by area of tenderness and pain with attempted flexion of trunk
- Treatment is usually conservative, with ice, stretching, and taping of trunk to limit flexion and rotation on return to sport.

### Athlete's Groin (Sports Hernia)

- Often confused with osteitis pubis; diagnosis can be difficult
- Radiographs necessary to rule out symphysis pubis changes; ultrasound can be helpful. Conservative management often prolonged and requires extensive physical therapy efforts for mobilization of all muscles attaching to anterior pelvic rim; surgical treatment has good results.

## THORACOLUMBAR SPINE

### Thoracic or Low Back Strain

- Most common injury to spine in contact sports; muscle spasm or ligamentous strain usually occurs with bending or twisting during forceful activities
- Often secondary to fatigue or inadequate conditioning of the paraspinal muscles
- Most commonly occurs without radicular symptoms; suspect disc pathology if radiation of pain into lower extremities
- Plain anteroposterior, lateral, and oblique radiographs helpful to rule out any osseous injury, neural arch involvement, or Scheuermann's kyphosis
- Treatment consists of control of initial inflammatory phase with ice and rest; progression to back rehabilitation program when tolerated
  - Most uncomplicated sprains respond in 7–10 days.
  - Flexibility and conditioning program during season and in off-season important to prevent recurrence

### Herniated Nucleus Pulposus

- Injury pattern often similar to that of strain, but with radiculopathy into one or both lower extremities, weakness, with or without diminished reflex; straight leg raising often abnormal; often with complaints of paresthesias and pain in leg, which are more bothersome than back complaints
- Plain films of lumbar spine and MRI often help document injury.

- Initial treatment is same as for lumbar strain. Consider epidural steroids or disc excision if no improvement with conservative care or with progressive neurologic insult.

## Spondylolysis/Spondylolisthesis

**Description:** Common in linemen secondary to repetitive loading on posterior elements in extended position

**Diagnosis:** Complaints similar to low back strain, but hamstring spasm and pain with resisted extension are also common; single-legged back extension is painful. Previous history of spondylolisthesis or significant back pain important to determine chronicity; anteroposterior, lateral, and oblique **radiographs** necessary to evaluate for "broken neck on Scotty dog" to confirm spondylolysis and to evaluate for presence of translation of vertebral body (spondylolisthesis); bone scan is often helpful if radiographs are equivocal or to differentiate acute neural arch injury from low back strain with previously healed spondylolysis.

**Treatment:** Consists of restriction from play, abdominal and lumbar stretching, strengthening activities, and occasional use of lumbar orthosis. Resolution of hamstring spasm is often a reasonable gauge to begin functional progression in rehabilitation as tolerated. Clinical progress more reliable indicator than radiographs, which often do not demonstrate bony healing even when patient becomes symptom free; known presence of spondylolysis or spondylolisthesis in asymptomatic player is not a contraindication to play; symptomatic, skeletally immature patients with high-grade spondylolisthesis (slippage greater than half width of vertebral body) are candidates for fusion.

## Thoracolumbar Fractures

**Description:** Infrequent injuries that can occur with severe direct impact to spine or violent muscle contractions

**Diagnosis:** If fracture is suspected, important to maintain spine precautions before any movement of patient; neurologic assessment important in initial examination; patients generally complain of severe back pain, exacerbated with any attempted movement. **Radiographs** of spine, including anteroposterior, lateral, and oblique views to evaluate injury; MRI if any neurological injury is present with fracture; consider CT scan or MRI of abdomen if visceral injuries (rare) are suspected.

**Treatment:** Depends on fracture pattern and stability; isolated stable transverse process or spinous process fractures usually amenable to brief period of rest with return to sport when symptoms tolerate (3–6 weeks). Fracture treatment should follow accepted guidelines for particular fracture pattern (e.g., patients requiring lumbar fusion should not be allowed to return to contact sports). Acute fractures with no history of prodromal symptoms can often heal if patient is adequately immobilized with lumbar orthosis and pantaloon leg extension.

# HIP AND PELVIS

## Soft Tissue Injuries

### Hip Pointers

**Description:** Common term applied to injuries of iliac crest, including contusions from direct injury, muscle avulsions from violent contractions, and periostitis from repetitive abdominal muscular contractions or contusions

**Diagnosis:** Skeletally immature players should be radiographically evaluated to rule out displaced apophysis from avulsion-type injuries.

**Treatment:** Consists of rest, ice, stretching, and NSAIDs; most apophyseal injuries and muscle avulsions treated conservatively except for widely displaced apophyseal fractures and large muscle avulsions, which should be surgically repaired

**Return to sport:** When symptoms tolerate with padding, heat, and stretching before play and ice afterward; can consider injection with local anesthetic during game or pregame to mitigate symptoms from contusion over the iliac crest

**Trochanteric bursitis**: From direct blow also common injury in this region; treatment involves same protocol with judicious use of steroid injections, if necessary

### Groin Pull

**Description:** Common term for injury to iliopsoas or adductor group of leg, usually caused by forced abduction and external rotation; common in the deconditioned athletes

**Diagnosis:** Symptoms include pain with resisted adduction of leg or passive abduction, located along border of pubic ramus. Radiographs of hip and pelvis to rule out avulsion fracture caused by iliopsoas (lesser trochanter) or adductor group

**Treatment:** Initially includes ice and rest, followed by stretching and strengthening when symptoms allow; best treatment is preventive by means of a preseason strengthening program. Symptoms can be persistent depending on severity; adequate strength necessary before play to prevent exacerbations; most avulsion injuries treated similarly, if not widely displaced

### Osteitis Pubis

**Description**: Common in football players and weightlifters; exact cause unknown, but thought to be secondary to overuse of adductors and gracilis with gradual resorption of bone at muscular attachment sites (medial aspect of pubic bones)

**Diagnosis:** Symptoms include gradual onset of groin pain exacerbated by resisted adduction and tenderness along inferior and medial borders of pubic bones. Radiographs may reveal resorption of bone at pubic symphysis with resultant widening and sclerosis of inferior pubic rami. Bone scans reveal diffuse uptake in involved areas of the pubic rami.

**Treatment:** Relative rest and anti-inflammatory medications are most successful for this overuse injury, which often takes 2–3 months to resolve. Sometimes, a corticosteroid injection may be helpful in refractory cases.

## Fractures/Dislocations

### Hip Subluxation/Dislocation

**Description:** Unusual but potentially devastating injury secondary to possible osteonecrosis of femoral head; player experiences severe pain in hip with any attempted movement; posterior dislocation most common; leg appears shortened and internally rotated

**Diagnosis:** Radiographs before reduction necessary to rule out femoral neck fracture

**Treatment:** Gentle reduction should be performed urgently in controlled setting after neurovascular status and radiographs are assessed.

### Lesser Trochanter Avulsion/Other Apophyseal Avulsions

**Description:** Most common under age 20 and results from severe contraction of iliopsoas muscle (or other muscular attachment); avulsion fractures in mature adults (aged ≥20 years) are often associated with neoplastic disease

**Diagnosis:** Symptoms include immediate severe groin pain exacerbated by attempts to flex the hip joint or pain and tenderness to palpation in the suspected apophyseal location with exacerbation on resisted muscular contraction.

**Treatment:** Involves bed rest followed by gentle range of motion and partial weight-bearing when tolerated when radiographs reveal nondisplaced or minimally displaced fractures; role of open reduction (based on degree of displacement) is controversial.

# LOWER EXTREMITY

## Thigh

### Quadriceps Contusions/Exostosis

**Description:** Direct impact onto quadriceps muscle belly with resultant pain, decreased range of motion, and occasional palpable mass

**Treatment:** Initial treatment of flexion, ice to minimize hematoma (not heat), followed by pain-free range of motion and functional rehabilitation (avoid heat, ultrasound, painful exercise); immobilization in flexion overnight to minimize bleeding is often helpful. Conservative treatment for myositis ossificans unless functional impairment persists for >6 months

**Return to sport:** With extra thigh padding when full strength, function, and range of motion are achieved

### Hamstring Injuries

**Description:** Sudden pain in posterior thigh with rapid hamstring contraction; associated with fatigue, poor conditioning, and inadequate stretching; short head of biceps is most common; prophylaxis with off-season conditioning, baseline hamstrings-to-quadriceps strength ratio of ≥0.6; Nordic hamstring exercises can be very helpful.

**Treatment:** Initial treatment includes RICE, jogging, and pain-free functional rehabilitation when hamstrings 70% of baseline. If area of hamstring strain is focal, then local corticosteroid injection can be considered and is supported by the literature.

**Return to sport**: When isokinetic strength ratio is approximately 0.6

## Knee and Leg

### Ligament Injuries

#### MEDIAL COLLATERAL LIGAMENT

**Description:** Most commonly caused by direct impact onto lateral aspect of knee with applied valgus stress

**Diagnosis:** Pain on medial aspect of knee with tenderness to palpation along course of medial collateral ligament: increased laxity to valgus stress at 30 degrees of flexion; increased laxity when knee at full extension indicates more severe knee injury (often cruciate and/or posteromedial capsular disruption)

**Treatment**: Usually treated nonsurgically with protected weight-bearing, inflammation control, range of motion 0–30 degrees, with full weight-bearing as tolerated; progression to full range of motion as symptoms allow; addition of progressive resisted exercises, and return to sport with functional knee brace as injury allows; complete or grade III injuries often require 4–6 weeks for return to full activity with protective brace.

#### ANTERIOR CRUCIATE LIGAMENT

**Description:** Most commonly results from noncontact twisting injury, but can result from direct blow to knee as well

**Diagnosis:** History of hearing or feeling "pop" in knee, rapid effusion (within few hours) highly suggestive of ACL disruption; examination reveals positive Lachman test, pivot shift. Radiographs usually negative or may show small proximal lateral tibial plateau bony avulsion (Segond's sign)

**Treatment:** for cruciate-dependent patients or those who plan to continue to participate in high-risk sports is surgical in most instances; delaying surgery until knee range of motion is from full extension to >110 degrees of flexion is currently believed to reduce incidence of arthrofibrosis. Concomitant injuries most commonly treated by conservative medial collateral ligament management with delayed ACL reconstruction; preferred graft choice for ACL in NCAA and professional football orthopedic team physicians is bone–patellar–bone autograft; functional knee bracing on return to sport remains controversial.

#### POSTERIOR CRUCIATE LIGAMENT

**Description:** Mechanism of injury most commonly hyperflexed knee or direct blow to anterior tibia while foot is firmly planted

**Diagnosis:** Injury is often missed, and a high index of suspicion is necessary. Examination reveals increased posterior tibial translation at 90 degrees of flexion. Suspect more severe injury if posterior sag does not improve with internal rotation of the tibia. Radiographs are often negative or may reveal bony avulsion at tibial insertion.

**Treatment:** For isolated posterior cruciate ligament is controversial; conservative approach most common, with focus on functional restoration before play; surgical proponents recommend reconstruction because of high incidence of patellofemoral and medial arthritis with this injury, although current studies fail to show improved outcomes with surgical intervention

#### POSTEROLATERAL INSTABILITY

**Description:** Isolated injury to this complex uncommon; usually accompanied by cruciate ligament injury; direct blow to anteromedial tibia or severe twisting with foot planted is the most common mechanism.

**Diagnosis:** Examination shows increased laxity at 30 degrees with varus stress and increased external rotation (Dial test); effusion often absent with combined cruciate and posterolateral injuries secondary to capsular tear; important to assess peroneal nerve function at time of injury; radiographs often negative but necessary to rule out avulsion fractures; MRI helps delineate structures injured. Beware of fibular avulsion of lateral hamstring.

**Treatment:** Involves direct surgical repair/reconstruction of posterolateral structures (or biceps tenodesis if tissues severely compromised) and reconstruction of cruciate injury, if present

### Meniscal Injuries

**Description:** Common injuries that can occur by contact or noncontact mechanisms; often twisting-type injury with joint line and/or posterior pain and complaints of locking, catching, or buckling; unless tear is peripheral, effusion is usually not noticed until after initial 24 hours.

**Diagnosis:** Examination often reveals joint line tenderness, effusion, pain with extremes of flexion, and lack of full extension (or "locked" knee) if displaced bucket-handle is present. Nonspecific pain may be present with circumduction maneuvers. radiographs are negative; MRI is the diagnostic test of choice when examination is equivocal.

**Treatment:** Arthroscopy is indicated when symptoms do not respond to rest and control of inflammation; repair instead of excision of meniscus should be attempted whenever feasible.

### Patellofemoral Disorders

#### PATELLAR DISLOCATION

**Description:** Usually noncontact, external rotation injury to leg resulting in severe anterior knee pain with buckling or an inability to bear weight

**Diagnosis:** Knee diffusely swollen with significant tenderness along medial retinaculum, lateral trochlea, and patellar articular surfaces; apprehension with attempts at patellar translation (usually reduces spontaneously); radiographs, including Merchant view, necessary to rule out patellar or trochlear ridge fracture or bone fragments in joint

**Treatment:** Includes RICE, with progression to range of motion and quadriceps strengthening for initial injury; return to sport usually requires 3–6 weeks; repeat dislocators often require surgery to reconstruct medial structures and alter mechanical alignment.

#### PATELLAR/QUADRICEPS TENDINITIS (JUMPER'S KNEE)

**Description:** Overuse injury common to football; secondary to frequent stops and starts and eccentric loading of extensor

mechanism; most common areas of tendinitis are patellar tendon at inferior pole of patella and quadriceps tendon insertion into superior pole, but can occur anywhere along extensor mechanism

**Treatment:** Prevention is an optimal treatment, with conditioning including eccentric strength training and adequate stretching; symptomatic players require relative rest (exercises that do not provoke symptoms) and anti-inflammatory medications, with progression to concentric and isometric strength training, and finally eccentric training when asymptomatic during other therapy; the use of platelet-rich plasma has shown to have potential benefit in recalcitrant cases.

FAT PAD SYNDROME (HOFFA'S DISEASE)

**Description:** Seen frequently secondary to contusions and resultant irritation to anterior aspect of knee

**Diagnosis:** Tenderness to palpation usually present medial and lateral to patellar tendon and inferior patella; capsule in this region often feels "boggy"

**Treatment:** Involves relative rest, anti-inflammatory medications, and stretching; sometimes corticosteroid injections can be helpful. Recalcitrant cases are uncommon but usually respond to arthroscopic debridement.

### Medial Tibial Stress Syndrome (Shin Splints)

**Description:** Characterized by diffuse pain along medial border of tibia; common in early season with increased training, exacerbated during exercise, and may persist for hours to days

**Diagnosis:** Area of tenderness usually along posteromedial border of tibia—less well localized than a stress fracture; should not be exacerbated with passive ankle or foot motion. Radiographs are usually normal but may reveal mild cortical hypertrophy; bone scan helps distinguish from stress fracture and is negative or reveals diffuse linear uptake approximately one-third the length of tibia on posteromedial border.

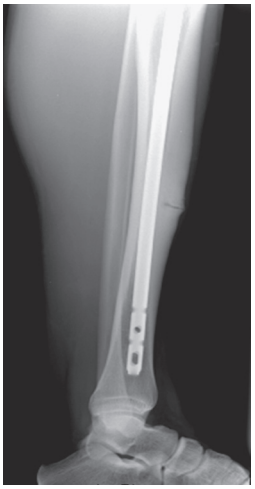
**Treatment:** Rest is most effective treatment; anti-inflammatory medications and stretching by themselves do not resolve the condition. Gradual resumption of activities when pain free (usually 2–6 weeks); orthotics may help prevent recurrence when significant pronation is present.

### Tibial Stress Fractures

**Description:** Most common mechanism is rapid increase in training, particularly running activities

**Diagnosis:** Point tenderness on anteromedial aspect of tibia is highly suggestive of diagnosis. Radiographs of stress fracture are often negative or reveal only cortical thickening; if no fracture is seen on plain radiographs, bone scan is the procedure of choice to achieve a definitive diagnosis. MRI is often used to determine severity of stress fracture and thus predict recovery time. Beware of the "dreaded black line" (Fig. 66.5).

**Treatment:** Involves rest followed by gradual **pain-free** progression of activities; one study suggests that use of pneumatic leg brace during rehabilitation protocol can shorten return-to-play time by several weeks. If fracture line and pain persists, then intramedullary tibial nailing is the procedure of choice (Fig. 66.5).



Radiograph demonstrating intramedullary nailing for recalcitrant anterior tibial stress fracture in football player.

**Figure 66.5.** Recalcitrant anterior tibial stress fracture.

## Ankle and Foot

### Ankle Sprains

**Description:** Most common ligamentous injury; structures injured depend on force and applied direction of injury; therefore, **history of injury important**

- Plantarflexion and inversion stresses injure anterior talofibular ligament (ATFL), with progressive force injuring calcaneofibular ligament (CFL) and posterior talofibular and tibiofibular ligaments in severe injuries.
- Inversion stress in a neutral position injures CFL.
- Dorsiflexion stresses tibiofibular ligaments.
- Eversion and external rotation injures deltoid ligament medially and tibiofibular ligament laterally, often accompanied by fibular fracture.

**Diagnosis: Palpation and stress tests** are used to determine the injured ligaments. Anterior drawer with 30 degrees of plantarflexion to assess ATFL; compare with contralateral side

- Inversion test: Grasp heel and apply inversion stress; ability to palpate talar head indicates complete tears of both ATFL and CFL.
- Squeeze test: Squeeze fibula and tibia together near ankle joint; pain indicates probable syndesmotic/tibiofibular ligament injuries.
- Evaluate for associated injuries, including peroneal tendon dislocation, fifth metatarsal fracture, posterior tibial tendon rupture, and midfoot sprains.

**Radiographs** are necessary to rule out fracture of medial malleolus, lateral malleolus, and talar dome. Stress radiographs are occasionally helpful in chronic ankle instability to evaluate ligament integrity—anterior drawer assesses ATFL, talar tilt test (inversion stress) with >5 degrees of asymmetry indicates CFL instability, and loss of parallelism with Broden's view and inversion stress indicates subtalar instability. External rotation stress with mortise view to evaluate syndesmosis; standing bilateral mortise view can identify syndesmosis laxity; MRI useful to evaluate talar dome osteochondral injuries when suspected

**Treatment** for acute injuries includes anti-inflammatory medications, early restoration of range of motion, and functional management dependent on severity; ankle braces to limit inversion/eversion stress used on grades II and III; cast immobilization rarely indicated in healthy athletes; proprioceptive and peroneal strengthening immediately for grade I, immediate to 1 week for grade II, when tolerated in grade III (1 week to 1 month)

**Return to sport:** Depends on athlete's ability to demonstrate adequate performance in position-specific drills with few or no symptoms (usually <1 week for grade I, 1–2 weeks for grade II, approximately 1 month for grade III); unstable fracture patterns and syndesmotic disruption require surgical stabilization

**Functional instability:** Peroneal weakness, not ligamentous instability, responsible for approximately 50% of cases of persistent functional instability; Broström lateral ligamentous repair currently most popular technique for ligamentous instability

### Midfoot Sprains

**Description:** Usually indirect injuries from forceful foot abduction when foot in fixed position or foot forced into severe plantarflexion

**Diagnosis:** Patient demonstrates pain or tenderness in midfoot region, unable to tiptoe, may have flattening of longitudinal arch. Lateral and anteroposterior weight-bearing views: in normal anteroposterior view, medial border of second metatarsal base lines up with medial border of middle cuneiform; distance between second metatarsal base and lateral border of medial cuneiform should be symmetric on comparison views.

**Treatment:** Conservative for normal radiographs; for severe medial sprain, use a short-leg nonweight-bearing cast for 4–6 weeks, followed by a weight-bearing cast for 2–3 weeks. Lateral midfoot sprains tend to resolve earlier. For Lisfranc's dislocation, internal fixation to reduce second metatarsal base to medial cuneiform (weak link)

**Return to sport:** With evidence of healing, such as ability to tiptoe and perform functional drills

### Fifth Metatarsal Base Fractures

**Description:** Three types:

- Tuberosity fractures from traction/avulsion-type injuries involving peroneus brevis or ligaments
- Metaphyseal fractures by adduction and inversion forces
- Jones fracture (approximately 3 cm distal to tuberosity) by repetitive or single force by sharp turning

**Diagnosis**: Tenderness to **palpation** over fifth metatarsal base and pain with eversion forces present with all fracture types; **radiographs** of foot determine fracture type; bone scans help rule out Jones fracture when sclerosis, but no obvious fracture present

**Treatment:** Walking boot for tuberosity and metaphyseal fractures Jones fracture: In a high-demand athlete, percutaneous intramedullary compression screw improves healing time. Screw fixation is also most common treatment when significant sclerosis present or delayed union with conservative treatment.

**Return to sport:** With radiographic evidence of union and ability to perform functional tests

### Turf Toe

**Description:** Hyperextension or, less commonly, hyperflexion injury to first metatarsophalangeal (MTP) joint, associated with flexible footwear and more common on artificial turf; severity of injury can range from capsular or ligamentous sprains, to axial compression with resultant chondral injury, or MTP dislocation

**Diagnosis:** Examination reveals tenderness about MTP joint, pain with passive dorsiflexion and often with decreased range of motion, and difficulty with push-off. **Radiographs** to evaluate for bony injury to MTP joint; may see avulsions secondary to capsular injury; rarely, may have chondrolysis with repeated injuries; if plantar tenderness is present, obtain sesamoid views to rule out fractures in these structures.

**Treatment:** Depends on severity of injury; capsular or ligamentous strains (characterized by localized tenderness) treated with ice, continuous play with taping to restrict motion; also, stiff forefoot plate is helpful to limit motion of great toe; significant swelling, ecchymosis, and restricted range of motion indicate partial or complete capsular tear: treat with anti-inflammatory medications, protective footwear, and restriction of play until clinical symptoms allow (usually 3–6 weeks). Chondral injuries or dislocation require treatment similar to lesser injuries, as well as limited weight-bearing and immobilization. Return to sport depends on clinical symptoms (often prolonged).

## PROTECTIVE EQUIPMENT

### Football Helmet

Helmets must meet National Operating Committee on Standards for Athletic Equipment (NOCSAE) specifications.

#### Construction

- Outer shell is made of polymer plastic (polycarbonate or acryl butadiene styrene) that is both lightweight and able to withstand high impacts.
- Padding or suspension systems include pads, air- and fluid-filled cells, combination of cells and pads, and suspension.
  - Air cells can be inflated or deflated to provide better fit.
  - Suspension types offer inferior protection and are being phased out.
  - Important to check for cracks in outer shell (particularly at locations of face mask attachments), leaks, or other damage to padding system to ensure appropriate protection
  - Helmet should have a NOCSAE label on outer shell warning of potential head or spine injury with inappropriate helmet size.

#### Helmet Fitting

- Helmet should be donned by spreading ear holes apart with thumbs; helmet should turn minimally with attempts to rotate on head.
  - No space between jaw pads and face or back of head
  - There should be one to two fingerbreadths above eyebrows; helmet should not come over eyes even with firm pressure on top of helmet.
  - Base of skull should be covered, but helmet should not impinge on cervical spine with full extension.
  - Jaw pads should provide snug fit and prevent lateral rocking.
  - Newer lightweight helmets are under development at present.
- Hair should be same length at time of fitting as it will be throughout season. Long hair should be wet when fitting to simulate sweaty conditions.
- Chin strap (4-point more secure than 2-point) helps prevent forward and backward helmet motion; inappropriate tension or strap failure can cause helmet to come off or result in lacerations to bridge of the nose.
- Face masks used depending on position; ball-carrying positions require mask that protects yet allows unobstructed view, whereas linemen need additional protection to prevent eye and facial injuries.
- Nontinted plexiglass shields are also worn for those seeking additional eye protection. Tinted plexiglass shields can be worn only by players requiring special assistance for elimination of sun because of a medical condition. They require physician's approval, and use is determined at time of play by officiating staff.
- Mouth guards (required in college and high school) can be custom-made, mouth formed by heating, or obtained in standard sizes. They help dissipate blows to chin, reducing intraoral and mandible injuries.

### Shoulder Pads

- Cantilever and flat are two basic types.
  - **Flat pads** are worn by positions requiring greater glenohumeral motion.
  - **Cantilever pads** (named for bridge extending across shoulder) are worn by players in constant contact and requiring additional protection.
- Modifications depend on position and preference (e.g., linebackers and other players hit in standing position have pads that are larger anteriorly and slanted forward).

### Shoulder Pad Fitting

- Measure from acromioclavicular joint to acromioclavicular joint, and compare with manufacturer's chart to check approximate fit.
- Neck opening should be large enough to prevent neck impingement when lifting arm overhead but not large enough to allow excessive sliding (1–2 fingerbreadths between the neck and inside padding).
- Lateral aspect of pad should be just lateral to shoulder, with flaps or epaulets large enough to cover deltoid region.
- Elastic axilla straps should be adjusted snugly but comfortably to allow blows to be distributed across pads.
- Anterior pads should meet but not overlap when laced up.
- Pads should extend below nipple line anteriorly and approximately 1 inch below scapula posteriorly.
- Important to inspect frequently for strap fraying or breakage, loose rivets, or cracks; also check for sweat-compressed lining pads.

## Other Protective Equipment

### Standard Equipment

- Hip pads are used to protect iliac crest and tailbone from injury.
- Thigh pads and knee pads are inserted into pants snugly and should not slip easily.

### Special Equipment

- Neck rolls and collars are used frequently by players who have previously experienced "burners and stingers" or on prophylactic basis; efficacy of these devices in preventing further injury remains debatable. Neck rolls in college are often ineffective because of inappropriate wear of shoulder pads. Shoulder pads must fit securely to the chest to stabilize collar or neck roll and help decrease frequency of "burners."
- Shoulder restraint devices restrict abduction and external rotation in chronic shoulder dislocators, but these limitations may affect player's capabilities in several positions.
- Gloves and upper extremity pads are worn by linemen and other players to prevent finger injuries, contusions, and myositis ossificans.
- Rib pads or vests are commonly worn by quarterbacks to prevent injuries to thorax.
- Value of prophylactic knee brace in preventing injury remains questionable; therefore, it is not a mandatory protection device.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# SOCCER

# 67

Chitra Kodery • Margot Putukian

## INTRODUCTION

- Soccer, or football, is the world's most popular sport.
- The popularity of soccer has steadily risen in the United States (US) with over 13 million Americans playing at all levels.

## GENERAL PRINCIPLES

### Rules of the Game

- Objective of the game: To win the game by outscoring your opponent, which is achieved by hitting the ball into the opponent's goal
- A game lasts for 90 minutes; divided into two 45-minute halves
- 11 players on the field—10 field players and one goalkeeper
- The ball cannot be played by the hands (except goalkeeper), but all other surfaces of the body can be used.
- Rules protect against rough play—there are direct and indirect fouls.
  - **Direct fouls** include kicking the opponent, tackling from behind, using hands, and pushing.
  - **Indirect fouls** include offside, obstruction, and certain unintentional fouls.
    - Offside rule: must have two players between an offensive player and the goal; this often includes the goalkeeper
- Required equipment: Ball, uniforms, and goalposts; most leagues require shin guards
- There are different methods of putting the ball back in play when it goes out of touch (out of bounds):
  - **Throw in:** when the ball goes out over the sideline
  - **Goal kick:** when the ball goes out over the endline, last touched by an offensive player; kicked in from the goal box by the defensive team
  - **Corner kick:** when the ball goes out over the endline, last touched by a defensive player; kicked in from the field corner by the offensive team

### Sport-Specific Demands and Skills

**Physical demands:** Players engage in discontinuous, high-output activity that involves aerobic and anaerobic metabolism; activity further divided into sprinting, cruising, running, and walking
- Energy expenditure requires approximately 75% $\dot{V}O_2$ max; differs with position (midfielders expend more energy than those in other positions); average heart rate: 165 beats per minute. Players typically cover 8–12 km during a game.
- Ratio of time spent in low-intensity versus high-intensity efforts is 7:1.
- Sprints occur once every 90 seconds; high-intensity efforts (cruise and sprint) once every 30 seconds; only 2% of the total distance covered involves time spent with the ball

**Physiologic demands:** Implications for thermoregulation and nutritional needs; energy needs increased by sport-specific skills

**Strength demands:** High demand on trunk and lower extremities; most skills require balancing on one leg, which emphasizes lower extremity strength and proprioception.

**Soccer specific skills:** Various surfaces of the lower extremity are used to strike or control the ball and tackle, thus putting various structures at risk.
- **Inside of foot pass/block tackle:** Foot and hip externally rotated, knee flexed, foot "locked" in dorsiflexion (Fig. 67.1); results in significant valgus stress at the knee and may be associated with traumatic injury (sprain of medial collateral ligament [MCL], fracture) or muscle strain
- **Outside of foot pass/shot:** Internal rotation of the leg, foot in inversion, plantarflexion; may place an athlete at a risk of forceful inversion plantar flexion injuries, mid/forefoot injuries, ligament sprains, and peroneal tendon problems (see Fig. 67.1); in young athletes, this may lead to apophysitis or avulsion fracture.
- **Instep of foot:** Extreme plantar flexion in "locked" foot, hip flexors, quadriceps, rectus, and hamstrings (see Fig. 67.1); ball velocity at release is between 17 and 28 meters/second.
- In the approach and ball strike phase of an instep kick, a varus torque of >200 Newton meters (Nm) and extension torque of >280 Nm is generated on the proximal tibia.
- A total of 2000 Nm is generated during a soccer kick. Only 15% is transferred to the ball; the remainder is absorbed by the eccentric contraction of the hamstrings.
- Kicking the ball on the ground can injure the forefoot, ankle, and toes (particularly the great toe).
- **Heading:** Purposeful, forceful striking of the ball with a player's head to control, clear, or redirect on goal; increases the risk of head and neck injuries because of player contact (head to head or other body part, or head to goal post or ground) that occurs while fighting for possession in the air (see Fig. 67.1)
- Appropriate **technique is important in all skills to avoid and decrease injuries.**

### Factors Affecting Performance and Recovery

#### Fatigue

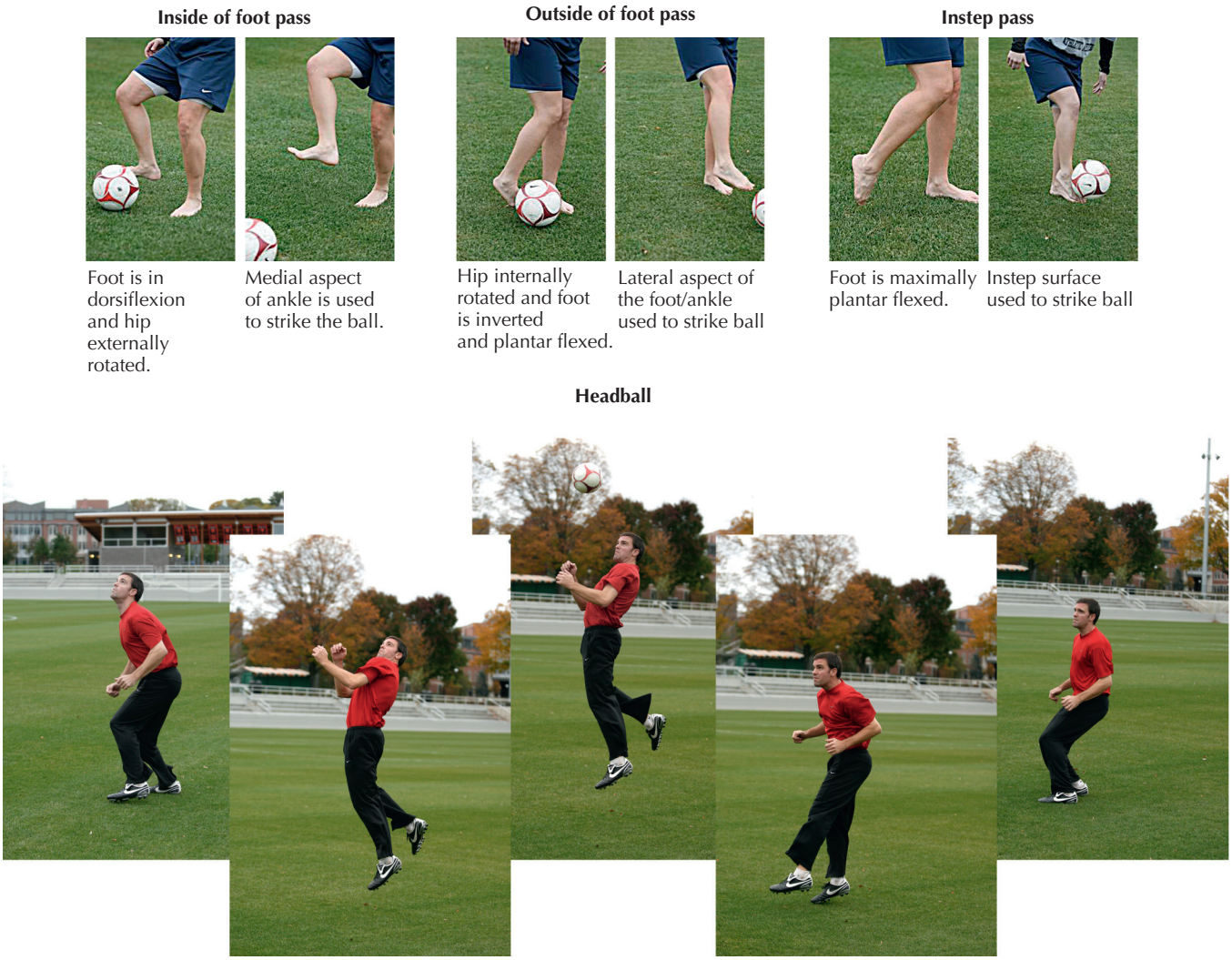
- **Fatigue** or decreased performance in soccer players is multifactorial—can be related to environmental or nutritional factors including dehydration, glycogen depletion, aerobic fitness, and mental fatigue.

#### Dehydration

- Occurs when sweat losses exceed fluid intake, which is particularly a concern in warm weather
- A decrease in performance is noted when there is fluid loss of >2% of an athlete's body weight. At >3% loss, more serious consequences can arise such as exertional heat illness.
- A study following soccer players over a 2-week training period showed that on average, athletes drank approximately 2.7 L of fluid on their own. **Voluntary intake resulted in a deficit of 1.1 L, which is not enough to meet needs.**

#### Heat Illness

- Risk factors for development of heat illness include pre-existing medical disease, age, poor conditioning, inadequate acclimatization, dehydration, obesity, fatigue, prior heat injury, febrile illness, and medications.
- Modifications should be made to decrease the risk of heat injury: more water breaks, change game times, and shorten the length of play.
- Studies have investigated the effect of hyperhydration on improving thermoregulation.




Jumping header. These photographs show the series of technique involved in the jumping header. The ball is struck when the player is at the maximal height of the jump, with the player's eyes open. Impact with the ball is made at the forehead/hairline with the head striking forward through the ball. The arms are held up for balance and to create space for the player and thus protect him from inadvertent contact with another player.

**Figure 67.1.** Soccer-specific demands and skills. (Photographs © 2008 Beverly Schaefer.)

## Nutrition

### CARBOHYDRATE INTAKE

- **Level of muscle glycogen stores** before exercise important
- Muscle glycogen is depleted with 90–180 minutes of exercise at 60%–80% $\dot{V}O_2$ max or 15–30 minutes of exercise at 90%–130% $\dot{V}O_2$ max, performed in intervals of 1–5 minutes.
- Athletes need 10–12 g of carbohydrate per kg/day to maximize glucogen stores (Chapter 5, Sports Nutrition).
- Comparison of soccer players with high versus low prematch muscle glycogen content:
  - Low glycogen group covered 24% less total distance (most at high speeds).
  - Players with low pregame values had no glycogen stores at end of game compared with 10% of remaining glycogen stores in players with high pregame values.
  - One study looked at the effects of a high-carbohydrate diet (65% of calories) versus low-carbohydrate diet (35% of calories) on technical and physiologic variables.
  - Technical variables included number of successful/unsuccessful "green" (low-risk, nonpenetrating) and "red" (high-risk, penetrating) passes and total number of touches per possession.
  - After a high-carbohydrate diet, higher pregame muscle glycogen content was observed along with 33% more high-intensity work. No difference noted in any technical variables.

## Recovery

- Often refers to physical and mental restoration that leads to performance readiness; it employs multiple tools to enhance performance
- **Active Recovery**
  - A study evaluated the use of a "cool down" period, performed by 81% of French professional soccer teams and involved a strategy of either running, biking, or swimming for 15–30 minutes after a match and found that this enhanced blood lactate removal compared with passive recovery.

- However, other studies identified decreased glycogen stores after this strategy. No effect was noted on oxidative stress markers.
- Unclear at this point if active recovery is beneficial or detrimental.

### Cold-Water Immersion (CWI)

- Cold-water immersion (CWI) has gained tremendous popularity among athletes to minimize fatigue and enhance postexercise recovery.
- There are various CWI protocols; typically involves immersion in 9°C–10°C water for 5–20 minutes immediately after exercise.
- Studies have suggested an effect on short- and long-term recovery by:
  - Ameliorating hyperthermia and rapidly reducing the body temperature, which, in turn, decreases CNS fatigue and possibly decreases cardiovascular strain
  - Facilitating the removal muscle metabolites, which may lead to delayed-onset muscle soreness
  - Enhancing parasympathetic reactivation, which leads to a decrease in heart rate and is associated with improved long-term recovery

## GENERAL INJURY PATTERNS

### Overall Injury Incidence

- Overall injury incidence in soccer is favorable (much lower than in American football).
- Number of injuries increases as skill and age increase—low levels of injuries are seen in children aged <10 years, with the rate increasing 10-fold at the high school level.
- At younger age levels, girls are noted to have a higher incidence of injury than boys (Table 67.1).
- At the collegiate level, gender differences are less striking, with the exception of anterior cruciate ligament (ACL) injuries.
- **National Collegiate Athletic Association** (NCAA) data from 2004/05 to 2008/09 seasons showed an overall injury rate of 7.7/1000 athlete exposures (AEs) in men's soccer and 7.3/1000 AEs in women's soccer.
  - Lower limb injuries (ankle, knee, and upper limb) were most common and accounted for 65.6% of men's injuries and 65.3% of women's injuries (Table 67.2).
- Soccer players are three times more likely to be injured in a game compared to practice (Table 67.3).
- Female soccer players noted to have a higher incidence of ACL injuries compare to their male counterparts.
  - A 2005 study by Angel et al. reviewed ACL injuries in NCAA male and female basketball and soccer athletes from 1990 to 2002 and found that females sustained 67% of all noncontact ACL injures compared with 58% in males.
- Injury patterns are similar for indoor and outdoor soccer.
- Goalpost injuries can be fatal; should be preventable.
  - In the US during 1980–1994, 27 injuries were caused by falling goalposts, with 18 fatal cases (14 involved head trauma).

### Injury Surveillance System (ISS)

- NCAA has used the Injury Surveillance System (ISS) to collect injury and exposure data from intercollegiate athletes since 1982. Injury is defined as time loss.
  - **Advantages**: Data are shared with NCAA sport and policy committees as well individual institutions to make evidence-based decisions regarding health and safety issues.
  - **Disadvantages of ISS**: Injuries without time loss are missed, certain injuries may not be "time loss injury" (i.e., no time loss if in season, but time loss if off season).
- Despite these limitations, ISS data provide a denominator (number of AEs) and are thus useful for comparing sports, genders, and injury types.

**TABLE 67.1 INJURIES IN SOCCER PER 1000 PLAYER HOURS: BOYS VERSUS GIRLS**

| Study | Girls | Boys |
|---|---|---|
| Engstrom et al., 1991* | 12 | 5 |
| Nilsson and Roaas, 1978 | 32 | 14 |
| Maehlum et al., 1986 | 17.6 | 8.9 |
| Schmidt-Olsen et al., 1991 | 17.6 | 7.4 |
| Powell 1999 (injuries/1000 AEs) | 5.3 | 4.6 |

*Denotes studies using time loss from practice/play as definition of injury.

**TABLE 67.2 NCAA DATA OF INJURY PERCENTAGE BREAKDOWN FROM THE 2004/05 TO 2008/09 SEASON**

| Men's Soccer | | Women's Soccer | |
|---|---|---|---|
| Concussion | 5.5% | Concussion | 9.2% |
| Head, face, neck | 4.3% | Head, face, neck | 4.1% |
| Upper limb | 6.2% | Upper limb | 5.6% |
| Torso and pelvis | 14.7% | Torso and pelvis | 12.3% |
| Lower limb | 65.6% | Lower limb | 65.3% |
| Other | 3.7% | Other | 3.5% |

Adapted from NCAA Data, 2004–2005 through 2008–2009; National Collegiate Athletic Association.

**TABLE 67.3 INJURY DATA FROM NCAA INJURY SURVEILLANCE SYSTEM 2009–2010 THROUGH 2013–2014**

| Sport | Event | Avg Annual Estimate of Injuries | Avg Annual Estimates of No. of Athlete-Exposures | Estimated Injury Rate Per 1,000 Athlete-Exposures |
|---|---|---|---|---|
| Men's Soccer | Competition | 6,458 | 360,880 | 8.0 |
| | Practice | 6,977 | 1,323,974 | |
| | Overall | 13,435 | 1,684,854 | |
| Women's Soccer | Competition | 7,434 | 432,347 | 8.4 |
| | Practice | 7,679 | 1,367,650 | |
| | Overall | 15,113 | 1,799,997 | |

Adapted from Kerr ZY, Marshall SW, Dompier TP, Corlette J, Klossner DA, Gilchrist J. College Sports-Related Injuries—United States, 2009-10 Through 2013-14 Academic Years. *MMWR Morb Mortal Wkly Rep.* 2015;64(48):1330-1336.

**TABLE 67.4 KNEE STRUCTURES INJURED IN SOCCER (1989–1993)**

| | *Men's Soccer* | | *Women's Soccer* | | |
|---|---|---|---|---|---|
| | **No.** | **Rate*** | **No.** | **Rate*** | **P (Variance)** |
| Collateral ligament | 316 | 0.51 | 192 | 0.62 | 0.02 |
| Torn meniscus | 119 | 0.19 | 105 | 0.34 | 0.00 |
| Patella/patellar tendon | 130 | 0.21 | 92 | 0.30 | 0.01 |
| Anterior cruciate ligament | 81 | 0.13 | 97 | 0.31 | 0.00 |
| Posterior cruciate ligament | 22 | 0.04 | 12 | 0.04 | |
| Athlete exposures | 626,223 | | 308,748 | | |

*Per 100,000.
Data from Arendt E, Dick R. Knee Injury Patterns Among Men and Women in Collegiate Basketball and Soccer. *The American Journal of Sports Medicine*, 23(6), 694-701, 1995.

## Indoor Soccer

- Patterns may differ slightly, but in general, type, location, and severity of injury appear similar in outdoor and indoor soccer.
- Injury data sparse; only two studies provide a denominator and include women.
  - A 1996 study looked at the Lake Placid Dawn to Dark Festival (3-day indoor tournament)—injury incidence for men was 5.79 per 100 player hours and for women was 4.74. No significant difference in incidence of injury between men and women; no difference in knee injuries. Severe injuries were more likely to be noncontact; 71.4% injuries were in lower extremities.
- Data evaluating injury incidence, severity of injury, and mechanisms of injury on natural grass versus third generation artificial turf by male and female players demonstrated no significant differences between these surfaces.

## Prevention of Injuries

- In recent years, the Fédération Internationale de Football Association (FIFA) and its Medical Assessment and Research Center (F-MARC) have developed injury prevention programs such as the FIFA 11 and FIFA 11+ programs.
  - FIFA 11+ is a restructured program that addressed the compliance issues and inadequate therapeutic exercises of the original FIFA 11 program.
  - FIFA 11+ consists of a 20-minute warm-up program that should be performed twice a week before each training session. Before a match, only running exercises should be performed.
  - Studies evaluating the efficacy of this program in varied settings (high school, collegiate, and professional levels) have showed lower extremity injury prevention by up to 30%–50%.
- Extensive research has been performed specifically targeted toward ACL injuries in women and incorporates the use of neuromuscular (NM) training.
  - Hewett et al. (1999): 60- to 90-minute training sessions 3 times per week for 6 weeks in soccer, volleyball, and basketball resulted in a 72% decrease in noncontact ACL injuries.
  - Mandelbaum et al. (2005): Studied the Prevent Injury and Enhance Performance Program (PEP), which emphasized proprioception and NM training. The program included 20 minutes of soccer-specific agility drills, plyometrics, lower extremity and trunk stretching, and strengthening exercises. A 74% reduction in ACL tears was seen over 2 years (see Recommended Readings).

## SPECIFIC INJURY PATTERNS

### Knee Injuries (see Table 67.4)

#### Overall Injury Patterns

- One needs only to watch soccer players pivot and cut back and forth, change direction while decelerating, take cross with half-volley, or cross ball from one side of field to another to understand why they may be at an increased risk of knee pathology.
- Knee injuries account for 8%–18% of injuries in practices and games (NCAA, 1988–1989 through 2002–2003).

#### Meniscal Injuries

- Same sport-specific skills that put player at a risk of ACL injury
- In setting of ACL deficiency, impact of meniscal injuries dramatic
- Coexistence of meniscal and ligamentous injuries to knee should not be underestimated in soccer players; isolated injury may not prohibit activity, whereas additional injuries make it difficult to return to high-level play.

#### Medial Collateral Ligament (MCL) Injuries

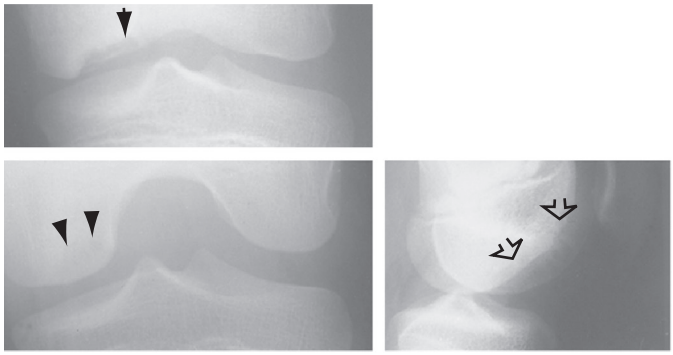
- Inside of foot pass/redirection and block tackles put athlete at risk and also make early return difficult because of direct valgus force.
- Risk increases with poor technique, tentative athlete, and leg extended away from the body.

#### Osteochondral Injuries

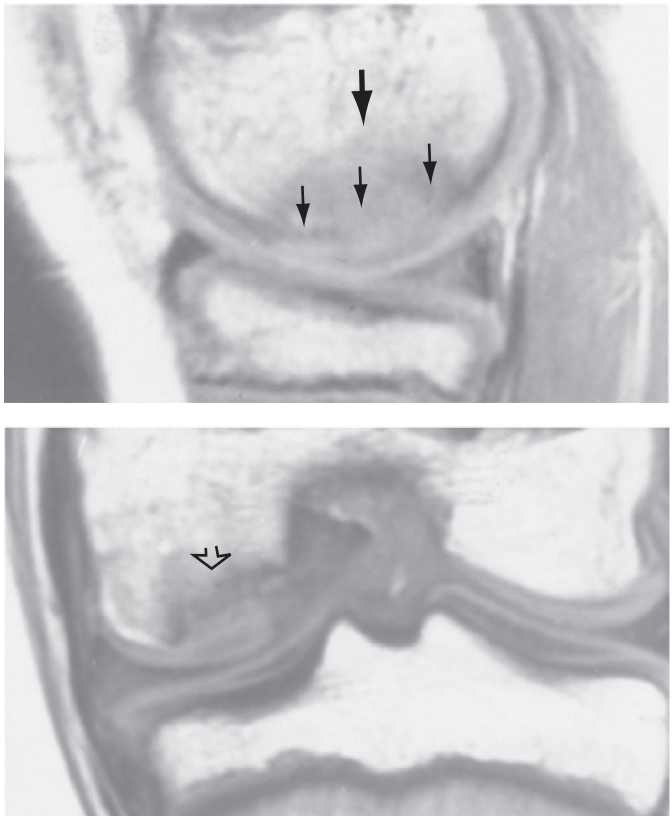
- Injuries involving the bone and overlying articular cartilage
- Most commonly occurs in the knee at the medial femoral condyle and presents as pain and/or swelling with activity
- Diagnosis made with plain radiographs, although may need additional testing such as magnetic resonance imaging (MRI) (Fig. 67.2)
- Treatment depends on clinical presentation as well as diagnostic tests. If significant lesion and/or separation of fragment, arthroscopic treatment with microfracture procedure versus osteochondral autograft transfer system (OATS) procedure often indicated.

## Muscle Contusions

- Common in soccer and most common in pediatric age group
- Accounted for 35.2% of injuries in a summer training camp
- Rate of injury increases with age; boys at a greater risk than girls
- Shin guards significantly decrease the risk of contusions (and fractures) to lower leg.



**A.** Radiographs of a 12-year-old soccer player with symptomatic medial femoral osteochondritis dissecans.



**B.** Magnetic resonance imaging shows loss of articular surface continuty *(small arrows)*. *Open and larger arrows* indicate the osteochondritis dissecans fragment subchondral junction.

**Figure 67.2.** Knee injuries. (From DeLee J, Drez D, Miller M. *DeLee and Drez's Orthopaedic Sports Medicine*, 2nd ed. Philadelphia: Saunders; 2002.)

- Treatment: rest, ice, compression, and elevation (RICE); avoidance of aggressive passive stretching in acutely injured muscle
- For large contusions of quadriceps, flex knee 120 degrees and use compressive wrapping in this position for 24 hours (stops bleeding, avoids more significant injury)
- Re-injury to the quadriceps is a major factor in developing **myositis ossificans,** also called *heterotopic (ectopic) ossification*.
  - Defined as new bone formation in a muscle after injury
  - Occurs in 9%–20% all quadriceps contusions; average time of disability is 73 days

## Muscle Strains

- Muscle strains often result from excessive stretch while muscle is activated.
- Tears tend to occur near the muscle–tendon junction.
- Common, frustrating injuries in older adolescents/college athletes; less common in younger population
- Eccentric contraction plays a key role in production of muscle strain.
- Muscles with increased content of type II fibers and muscles that cross two joints are more susceptible.
  - Rectus femoris crosses two joints, has a high content of type II fibers, and contracts eccentrically in decelerating hip and knee.
  - Hamstring muscles contract eccentrically with running and deceleration and have a high content of type II fibers.
- Adductor longus and gastrocnemius also commonly involved
- Fatigue increases the susceptibility to muscle strain.
  - Amount of energy absorbed by a fatigued muscle is 69.7%–92% of that by muscles of a control leg.
  - Lowest energy absorption occurs in muscles that are most fatigued.
  - Fatigued muscles absorb less energy before reaching a degree of stretch that causes injuries.
- Established continuum of injury during passive muscle stretch.
  - Rabbit skeletal muscle stretched at 10 cm/second to 60%, 70%, 80%, and 90% of force required to cause passive failure of muscle
  - Continuum of diminished maximal isometric contractile force, muscle fiber disruption, edema, hemorrhage, and decreased electromyography (EMG) maximal voltage amp
  - In previously injured muscle, failure occurs in muscle itself; hence need to protect injured muscle from further injury before healing is complete

### Prevention

- Adult players with decreased flexibility have a higher incidence (not significant) of muscle strain; dominant leg more often involved
- Emphasize beneficial effects of warm-up, temperature, and stretching on mechanical properties of muscle.
- Several factors protect muscle: strength, endurance, and flexibility.
- Eccentric hamstring strengthening ("Russian Hamstring Curls") useful in preventing hamstring injuries

## Stress Fractures

- Represents overuse injury to the bone; generally caused by training regimen
- Bone scans indicate that most overuse injuries and stress fractures occur in lower extremity: tibia (49.1%), tarsals (25.3%), femur (7.2%), metatarsals (8.8%), fibula (6.6%) pelvis (1.6%), sesamoids (0.9%), and spine (0.6%).
- Fifth metatarsal stress fractures, specifically Jones type (metaphyseal–diaphyseal junction), have unpredictable healing and thus screw placement for internal fixation should be considered (Fig. 67.3).
- Training errors accounted for 22.4% of fractures.
- Other factors: playing surface, cleat and indoor shoe requirements, changes in training regimen/intensity, and (in female athletes) hormonal and/or nutritional influences
- Address biomechanical issues (leg length discrepancies, femoral anteversion, foot pronation, and increased knee valgus) during preparticipation physical examination.
- Consider female athlete triad (disordered eating, menstrual dysfunction, and decreased bone mineral density [BMD]).

**A-C.** Type IA (DeLee) fifth metatarsal fracture in collegiate soccer player.
**D-F.** After fixation with variable pitch compression screw.

**Figure 67.3.** Stress fractures. (From Canale T, Beaty J. *Cambell's Operative Orthopaedics*. 11th ed. Philadelphia: Mosby; 2007.)

- Although more common in sports emphasizing leanness (cross-country running and swimming) and sports with subjective scoring (gymnastics and skating), triad occurs in all sports.
- Menstrual dysfunction and eating disorders are associated with low BMD at axial as well as appendicular sites.
- Early recognition of eating disorders and menstrual dysfunction is essential.
- Optimize prevention and early treatment through education.

## Groin Injuries

- Accounts for 5% of all injuries in soccer; return to play difficult
- Direct trauma can result in injury to external genitalia, muscles, vessels, or joints
- Other injuries include osteitis pubis (symphysis pubis instability), adductor, rectus or iliopsoas muscle strain, stress and avulsion fractures, hernia, and sportman's hernia/inguinal disruption (ID)
  - Osteitis pubis: low-grade inflammation at symphysis
  - Pain at adductor insertion and symphysis for >2 months; pain at morning or night, with cough/sneeze; positive bone scan and radiograph
    - Treated with either standard physical therapy (stretching, friction massage, and other modalities) or active training (strengthening, proprioceptive NM facilitation, and balance)
    - 79% of those treated with active training (vs. 14% treated with standard physical therapy) returned to play

### Sportsman's Hernia (Inguinal Disruption [ID])

- Defined as pain, either of insidious or acute onset, which occurs predominantly in the groin region near the pubic tubercle; diagnosis of exclusion after other pathology (i.e., hernia) ruled out
- 90% male, 70% insidious onset, 30% report injury
- Affects soccer, rugby, track, hockey athletes; muscular imbalance common
- Diagnosis can be made if 3 out of 5 clinical signs are detected: tenderness over the pubic tubercle, palpable tenderness over the deep inguinal ring, pain or dilation over the external ring with no obvious hernia, pain at origin of the adductor longus tendon, and dull diffuse pain in the groin with possible radiation to the perineum (see Recommended Readings for British Hernia Society's 2014 Position Statement).
- Pain worse with activity, particularly sudden movement, twisting, striding, sprinting, long or dead ball kicks, sidestepping, coughing/sneezing, and sit-ups.
- Radiographs, bone scans, and MRI often used to exclude other diagnoses; newer use of ultrasound to diagnose sportsman's hernia
- Treatment: Initially conservative rehabilitation with strengthening, relative rest, and NSAIDs; if no improvement and workup otherwise negative, surgical treatment indicated
- Surgery: In an 8-year study, 97% successful; return to play within 6 weeks

## Ankle Injuries and Impingement Syndromes

- Ankle sprains most common injury in soccer; important to detect and treat appropriately

- Usual mechanism is inversion plantar flexion injury; other mechanisms in soccer: outside of foot pass/shot
- Because soccer players rely on mobility and "touch" on ball, challenge often lies in returning athletes to 100% functional activity.
- If significant ankle laxity detected during preparticipation examination, strengthening and proprioceptive programs should be initiated; consider prophylactic taping or bracing.
- Athletes with ankle sprains who experience pain and disability after injury develop "ankle impingement" syndromes; a difficult problem in soccer players
  - Consider in patients with anterolateral pain, no instability, history of prior sprains
  - Soccer players complain of pain while trying to pivot or push off one foot or with instep shooting.
  - Cause: hypertrophic scar formation, synovium, and fibrocartilage in anterolateral tibular–talar space from the anterior capsule into the lateral gutter
  - Occasionally described as "meniscoid" lesions in soccer players

## Heading and Concussions

- Heading is soccer-specific skill wherein the head is used to control the ball, clear it from defensive area, or strike it at the goal.
- In 300 games, typical European player receives 2000 blows to head.
- Regulation ball weighs 396–453 g, with a circumference of 68–71 cm, and is inflated to a pressure of 1 kg/cm$^2$, achieving speeds of 26.82–53.64 meters/second during match.
- Older leather soccer balls can become waterlogged, which increases their weight by 20%; modern balls are water resistant.
- Kicked ball achieves average speed of 114.4 km/hour from 10 meters away; average impact speed is 116 kpm (can reach 200 kpm at full force).
- Accelerometer data measuring ball speed at 15.5 meters/second (35 mph) demonstrated head acceleration forces of approximately 20 gravity, with peak forces of 1200 Nm.
- Greater than forces in boxing, wherein a punch generates head acceleration at 100 gravity. Forces in soccer tend to be linear, whereas they are rotational in boxing, and are thus associated with increased shearing forces and more damage.
- Ball in contact with head for $\frac{1}{63}$ to $\frac{1}{128}$ second; longer the impact, smaller the force.
- Repetitive and cumulative forces have raised concerns regarding head and neck injury.
- Skilled players maintain neck rigidly as head impacts ball; decreases angular acceleration of head, ultimately protective
- Applying Newton's second law (force = mass × acceleration), if neck musculature is rigid, mass of head now approximates mass of body; same force applied to greater mass yields less acceleration, thus decreasing forces acting on the skull
- The risk may be higher in younger player with weak musculature and unmastered technique.

### Head Injuries

- The number of head injuries and concussions has increased in NCAA soccer for both men and women (see Table 67.2). (For NCAA data, see Chapter 45, Head Injuries.)
- At high school level, mild traumatic brain injury occurs at rates of 1.14 and 0.92 per 100 player-seasons in girls' and boys' soccer, respectively, accounting for 4.3% and 3.9% total injuries in those sports, respectively.
- Assessment and treatment of head-injured athlete and return to play issues no different in soccer players than in other athletes (see Chapter 45)
- Neuropsychological testing techniques reliably quantify brain function by examining brain–behavior relationships (see Chapter 45).

#### 1993 US OLYMPIC SPORTS FESTIVAL STUDY

- A questionnaire study of male and female soccer players in 1993 US Olympic Sports Festival determined mechanisms, frequency, and sequelae of head injuries.
- Participants estimated the average number of times that they headed ball during practice and game as well as the frequency of heading ball collision with other players. History of concussive episodes outside soccer was noted as well as symptoms associated with heading (Colorado Guidelines grading system).
- Injury defined as event that (1) required evaluation by physician, certified athletic trainer, or dentist; (2) required removal from game/practice; (3) resulted in play stoppage; (4) resulted in sequelae; or (5) prevented participation in later games/practice.
- Weaknesses of study: self-report, retrospective, and selection bias
- Results of data from144 athletes:
  - Men more likely than women to have sustained concussion (2.16-fold higher risk); 89% of male and 43% of female players experienced head injury during soccer (forwards reported higher rate than goalkeepers)
  - Over half of players experienced at least one headache after heading the ball, but large variation in frequency and duration of headache; most often associated with poor technique, heavy or overinflated ball, weather conditions
  - 50% probability that male player would have a concussion in 10 years of playing versus 22% probability for female player (true rate impossible to determine given the lack of denominator)
  - Mechanism of injury summarized in Table 67.5

#### ONGOING DEBATE ABOUT HEADING

- **Early studies flawed** by methodology, lack of good control groups, lack of screening for acute head injuries, alcohol use, or previous motor vehicle accident
  - **US National team players and age-matched track athletes** were assessed using MRI, surveys about symptoms of head injury, and screens for alcohol use.
  - Heading exposure index: length of season and amount of potential heading for various soccer activities used to derive participation score for each player
  - Nine soccer players with positive MRI findings (cortical atrophy, ventricular enlargement, focal atrophy, and cavum septi pellucidi); similar findings in six track and field athletes
  - Symptoms and MRI findings in soccer players did not correlate with age, number of years of play, heading exposure index, or number of headers.

**TABLE 67.5 MECHANISM OF HEAD INJURY IN US OLYMPIC FESTIVAL SOCCER PLAYERS**

| | Men | Women |
|---|---|---|
| Collision with another player | 48 | 20 |
| Collision with ground or indoor wall | 10 | 3 |
| Collision with goalpost | 1 | 2 |
| Collision with ball | 15 | 3 |

Data from Barnes BC, Cooper L, Kirkendall DT, McDermott TP, Jordan BD, Garrett WE Jr. Concussion history in elite male and female soccer players. *Am J Sports Med.* 1998;26(3):433-438.

- None of the MRI findings or total MRI scores correlated with the number of years of participation, number of headings, number of head injuries, and total symptom scores.
- Only significant predictor of symptoms was history of prior acute head injury ($P = .003$; r value: 0.63).
  - **Haglund (1993) compared Swedish amateur boxers with age-matched track athletes and soccer players** and track athletes. Multiple parameters were evaluated, including Mini-Mental Status examination, personality trait study, CT, MRI scans, EEG, and neuropsychological tests.
- No differences in physical, neurologic, CT, or MRI abnormalities among groups
- Significantly higher incidence of mild-to-moderate EEG abnormalities in boxers; no EEG changes noted in soccer players (except 1 arachnoidal cyst).
- Conclusion: No signs of chronic brain damage in soccer players who are known as frequent headers
  - **Matser (1998) compared 53 active Dutch soccer players with 27 elite swimming and track athletes** in the Netherlands, using neuropsychological tests.
- Classified soccer players as headers or nonheaders (goalkeeper and midfielders classified as nonheaders [rationale unclear], forwards and defenders as headers)
- Forwards and defenders performed significantly lower on certain neuropsychological tests compared with midfielders and goalkeepers.
- Effect of sports-related concussions and number of headers not separated; also difficult to ascertain how these groups were identified
  - **Matser's cross-sectional study (1999):** Soccer players scored significantly lower on neuropsychological tests of planning and memory than swimmers and track athletes.
- Number of concussions inversely related to performance on several neuropsychological tests
- The study concluded that repetitive heading may explain poor cognitive function.
- Several methodological concerns: soccer players drank more alcohol, effect of prior concussions not separated from heading exposure, authors did not explain how they obtained some of their data regarding severity of concussions

### Additional Considerations

- Concerns regarding the safety of soccer ball heading have been an ongoing debate, particularly in youth soccer.
- Reports of increasing rates of concussions have questioned the safety of soccer heading in children aged <14 years and led to the initiation of policy changes for heading in youth soccer.
- Recent recommendations from the Recognize to Recover (R2R) Program by the US Soccer Federation advises:
  - Children aged ≤10 years: no heading in games and practices
  - Children aged 11–13 years: heading training limited to maximum of 30 minutes/week than 15–20 headers per player per week
- **Constock et al. (2015):** Retrospective analysis of longitudinal surveillance data of high school soccer players from 2005–2006 to 2013–2014 evaluating trends of boys' and girls' concussions and patterns and mechanisms of injury.
  - Overall, there were 627 concussions per 1,393,753 AEs among girls and 442 concussions per 1,592,238 AEs among boys.
  - Heading was the most common soccer activity that led to concussions, with contact with another player being the most common mechanism of injury in heading-related concussions (78.1% in boys and 61.9% in girls).
  - Study conclusion: While banning heading in youth soccer may prevent certain concussions, a more effective approach may be to target prevention of player to player contact.
- Other recent studies have evaluated anatomic changes seen in the brain of amateur soccer players.
- **Lipton et al. (2013):** Used diffusion tensor imaging (DTI) imaging and neuropsychological testing to evaluate for subclinical evidence of traumatic brain injury in 37 amateur soccer players (29 men and 8 women)
  - All players were asked to complete a questionnaire, which determined the number of heading exposures in past year and history of prior concussions.
  - High frequency of soccer heading (>885–1800 heading per year) was associated with lower white matter fractional anisotropy (FA) and poorer neurocognitive performance on a test of memory compared with players who performed less heading.
  - Lower FA was noted in the temporo-occipital white matter.
  - This study suggested that these microstructural changes appear to be similar to those seen in patients with known traumatic brain injuries.
  - Heading found to correlate with FA and poorer memory performance, independent of the player's history of concussion; significant methodological flaws exist in study.
  - A major drawback of this study was the use self-reported heading exposure in the previous 12 months by the athlete.
- **Other prospective studies**
  - Putukian (2000): no acute effect of heading on cognitive function as assessed by neuropsychological tests before and after practice session
  - Putukian (2001): no effect of heading on cognitive function in men and women college soccer players as assessed by neuropsychological testing

### Prevention of Head Injury

- The R2R program has been established by the US Soccer Federation to improve the safety of soccer player of all ages and reduce injuries. It aims to strengthen the role of parents, players, coaches, and officials and expand their knowledge regarding injury prevention.
- Helmets do not prevent concussion; no evidence to suggest helmets or headbands will play a role in decreasing concussion. Prospective data show no effect of headbands on forces incurred during heading. Some data suggest that headbands may actually make athletes more aggressive by giving an unfounded sense of protection from head injury.
- Potential role for strengthening of neck musculature, appropriate technique, and enforcement of rules in preventing illegal play/contact
- Mouth guards: decrease the risk of dental injury; newer materials and construction make it easier to use a mouth guard without inhibiting breathing or communicating

## GUIDELINES FOR SUCCESSFUL PARTICIPATION IN SOCCER

- Preparticipation physical examination: focus on previous ankle or knee injuries, careful assessment for ligamentous laxity as well as history of concussion.
- Prophylactic taping or bracing for ankle laxity
- Ensure adequate equipment, including shoes, clothes, and shin guards; consider mouth guards
- Optimize sport-specific cardiovascular training; endurance and intermittent sprints
- Appropriate 15- to 20-minute warm-up and flexibility programs (e.g., FIFA 11+) before practice and game with emphasis on adductors, hamstrings, gastrocsoleus complex, iliotibial band, neck, and upper extremities
- Proprioceptive training for injury prevention with stress on jumping skill and technique; particularly important for ACL injuries in women

- Ensure appropriate technique for all skills, particularly heading, blocking, and tackling.
- Appropriate nutrition and hydration; education about performance enhancement
- Use smaller balls for younger participants.
- Ensure communication with parents and coaches before and throughout the season about methods to decrease injuries and treatment of injuries.
- Appropriate education of athletes about injury and prevention; explain the increased risks with foul play.
- Appropriate supervision of practice sessions by certified athletic trainer and of games by trainer and physician

## SUMMARY

- Soccer enjoys a relatively low injury profile, making it an ideal sport for youngsters and adults. There are significant physiologic demands, requiring both aerobic and anaerobic metabolism and adequate balance and proprioception.
- Soccer-specific nutrition is important and may improve performance.
- Lower extremity injuries predominate; ankle most common, knee most severe
- ACL injuries in female players remain of paramount concern.
- Head injuries of increasing concern, particularly in youth soccer; limiting/banning of heading in children aged <14 years may decrease concussion rates. Long-term effects of severe or cumulative concussions is less certain, and additional studies are warranted to determine the same.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 68 RUGBY

Jon S. Patricios • William Dexter

## INTRODUCTION

- Rugby Union (rugby) is a continuous, multiple-sprint, unhelmeted (padded headgear is permitted) collision team sport (>200 tackles per game) played by men and women, boys and girls, in 120 countries across six continents.
- The game lasts for 80 minutes with a 10-minute half-time interval.
- The fifteen-a-side version is the traditional and most popular version, but seven-a-side and ten-a-side versions are also played.
- The differentiating feature from American Football is that rugby is continuous in nature with no "time outs"; this requires injured players to be assessed during play before a decision is made whether to replace them with one of eight reserves.
- The international federation, World Rugby (WR) (formerly the International Rugby Board), governs the sport.

## EPIDEMIOLOGY

- Rugby has one of the higher overall rates of injury (69/1000 player hours) compared to soccer (28/1000 player hours) and ice hockey (53/1000 player hours).
- Risk of injury increases with age and rugby level; this has been attributed to greater size, speed, increased competitiveness, more aggression and foul play.
- Risk of injury is higher in men than in women with the distribution of injuries varying by sex.
- Different facets of the game allow players of differing physiques and talents to contribute in different positions. All players may run or kick the ball, tackle and be involved in rucks and mauls.
- The forwards are divided into tight forwards comprising two props, a hooker, and two locks; they are largely responsible for the set pieces of play such as scrums and lineouts, and three "loose forwards" who are also involved in set pieces but who have a mandate to roam more.
- Forwards tend to be heavier and place an emphasis on strength and power.
- The seven backs tend to be smaller in stature with speed, agility, and skill being the assets.
- Because of the different player profiles, large discrepancies in size may occur resulting in physical challenges for smaller players.

## GENERAL PRINCIPLES

### Rules

- Field-based team sport lasting up to 80 minutes with 30 players involved in continuous play interrupted only by:
  - Set play—scrums and lineouts
  - Penalties
  - Injury
  - Half time (10 minutes)

### Terminology

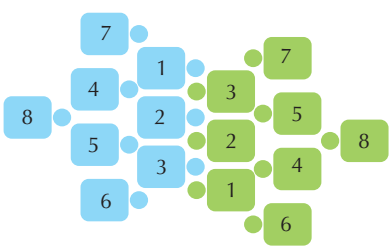
- **Scrum:** Formation used in the set play re-starting play after a knock-on (spilling the ball forward) or forward pass; the forwards from each side bind together and then the two packs (groups of forwards) come together to allow the scrumhalf to deliver the ball to the scrum. A scrum can also be awarded or chosen in different circumstances by the referee (Fig. 68.1A, B).
- **Lineout:** The set play re-starting play after the ball has been carried or kicked to touch (out of bounds); both sets of forwards will line up opposite each other with one team throwing the ball between the two lines of forwards. The throw must be directly down the middle of the two lines (Fig. 68.1C).
- **Ruck:** Typically, after a runner has come into contact with another player and the ball has been delivered to the ground, once any combination of at least three players have bound themselves over the ball, a ruck has been formed. The primary difference from the maul is that the ball is on the ground.
- **Tackle:** The act of physically bringing an opposing player who has the ball to the ground; the rugby tackle involves leading with the shoulder and wrapping arms around and placing the head behind the ball carrier; legal tackles must not be around the neck or head and tacklers are required to use both arms and not tip the tackled player head first into the ground (Fig. 68.1D)
- **"Blood bin":** A 15-minute period during which players who are bleeding or have open wounds may be removed from the field and temporarily replaced whilst their wounds are attended to and the bleeding stopped
- **Head Injury Assessment (HIA):** A 10-minute period during which players suspected of having suffered a concussion may be removed from the field and evaluated; this applies only to professional rugby.

### Injuries

- Injury patterns are influenced by position: forwards suffer more knee, shoulder, and ankle injuries, whereas backs have a greater incidence of thigh (particularly hamstring) injuries.

#### Injury Prevention

- Rugby is the first professional sport to produce tournament player welfare standards, a compulsory checklist for players before international competition. These include:
  - Confirmation before a tournament by the team doctor or Union chief medical officer that all players are medically, mentally, dentally, and physically fit to participate
  - Confirmation that each player has completed the WR cardiac screening questionnaire and that a cardiac examination as described (which includes a mandatory ECG) has been completed
  - Completion by all team and match day medical staff of the following WR education modules: Concussion Management; Keep Rugby Clean anti-doping module; and Keep Rugby Onside anti-corruption module
  - Completion of WR Level 2 ICIR accreditation (or equivalent) by all Team Medical Staff and Match-Day Doctors
  - The host union must indicate to WR what medical staff will be in place on match day at each venue. The following medical staff must be in attendance: (i) match doctor, (ii) surgery doctor, (iii) surgery nurse, (iv) ALS paramedic, (v) an on-site ILS ambulance (staffed), (vi) minimum of four BLS paramedics.




**A.** Positions adopted by opposing forwards in a scrum

**B.** Setting a scrum

**C.** A lineout

**D.** Correct tackle technique

**Figure 68.1.** Position terminology. (**B** and **D** from South African Rugby Union; **C** reused with permission from Gallo Images.)

- Completion of a concussion education session by all players and team management within the year before commencement of the tournament; this education session should at least cover the essential information outlined in the HIA protocol document.
- Completion of a baseline concussion assessment for each player and, as a minimum, using the SCAT 3. It is recommended that teams also include neurocognitive computer assessments.
- Confirmation that a concussion risk stratification has been completed on all players to support concussion management on an individual basis
- Acknowledge that the tournament has an untoward incident review system for potential medical mismanagement and specifically for incidents where criteria, identified in the HIA, for permanent removal from play following a head injury are not enforced. All team staff acknowledge that they will participate if requested in any untoward incident review and that a charge of misconduct can be applied following an untoward incident review.
- Acknowledgement that the match-day doctor has the power to unilaterally remove an injured player from further game participation
- Capture of all match injury data on an Injury Data Capture App required for the Injury Surveillance Monitoring System

### Neck Strengthening

- Specific neck strengthening programs may help reduce the incidence of neck injuries.

### Equipment

- Neither rugby headgear nor mouth guards have been shown to prevent concussion.
- Mouthguards have been unequivocally shown to reduce the incidence of dental and orofacial injuries. Customized mouthguards may be better tolerated and improve compliance.

## TRAUMATIC INJURIES

Collisions are more likely to result in injury, whereas tackles are the game event responsible for the highest number of injuries and the greatest loss of time in rugby because they are by far the most common contact event.

### Head Injuries

- Head injury management is an area wherein rugby has taken a lead.
- WR has focused on head injuries as part of its Player Welfare initiative (http://playerwelfare.worldrugby.org/concussion)
- National programs such as BokSmart (www.boksmart.com)

### Concussion

- Concussion is relatively common in rugby; the incidence has been reported as up to 1.2/1000 player hours in amateur rugby and 3.9/1000 player hours in professional rugby.
- Incidence appears to be increasing because of enhanced awareness and reporting.
- The most intense area of recent focus in WR has been head injuries, particularly concussion.
- An independent Concussion Advisory Group consisting of a neurosurgeon, sports physician, neuropathologist, and epidemiologist evaluate all rugby concussion protocols.
- Return-to-play (RTP) decisions following concussion should be individualized, taking into account player symptom score, general and neurologic examination, balance assessment, and verbal and possibly computerized cognitive scores.
- Minimum guidelines for RTP following concussion have been determined by WR (adults: a minimum 1 week complete rest followed by 5-day graduated return to play [GRTP]; 2 weeks minimum complete rest for <18 years followed by 5-day GRTP)
- Advanced Care Settings (ACS) are "gold standard" criteria determined by WR as being necessary for optimal concussion management, which include all of the following:
  - Medical doctors with training and experience in recognizing and managing concussion and suspected concussion
  - Access to brain imaging facilities and neuroradiologists
  - Access to a multidisciplinary team of specialists including neurologists, neurosurgeons, neuropsychologists, neurocognitive testing, and balance and vestibular rehabilitation therapists
  - ACS must be in place for any deviation from the minimum criteria

#### HEAD INJURY ASSESSMENT (HIA)

- HIA has been developed for elite-level adult players where experienced healthcare professionals support the teams.
- Recognizing that concussion may have a variable natural history, with transient, fluctuating, delayed, and evolving signs or symptoms, WR promotes a three-stage approach to assessment, diagnosis, and monitoring of head injuries.
- HIA process reinforces review of injured players at specific points in time in order to increase the recognition of concussion:
  - Stage 1: pitch-side assessment using HIA Form 1
  - Stage 2: postgame, same-day assessment using HIA Form 2
  - Stage 3: 36–48 hours postinjury assessment using HIA Form 3
  - Confirmed cases of concussion then still have to undergo GRTP process.
- WR recommends that players with head injury should be regularly monitored for evolving signs and symptoms between these identified timeframes.
- WR has introduced video analysis of suspected cases of concussion as an additional triage tool.

#### THE 6 "Rs" OF RUGBY CONCUSSION MANAGEMENT

1. **Recognize:** Learn the signs and symptoms of a concussion so that you understand when athlete might have a suspected concussion.

2. **Remove:** If athlete has a concussion or even a suspected concussion, he or she must be removed from play immediately.
3. **Refer:** Once removed from play, the player should be immediately referred to a qualified healthcare professional who is trained in evaluating and treating concussions.
4. **Rest:** Players should rest from exercise until symptom free and then start a GRTP program; WR recommends minimum rest periods for different ages (U/6 to U/18: 2 weeks minimum rest; adults: 1 week minimum rest).
5. **Recover:** Full recovery from concussion is required before RTP is authorized; this includes being symptom free. Rest and specific treatment options are critical for the health of an injured participant.
6. **Return:** For safe RTP, an athlete must be symptom-free and should have cleared in writing by a qualified healthcare professional who is trained in evaluating and treating concussions. The athlete completes the GRTP protocol.

## Spinal Cord Injury (SCI)

- Result from a combination of distraction and compression forces in either flexion or extension depending on scenario
- The tackle is the area with the highest risk.
- The scrum was previously the greatest area of risk, but rule changes have reduced both the number of scrums and the risk.
- Intervention programs have been internationally successful in reducing the incidence of SCIs; these include BokSmart (South Africa), RugbySmart (New Zealand), and May Day Safety Procedure (Australia)
- Recommended organogram for management of SCIs in rugby is illustrated in Fig. 68.2.

## Musculoskeletal Injuries

- Injury incidence is higher in professional rugby than academy and school rugby.
- Match incidence is higher than training incidence (2/1000 player hours) and has been reported as 91/1000 player hours in professional rugby 47/1000 player hours for academy rugby, and 35/1000 player hours for school rugby.
- Injuries differ according to gender: females have up to 5 times the incidence of anterior cruciate ligament (ACL) injuries, while male rugby players suffer more shoulder girdle injuries and fractures.
- Knee injuries account for the highest number of days absent due to injury and longest time out followed by upper-limb fractures and dislocations.
- While medial collateral ligament injuries are more common, ACL injuries cause greatest number of days missed.
- Shoulder injuries are associated with acute and repeated tackling causing SLAP lesions, Bankart injuries, and rotator cuff tears. The acromioclavicular joint is not only the most common shoulder injury but one of the most common injuries seen in rugby.

## WOUND MANAGEMENT

- WR emphasizes strong preventative measures against the spread of blood-borne diseases.
- Players with open or bleeding wounds must leave the field and have a 15-minute "blood bin" to control bleeding and to cover the wound.
- Players may not wear clothing contaminated by blood.

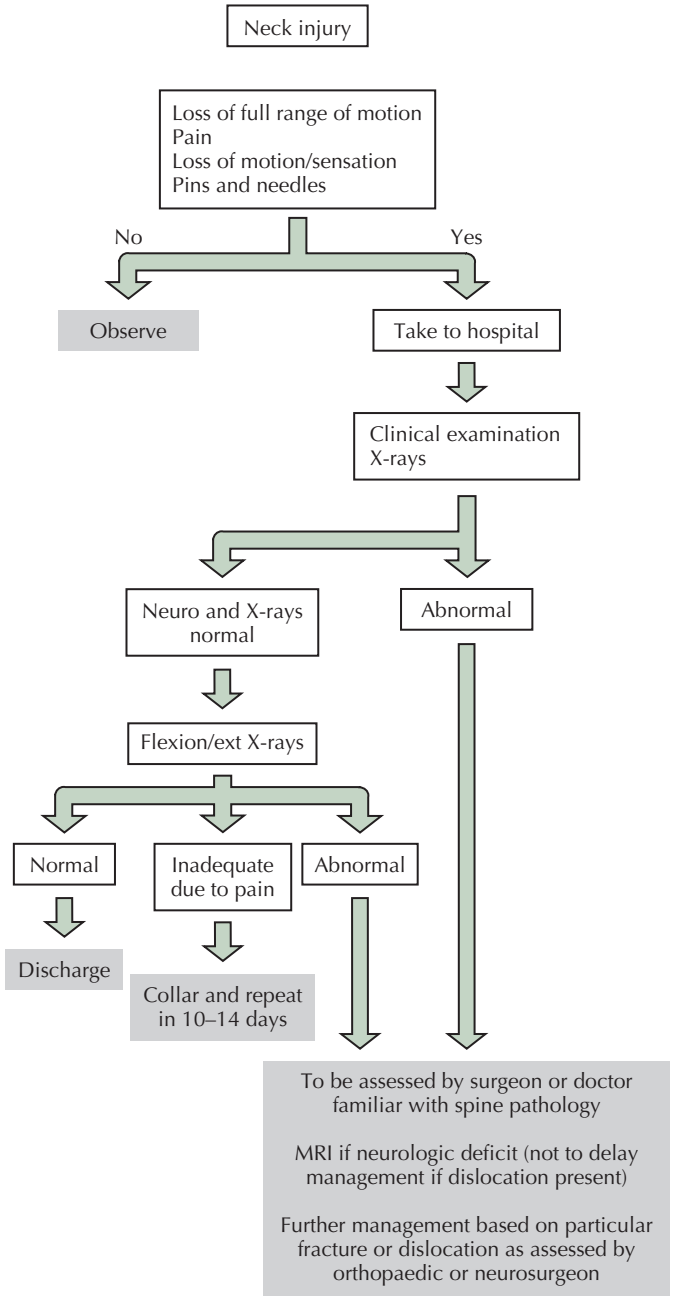



**Figure 68.2.** An approach to managing neck injuries in community rugby. (From Dunn R. BokSmart: medical management of suspected serious acute spinal cord injuries in rugby players. *SAJSM.* 2009;21[3], 91-96.)

## ILLNESS IN RUGBY

- Incidence of illness in rugby is higher than that of injury, particularly when frequent travel is involved.
- Most frequently affected are the respiratory (31%), gastrointestinal (28%), and dermatologic systems and subcutaneous tissues (23%)

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# LACROSSE

# 69

Eugene Hong • Ariel Nassim • Thomas Trojian

## INTRODUCTION

- US Lacrosse is the governing body of American lacrosse.
- According to the number of participants, lacrosse is in the top three fastest growing sports in high school and college.
- Approximately 680,000 athletes played lacrosse in 2011, a 40% increase since 2006 and a 200% increase since 2001.
- In total, 50% of lacrosse athletes are under the age of 16 years; hence, the continued growth of lacrosse is expected.

## GENERAL PRINCIPLES

### Men's Lacrosse

- Men's lacrosse is a contact and collision sport with hitting allowed by the body and stick.
- In boy's lacrosse, stick checks are permitted at the U11 (age 9–10 years), U13 (age 11–12 years), and U15 (age 13–14 years) levels. Body checks are allowed at the U13 and U15 levels.

### Women's Lacrosse

- Women's lacrosse is a noncontact sport, although controlled checking with the stick is allowed and incidental contact does occur.
  - In women's lacrosse, intentional body contact is not allowed.
- There is a theoretical 7-inch "bubble" around the head that cannot be invaded by the opponent. Stick handling is not permitted in this "bubble."
- The women's stick pocket is shallower, allowing easier dislodging of the ball by an opponent and potential differences in ball velocity as it is passed or shot on goal.

### Protective Equipment

- Men's lacrosse equipment protects against trauma caused by the stick, ball, and body contact.
- In men's lacrosse, field position players are required to wear a mouth guard; the National Operating Committee on Standards for Athletic Equipment (NOCSAE) has approved a helmet with a full face mask and a four-point chin strap, gloves, and arm, elbow, and shoulder pads (Fig. 69.1).
  - In 2014, US Lacrosse mandated rule changes that required young male players to wear protective cups.
  - Goalkeepers are required to wear arm pads, throat and chest protectors, and an athletic cup (Fig. 69.2)
- In women's lacrosse, goalies are required to wear a helmet with a full face mask, throat protector, and chest protector. Field position players must wear a mouth guard and an eye protector (Fig. 69.3).
- In 2014, the three rule-making bodies for lacrosse play in the United States—the National Collegiate Athletic Association (NCAA), US Lacrosse, and the National Federation of State High School Associations (NFSHA)— mandated the use of a ball meeting the NOCSAE standard.

### Rules Related to Safety and Injury Risk Reduction

- Men's lacrosse has both technical and personal fouls, which may result in 1- to 3-minute penalties (removing the offending player from the game temporarily creating a "man down" situation), loss of possession, and even ejection.
- Women's lacrosse has minor and major fouls, which result in free position and possible loss of possession.
  - US Lacrosse has planned implementation for 2016 of a new foul for dangerous contact in girls' high school and youth lacrosse.
    - Dangerous contact is defined as any action that thrusts or shoves any player, with or without the ball, who is in a defenseless position.
- The NFSHA published rule changes for the 2016 boys lacrosse season, with the following points of emphasis:
  - Penalties for excessive contact to the head/neck area and hits on defenseless players
  - Appropriate helmet fitting
  - Final 3 minutes of halftime to be used as a warm up to help with injury prevention
  - Safety equipment use during pregame and practice
  - Discouraging field players from defending the crease because they do not have the same degree of protective equipment as goalies to defend shots on goal
- These NFSHA rule changes add to previous years' rule changes prohibiting excessive body checks, minimum penalties for checks involving the head/neck, and emphasis on prohibition of slashing.
- NCAA rule changes, aimed to reduce head injuries, implemented in 2014 include increased, and nonreleasable penalty time for offending players who target an opponent's head or neck by initiating contact with a stick or any part of the body.

### Epidemiology

- The NCAA Injury Surveillance System collects data on collegiate lacrosse injuries.
  - In 2009, men's lacrosse had practice and game injury rates of 4.7 and 16.4 per 1000 athlete exposures (AEs), respectively.
  - In 2009, women's lacrosse had 2.8 and 6.8 per 1000 AEs for practice and game injury rates, respectively.
- Data published in 2014 on high school lacrosse injury rates showed a rate of 1.96 per 1000 AEs. Injury rates were higher during competition than during practice (3.61 vs. 1.23 per 1000 AEs, respectively)
  - Boy's had a higher rate than girls (2.26 vs. 1.54 per 1000 AEs)
  - Common injuries among both boys and girls were strains/sprains (boys: 35.6%; girls: 43.9%) and concussions (boys: 21.9%; girls: 22.7%).
- Top three collegiate game injuries for men's and women's lacrosse are ankle ligament sprains, knee internal derangement, and concussion.
- Most common collegiate practice injuries are ankle ligament sprains, muscle–tendon strains, and internal knee derangements.
- Although less common, upper extremity injuries such as hand or finger fractures are also significant, particularly in the women's game.

## SELECTED INJURIES AND MEDICAL PROBLEMS

### Head and Neck

- Above-the-neck injuries account for one-fifth of all game-related injuries in women's lacrosse.
  - Although rare, females may have a higher incidence of facial fractures, possibly attributable to the lack of any required protective head equipment for field players.

**Figure 69.1.** Men's lacrosse. (Courtesy of Sideline Photos, LLC.)

**Figure 69.2.** Men's goalkeeper. (Courtesy of Sideline Photos, LLC.)

**Figure 69.3.** Women's lacrosse. (Courtesy of Sideline Photos, LLC.)

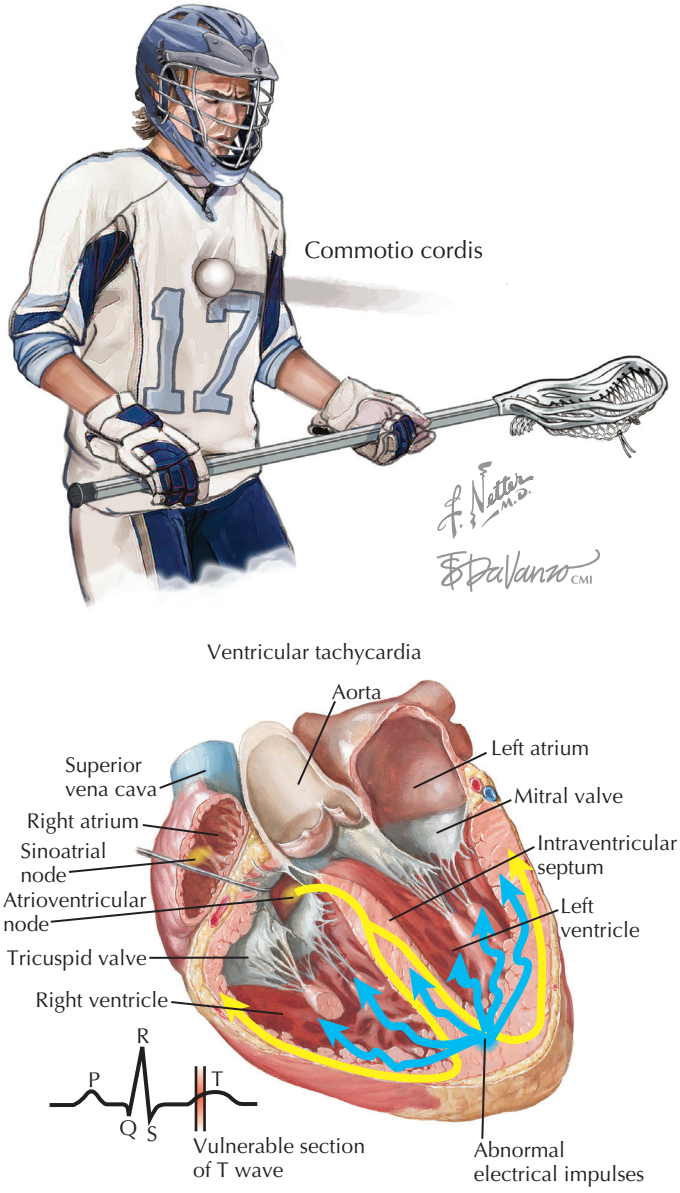


**Figure 69.4.** Mechanism of sudden cardiac death in commotio cordis.

- Sports-related concussions are not uncommon in lacrosse.
  - College concussion rates in competition: men 2.5/1000 AEs and women 1.2/1000 AEs
  - In high school lacrosse:
    - Concussion rates for boy's lacrosse are 0.4/1000 AEs, making lacrosse the next highest concussion rate after football and hockey.
    - Concussion rates for girl's lacrosse are 0.35/1000 AEs, placing girl's lacrosse ahead of girls' soccer (0.34/1000 AEs) for concussion rate.
    - 70% of all concussions occur with player-to-player contact.
    - 40% of concussions resolve within 1–3 days.
    - 55% return to play in 1–3 weeks.
- The potential for cervical spine injuries is concerning in all contact and collision sports.
  - Lacrosse helmet design is different from that used in football.
  - The decision to remove a player's helmet and shoulder pads may be made on a case-by-case basis.
  - The facemask alone may be removed using appropriate tools.
  - Emergency preparedness by medical staff can include face-mask removal, helmet and shoulder pad removal, and spine boarding.

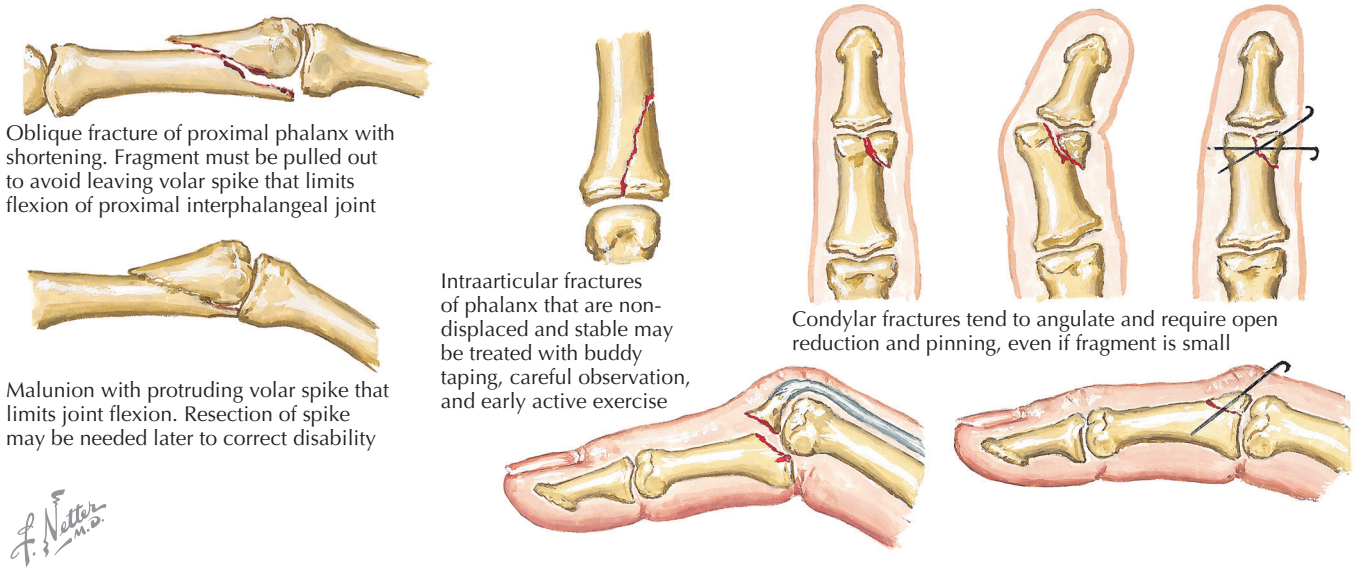


**Figure 69.5.** Fracture of middle and proximal phalanges.

## Eye

- In 2004, protective eyewear was mandated for women's lacrosse at the college level—high school and youth levels followed suit in subsequent years—to reduce the risk of catastrophic eye injury.
  - There was an 84% reduction in serious eye injuries after the mandated protective eyewear rule change.

## Chest

- Commotio cordis is a cause of sudden cardiac arrest (SCA).
  - May occur in sports where a projectile is used
  - Commotio cordis can occur when the ball impacts over the cardiac silhouette in a specific phase of the cardiac cycle (Fig. 69.4).
    - The impact causes an electrical disruption resulting in an abnormal rhythm (most commonly ventricular fibrillation).
    - Animal studies demonstrate that an impact at a speed >40 miles/hour is more likely to induce an arrhythmia.
    - Harder lacrosse balls have a higher incidence of inducing arrhythmia based on animal studies.
  - If an athlete sustains a blow to the chest and collapses, the emergency action plan should be immediately initiated, emphasizing early access to defibrillation.
  - Commotio cordis may have a high mortality rate even with early cardiopulmonary resuscitation.
    - Mortality rate estimated at 16%
  - According to an animal model, the survival rate is 90% if defibrillation is performed within 2 minutes; however, it drops to 40% if defibrillation occurs after 4 minutes.
  - Chest protector use has not demonstrated a preventive effect against commotio cordis.
  - Education is key to prevention; athletes should be discouraged from attempting to block shots with their chest.

## Hand

- 59% of hand injuries in men's lacrosse are thumb injuries. Thumb injuries are higher in lacrosse than in other gloved, stick-handling sports.
- Lacrosse goalkeeper's thumb is described as a fracture of the distal or proximal phalanx of the thumb.
  - Typical mechanism is ball impact on the thumb tip as the goalie attempts to save a shot on goal. An axial load across the interphalangeal joint (IPJ), causes a fracture of the distal or proximal phalanx (Fig. 69.5).
  - Injury is managed according to standard fracture management protocols:
    - If >2-mm displacement, or if >25% of joint surface involvement, open reduction with internal fixation is considered.
    - Nondisplaced fracture with minimal articular surface involvement can usually be treated with immobilization and protection.
  - Injury risk may be reduced by using a molded, hard, protective splint or shell within the goalie's gloves (PHOTO: Lacrosse Glove Images).

## Lower Extremity

- As with many explosive running and cutting type sports, strained hamstrings, anterior cruciate ligament (ACL) tears, and ankle sprains are common lower limb injuries in lacrosse.
- ACL injury rates for both men's and women's lacrosse place them in the middle third of 16 surveyed NCAA sports.
  - Men's lacrosse has an ACL injury rate of 0.12/1000 AEs (rank 10th), whereas women's lacrosse has 0.17/1000 AEs (rank 11th).
  - Decreased knee flexion angle during landing, consistent with sport-specific playing postures, may contribute to the higher incidence of ACL injury in female lacrosse players relative to field hockey.
  - See Chapter 55 (Knee Injuries)
- For ankle sprains, similar injury rates are noted in the NCAA.
  - Women's lacrosse ranks 9th and men's 8th, with injury rates of 0.70 and 0.66 per 1000 AEs, respectively.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- Basics of the game

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

### Basics of the Game

- Lacrosse derives its name from the netted stick, which resembles a crosier, a staff with a hook-shaped curve at one end used by religious figures.
- Lacrosse is played on a rectangular field with nets at opposing ends.
- The game begins with a face-off at midfield.
- Once possession of the ball is gained, the ball is passed, caught, and carried in the netted stick.
- The object is to throw the ball into the opposing team's goal, scoring one point.
- In men's lacrosse, there are 10 players per side: three defensemen, three midfielders, three attackmen, and one goalie.
- In women's lacrosse there are 12 players per side: one goalie, four attackers, four defenders, and three midfielders with similar functions as in the men's game.
- Defensive players may attempt to intercept passes and dislodge the ball from the stick.

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# 70 FIELD HOCKEY

Andrew Reisman • Chaney G. Stewman

## GENERAL PRINCIPLES

### History

- Oldest known stick and ball game in history, existing in the BC era
- Became a popular British sport in 1861, entering as an outdoor Olympic sport for males in 1908 and for females in 1980
- Primarily an outdoor sport, indoor field hockey (FH) is becoming more popular, with an Indoor Hockey World Cup in place since 2003. While both bear many similarities, this chapter is specific to outdoor FH.

### Equipment

- **Stick:** Consists of a handle and "J-" or "U-" shaped curved head; flat only on the left-hand side; maximum length of 41 inches; made of composite materials
- **Ball:** Spherical, hard, and smooth (indentations permitted); circumference between $8^{13}/_{16}$ and $9\frac{1}{4}$ inches; weight between $5\frac{1}{2}$ and $5\frac{3}{4}$ ounces (slightly larger than a baseball)
- **Goal cages:** 7 feet high, 12 feet wide, and 4 feet deep; consists of posts and surrounding nets; 18-inch high side- and backboards are positioned inside the bottom
- **Outdoor field/pitch:** Artificial turf or grass; measures 100 yards long by 60 yards wide; divided by a centerline and 25 yard lines on each side; "circles" are marked 16 yards from each goalpost. Penalty spots are marked 7 yards from the center of each goal-line. Artificial turf is water- or sand-based for player safety (allowing slide with stop) and improved flow of game (providing a smooth, faster surface).
- **Goalkeeper:** Footwear, kickers, leg guards, and headgear with fixed full-face protection and circumferential cover (international); throat, chest, and hand protectors (college); and wrap around the throat protector and attached mouth guard (high school [HS]) (Fig. 70.1)
- **Field players:** Footwear, shin guards, mouth guards (international and college), and eye protectors (HS); flat, conforming face masks may be worn for defending penalty corners. NCAA players may wear soft-covered frame goggles with a plastic (not caged) frame. Soft headgear may be worn at all levels. Left-handed foam padded gloves are encouraged but not mandatory.

### Basic Rules of the Game

- Two 35- (international and collegiate) or 30-(HS) minute halves, with a 5- (international) or 10-minute (collegiate and HS) half-time officiated by two umpires
- Up to 11 players per team, including a goalkeeper, whose presence is only required in HS. A minimum number of seven players are required in HS. Tied games in the NCAA go into overtime with each team consisting of seven players including a goalkeeper.
- The ball can be touched only with the flat side of the stick and passed or dribbled down the field. The ball may be played above the shoulder as long as it is blocked to the ground and not hit in mid-air (Fig. 70.2).
- All players must have an equal chance to play the ball and may not shield the ball with their body or stick. Players cannot intentionally come into physical contact with an opponent's body or stick. Players may not intentionally raise the ball off of the ground in a manner deemed dangerous to surrounding players.
- No offside rule, and self-pass is allowed to restart play.
- Penalties/fouls occur only when a player or team has been disadvantaged by the opponent breaking the rules. Penalties include a free hit, penalty corner, and penalty stroke (Fig. 70.3).
- A goal is scored when the ball is played within the circle and crosses the goal-line.

### Injury Epidemiology

- Injury patterns found in FH are similar to other field based sports, except for a higher rate of hand/finger and head/face injuries compared to other stick-held sports.
  - The overall injury rate in 2004–2009 NCAA field hockey was 6.3 per 1000 AE (injuries per 1000 athletic exposures).
- A majority of injuries occur during competition (2/3) and most occur within the circle (1/2).
  - Competition: 9.8 per 1000 AE
  - Practice: 5.1 per 1000 AE
- Major international FH tournaments injury data demonstrate differences amongst women's and men's competition.
  - Women's: 0.7 injuries per match
    - Head and face injuries are most common (40%–50%).
    - Most common mechanism of injury is an elevated ball (52%).
  - Men's: 1.2 injuries per match
    - Head/face and lower extremity injury rates are equal (22%–28%).
    - Most common mechanism of injury is an elevated ball (37%).
      - Stick contact and player collisions also significantly contribute (25% and 23%, respectively) to injury.
- NCAA injury data from 2004–2009 demonstrate:
  - 53% involve lower limb.
  - 13% involve upper limb.
  - 13% involve torso and pelvis.
  - 9% involve head, face and neck.
  - 6% are concussions.
- NCAA 2009–2014 female sports-related concussion data indicated lower overall rates of concussion (4.2/10,000) compared with ice hockey, soccer, lacrosse, and basketball.
  - FH, however, had the highest overall rate of recurrent concussions in women's sports (13.3%).
- National Center for Catastrophic Sport Injury Research 1982–2013 data demonstrate:
  - One nonfatal catastrophic event at the college level
  - Three direct nonfatal catastrophic events at the HS level
    - One indirect fatality in 2007
  - No direct or indirect catastrophic injuries at either level since 2008
- Injuries by playing position:
  - Goalies have highest injury rates of 0.58 injuries/athlete year.
  - Midfielders have 0.46 injuries/athlete year.
  - Forwards and defenders have equal rates at 0.36 injuries/athlete year.

**Figure 70.1.** Players during competitions exemplifying protective gear worn by the goalkeeper and field player. (Courtesy of Mark Campbell, University of Delaware Athletics.)

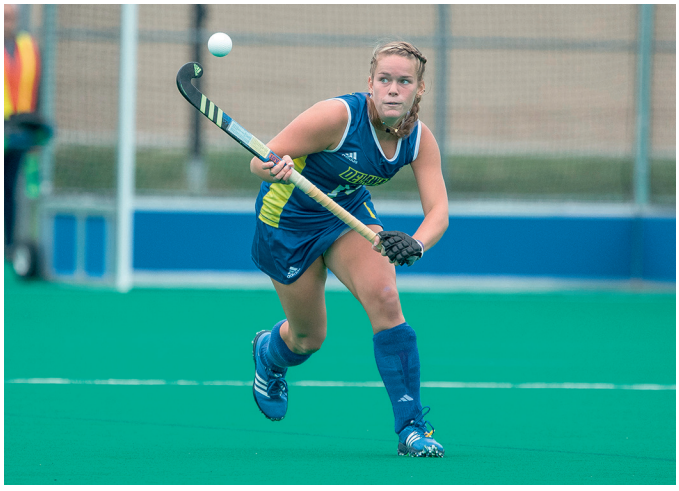

**Figure 70.2.** Permissible playing of a high ball being controlled to the ground. (Courtesy of Mark Campbell, University of Delaware Athletics.)

**Figure 70.3.** Defensive team flying out from the goal crease during penalty corners with protective equipment worn by the goalkeeper and field players. (Courtesy of Mark Campbell, University of Delaware Athletics.)

  - Defenders have higher rates of lower limb injuries.
  - Forwards have higher rates of head and face injuries.
- Activity at time of injury:
  - General field play 45.6%
  - Defending 22.5 %
  - Ball handling 7%
  - Blocking a shot 5.6%
  - Goaltending 4.2%
- Time loss
  - A majority of injuries (32.2%) cause 3–6 days of time loss.
  - 13.1% of injuries result in ≥21 days of time loss.

## SPORTS-SPECIFIC INJURIES

### Lower Extremity

- Over half of injuries involve the lower extremities (53.2%) and have multiple causes.
- Overall, ankle sprains are the most common FH-related injury.
- Contusions and abrasions are frequent from contact with the ball, stick, and field.
  - Injury data comparing grass to artificial-turf injuries are limited but not significantly different.
- Exercise-related leg pain (ERLP), encompassing lower-extremity overuse injuries, including medial tibial stress syndrome, chronic exertional compartment syndrome, stress fractures, and tendinopathies, is a common complaint.
  - A typical midfielder runs 5.6 miles (9 km) per game.
  - Rigorous conditioning for all players is necessary to keep up with the demands of this fast paced sport.
- Incidence of noncontact first-time ACL injury at the college and HS levels is lower compared to women's soccer (according to a single-state-wide epidemiologic study between 2008 and 2012).
  - College injury rate per 1000 person-days: 0.391 (soccer) and 0.038 (FH)
  - HS injury rate per 1000 person-days: 0.131 (soccer) and 0.048 (FH)

### Torso/Pelvis

- Lower back pain is a common complaint among players. Although commonly experienced, it is rarely an acute injury.
  - The inherent trunk flexion and rotation required to play in a semicrouched, primarily right-handed position, hypothetically places players at an increased risk (Fig. 70.4).

### Hand

- All types of hand injuries to metacarpals, fingers, and thumbs were almost two times higher in FH players compared to men's ice hockey and men's and women's lacrosse based on pooled NCAA data from 1986 to 2002.
- Phalangeal injuries are the most common injury overall to the hand; phalangeal fractures are the most common hand fracture.
  - Foam-padded gloves are frequently worn on the left hand, which is particularly exposed while block tackling (Fig. 70.5).
  - Right hand gloves, while not commonly worn, are theorized to decrease the injury risk to these fingers as they grasp the flat side of the stick close to the ground during play.
- Hand injuries represent 34% of pediatric American FH- and 32% of Australian FH-related injuries resulting in presentation to the emergency department.

### Head/Face

- Lacerations and contusions are the most common injury to the head and face.

**Figure 70.4.** Common forward flexed and axially rotated position noted during different aspects of the game. (Courtesy of Mark Campbell, University of Delaware Athletics.)

**Figure 70.5.** Ball stop by the offensive team at the top of the circle during a penalty corner. (Courtesy of Mark Campbell, University of Delaware Athletics.)

**Figure 70.6.** Stick follow-through after a pass can lead to risk of injury to the head and face area. (Courtesy of Mark Campbell, University of Delaware Athletics.)

  - Represent 21% of pediatric American FH related injuries resulting in presentation to the emergency department
- Facial fractures occur relatively infrequently, representing 13% of head/face injuries in a two-season study of NCAA Division 1 players.
  - A majority (50%) involved the nose
  - Sinus, zygomatic arch, and orbital fractures also occurred.
- Dental injuries denote a small proportion of injuries and are more common in elite players (men more than women) where mouth guard usage is not obligatory.
  - Represent 6% of head/face injuries in a two-season study of NCAA Division 1 players
  - Decreased rates from the 1980s to 2000s as mouth guard usage has increased from 31% to 85% among elite players
- Approximately two-third of injuries to the head and face occur during competition instead of practice, primarily due to contact with the ball (Fig. 70.2 and Fig. 70.6).

## Concussions

- NCAA 2009–2014 sports-related concussion data showed a low overall rate of concussions in FH (4.2/10,000). However, FH had the highest recurrent concussion rate in women's sports (13.3%).
  - Two-thirds occurred during competition.
  - Most involve player-to-player contact (53.3%) or are secondary to stick contact (20%).
- Soft molded head gear is allowed for field players, but there is no evidence to show it reduces the concussion incidence.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# ULTIMATE FRISBEE

# 71

Eric Traister

## INTRODUCTION

- A group of students at Columbia High School in Maplewood, NJ, first played Ultimate Frisbee in 1968.
- Commonly referred to as "Ultimate"
- Distinct from disc golf
- Played from the recreational to the international level
- Most commonly played outdoors on grass or turf fields, but variations include indoor and beach Ultimate
- In 2014, there were approximately 4.5 million participants in the United States.

## EPIDEMIOLOGY

- Few studies in the literature regarding Ultimate
- Noncontact sport with a high injury rate
- A previous study reported that among 32 club college teams, Ultimate was second only to Rugby in the number of injuries. Ultimate accounted for 31% of the injuries, while Rugby accounted for 33.8% of the injuries.
- 12.64 injuries per 1100 athlete exposures
- Overall, women may be at a higher risk of injury than men.
- Players are 43% more likely to be injured in a game than in practice.
- The most common injuries are sprains and strains.
- Lower extremity injuries account for up to 67% of injuries, with knee and ankle injuries being the most common.
- Men are more likely to be injured during a layout than women.
- Men are 3 times more likely to suffer a shoulder separation or dislocation.
- Women are 7 times more likely to suffer an ACL injury than men.
- Concussions are prevalent, but incidence is unknown owing to lack of standardized reporting.

## GENERAL PRINCIPLES

### Terminology

- **Backhand:** Most common and basic throw; the throwing arm starts in an adducted position and is brought across the body as the shoulder goes into external rotation. Power and distance are achieved through proper biomechanics transferring power through the hips and shoulder ending with forcefully snapping the wrist upon release of the disc; similar to backhand in tennis (Fig. 71.1A)
- **Forehand**: Second most common throw; also referred to as a "flick"; the shoulder starts in external rotation, elbow flexed, and wrist supinated; most of the power and distance is achieved with the wrist snapping upon release (Fig. 71.1B).
- **Hammer:** Overhead throw with the shoulder adducted and elbow flexed; the disc is released at an oblique angle to the ground. Most of the power and distance is achieved with the wrist snapping upon release (Fig. 71.1C).
- **Layout:** Dive at the end of a run wherein players leave their feet going horizontal to the ground in an attempt to catch or defend the disc; a significant number of injuries occur during a layout due to contact with another player and/or the ground (Fig. 71.1D)
- **Mark:** Person guarding the player with the disc; players have 10 seconds to throw the disc (Fig. 71.1B).
- **Pull**: Starts every point; defensive team throws the disc to the offensive team. Both teams start on their respective goal lines and cannot pass the goal line until the disc is released; similar to a kickoff in football

### Organizations and Levels of Competitions

#### Nationally

- The governing body in the United States is a nonprofit organization called USA Ultimate.
- In 2014, USA Ultimate became a member of the United States Olympic Committee (USOC) as a Recognized Sport Organization.

#### College Division

- College Ultimate is a club sport.
- Over 14,000 student athletes from over 700 colleges compete.
- Student athletes have 5 years of eligibility.
- Men's and women's divisions
- 10 regions with multiple conferences within each region
- Regular season is from January to March (see section Specific Training for competition format).
- Post season is from April to May and includes conference championships, regional championships, and a national championship.
- There is also a championship specifically for Division III colleges.

#### Club Division

- Open to any age group
- Over 700 teams compete.
- Men's, women's, and mixed (co-ed) divisions
- Each division contains 8 regions with "sections" within each region.
- Regular season is from June to August.
- Post season is from August to October and includes sectionals, regionals, and a national championship.

#### Masters Division

- Players must meet specific age requirements to compete in the masters division.
- Age requirement is 33+ for men's and 30+ for women's.
- Grand Masters division is 40+ for men's and 37+ for women's.
- Regional and national championships take place during the summer.

#### Youth Division

- Over 9000 student athletes from over 400 club- and school-based teams
- Includes championships at the high school, under 19, and under 16 levels

#### Professional

- A professional Ultimate league started in 2013 called Major League Ultimate (MLU).
- MLU functions independently of the USA Ultimate.
- Consists of 2 divisions and 8 teams, which compete across the United States

#### International

- The world governing body of Ultimate is the World Flying Disc Federation (WFDF).

**A.** Backhand throw.

**B.** Forehand with an aggressive mark trying to block the throw.

**C.** Hammer throw.

**D.** Layout showing contact between players, disc and ground.

**Figure 71.1.** Ultimate Frisbee position terminology. (Photographs from Ultiphotos.com courtesy of photographers A. Scobel Wiggins at Ultiphotos.com; B. Alex Fraser; C. Kevin Leclaire; and D. Paul Andris.)

- 65 countries on 5 continents represented in the WFDF
- The International Olympic Committee fully recognized the WFDF in 2015.
- Multiple international tournaments that include under 23, club, and world game competitions
- The 2015 World Championships had over 2500 participants with >100 teams representing over 40 countries.

## Equipment

- Only official equipment is a 175-g disc and cleats.
- The 175-g disc is standardized across all competitions except in youth competitions, where a lighter disc is used.

## Rules

- Spirit of the game is the concept of putting sportsmanship and fair play at the forefront of Ultimate.
- Most games are self-officiated, with players settling disputed calls among themselves.
- The highest levels of competition use observers to make rulings on out of bounds calls and goals. They also help settle calls between players if the players cannot reach an agreement.
- An Ultimate field is 40 yards wide, 70 yards from goal line to goal line, and 25-yard deep end zones.
- Teams play 7 versus 7.
- Game is played to a specific number of points or time limit.
- Each point starts with the defense throwing (pulling) the disc to the offense.
- Play is continuous until one team completes a pass (scores) in the defense's end zone.
- The disc can be thrown in any direction at any time. Players have 10 seconds to throw the disc, which is counted by the mark. Running with the disc is not allowed.
- A pass is incomplete when it is dropped, blocked, or intercepted. After an incompletion, possession changes where the defense becomes the offense and tries to score in the opposing team's end zone.
- Substitutions only occur after a goal or for injury.
- No intentional physical contact is allowed. Picks and screens are prohibited. Incidental contact is allowed.

## Specific Training, Physiology Issues, and Unique Environmental Issues

- Most Ultimate competitions are in a tournament format played over 2 days. A total of 6–9 games are played with each game lasting 1–2 hours.
- One study of a men's tournament showed average distance run in a single game is approximately 2.9 miles; approximately 0.4 miles of that is high-intensity running and 0.1 miles is sprinting.
- Extrapolating over the course of a typical tournament, a player could run 17.5–26.3 miles with 2.4–3.6 miles classified as high intensity and 0.8–1.2 involving sprinting.
- Games are played outdoors and only cancelled for lightning. Players must be prepared to play in adverse conditions including, rain, snow, wind, and freezing temperatures.
- Ultimate requires a significant baseline aerobic capacity (similar to soccer) with the capability of making intermittent sprints, jumps, and dives.
- Aerobic training with running, biking, and swimming is common.
- Anaerobic training with high-intensity intervals, plyometrics, and running stairs and hills is common.
- Nutrition is geared for prolonged aerobic activity and quick recovery because tournaments can consist of multiple games played over several days.

# INJURIES AND MEDICAL PROBLEMS

## Skin Abrasions

- Common injury in Ultimate due to layouts; worse abrasions occur on turf fields. Hips, lower abdomen, lateral leg, and forearms are the most common sites.

## Head Injuries

- Concussions typically occur due to contact with another player or the ground during a layout.

## Upper Extremity Injuries

- Shoulder injuries are common during layouts. These include acromioclavicular sprain/separation, glenohumeral subluxation/dislocation, and clavicle fractures.
- Repetitive overhead throws (hammer) can lead to rotator cuff tendinopathy, impingement, and subacromial bursitis.
- Elbow subluxations and dislocations are rare but can occur during a layout.
- Wrist sprains and triangular fibrocartilage (TFCC) injuries are common due to forceful impact of the wrist on the ground during a layout.
- Finger and thumb sprains, contusions, fractures, and dislocations can occur during impact with disc, other players, or ground contact.

## Pelvis and Hip Injuries

- Hip pointers are common due to a player laying out into another player's hip or direct impact with the ground during a layout.
- Constant running with repetitive acceleration, deceleration, sprinting, and change of direction lead to a high rate of hip flexor, quadricep, hamstring, and adductor strains.

### Lower Extremity Injuries

- Sprained ankles are the most common injury seen overall, lateral being the most common.
- Traumatic knee injuries (ligament tears and meniscus injury) are common; mostly occur during a noncontact pivoting or change of direction movement; also occur during impact from a player laying out into another player
- Patellar tendinopathy and patellofemoral syndrome are common owing the amount of running and jumping ultimate players perform.
- Overuse injuries such as medial tibial stress syndrome and stress fractures are common secondary to high volume of running during typical competition.
- Achilles tendon injuries (tendinopathy and ruptures) are common, particularly in the masters division, wherein players will compete well into their 40s.

## SUMMARY

Ultimate is a fast-growing sport both in the United States and internationally. It appeals to athletes of all ages, male and female. There are youth, high school, college, club, masters, and professional competitions. Ultimate is classified as a noncontact sport but has a high injury rate. It requires a significant baseline aerobic capacity, with the ability to do repetitive anaerobic actions such as sprinting, jumping, and diving. Competitions are compacted over 2 days, which also contributes to the high injury rate.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 72 BASKETBALL

Justin Conway • D. Thompson McGuire

## INTRODUCTION

- Physical education teacher James Naismith invented basketball in 1891 as a noncontact sport wherein teams competed to throw a ball into opposing peach baskets.
- Since then, basketball has become increasingly physical with regular contact between players expected, resulting in one of the highest overall injury rates among noncollision sports.

## EPIDEMIOLOGY

- Most basketball injuries are sustained in the lower extremity with contact mechanisms accounting for a majority of acute injuries (Table 72.1).
- The five most common injuries in pediatric ED visits are ankle sprains, finger sprains, finger fractures, knee sprains, and facial lacerations.
- US high school basketball players are over two times more likely to sustain an injury in practice than in game.

## GENERAL PRINCIPLES

### Biomechanical Principles

- Basketball requires a high degree of vertical movements, requiring 35–46 jumping and landing activities per game, over twice those of soccer or volleyball.
  - A combination of both single- and two-footed jumps and landings
  - A high demand of lateral movement and shuffling, particularly while defending
- Recent rule changes at the professional and collegiate level including shortening the shot clock and implementation of the charge circle have led to a faster pace of play, which places higher demands on individual participants.

### Equipment and Safety Issues

- Sudden cardiac death (see Chapter 35)
  - Male basketball players have the highest incidence of sudden cardiac death among all NCAA athletes, with black males at a higher risk than white males
  - An emergency action plan must be in place, including readily available AED and early defibrillation.
- A high rate of facial injuries in basketball including eye, oral, dental, and nasal trauma (see Chapter 47, Eye Injuries in Sports and Chapter 48, Maxillofacial Injuries)
  - Facial protective equipment, including protective goggles and mouth guards, are used by some but are not required.
  - A hard protective face mask recommended for at least 2–3 weeks after a nasal fracture.

## COMMON INJURIES

### Concussions (See Chapter 45, Head Injuries)

- The third most common form of basketball injury at the NCAA level (see Table 72.1)
- Immediate evaluation may require escorting the player to the locker room owing to close proximity of the sideline to the court.
- Do not return a symptomatic player to the competition.
- Follow the return-to-play protocol as per internationally recognized guidelines.

### Finger Injuries

Frequently caused by direct trauma from the ball, opponent, or rim of the basket

#### "Jammed Finger"—Collateral Ligament Injury

**Mechanism:** Forced ulnar or radial deviation of finger, resulting in partial or complete tear of collateral ligaments; most often PIP

**History/examination:** Tenderness at the involved joint; check for stability with varus and valgus stress testing with the involved joint at 30 degrees of flexion; ensure that the MCP is flexed to 90 degrees because an extended MCP will tighten collateral ligaments.

**Diagnostic considerations:** Obtain radiographs if concerns for fracture

**Treatment:** Stable—buddy tape to adjacent finger, may return to play; Unstable—refer to hand surgeon

#### Jersey Finger

**Mechanism:** Forced extension of the DIP joint during active flexion causing rupture of flexor digitorum profundus tendon (e.g., finger caught on opponent's jersey or rim of basket); most commonly the fourth finger because of relative flexor weakness (Fig. 72.1)

**History/examination:** Injured finger extended with hand at rest; palpate for retracted flexor tendon; patient unable to actively flex at involved DIP joint

**Treatment:** Immediate referral to a hand surgeon

### Knee Injuries

Knee injuries are the second most common basketball-related injury after ankle sprain (see Table 72.1).

#### Anterior Cruciate Ligament Injuries (See Chapter 49, Knee Injuries)

- ACL tears are up to 4 times more common in female than male basketball players at all levels of competition
- Among US high school athletes, basketball has the second highest incidence of ACL tears in female athletes (after soccer) and the fourth highest in males (after football, lacrosse, and soccer).
- Preventive neuromuscular training programs have proven effective in reducing rates of ACL tear.

#### Patellar Tendinopathy ("Jumper's Knee")

**Mechanism:** Repetitive vertical jumping and squatting (defensive stance) required in basketball puts a high degree of stress on the patellofemoral joint and quad/patellar tendon. Biomechanical risk factors include tight quadriceps and hamstrings.

**History/examination:** Anterior knee pain with squatting and resisted knee extension; most commonly tender at proximal

**TABLE 72.1 EPIDEMIOLOGY OF BASKETBALL INJURIES IN THE UNITED STATES**

| | NCAA Women | NCAA Men | WNBA | NBA |
|---|---|---|---|---|
| **Rate of Injury (AE = Athlete Exposure) G = Game P = Practice** | G: 7.68/1000 AEs<br>Practice: 3.99/1000 AEs | G: 9.9/1000 AEs<br>P: 4.3/1000 AEs | G: 24.9/1000 AEs | G: 19.3/1000 AEs |
| **Common Injuries (% of Total Injuries)** | Ankle ligament sprains (24.6% G, 23.6% P)<br>Knee internal derangements (15.9% G, 9.3% P)<br>Concussion (6.5% G, 3.7% P) | Ankle ligament sprains (26.2% G, 26.8% P)<br>Knee internal derangements (7.2% G, 6.2% P)<br>Concussion (3.6% G, 3.0% P) | 65% NBA injuries and 66% WNBA injuries to lower extremity<br>NBA injuries only: lateral ankle sprain (13.2%), patellofemoral inflammation (11.9%), lumbar strain (7.9%), hamstring strain (3.3%) | |
| **Mechanism of Injury** | Player contact (46% G, 31% P)<br>Other contact—balls, standards, ground (24% G, 18% P)<br>No contact (29% G, 47% P) | Player contact (52% G, 44% P)<br>Other contact—balls, standards, ground (24% G, 18% P)<br>No contact (22% G, 36% P) | Overuse and inflammatory conditions (tendinitis, bursitis, synovitis) accounted for greatest amount of time lost from practices and games in both leagues (22% NBA, 27% WNBA) | |

Data from Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association injury surveillance system, 1988-1989 through 2003-2004. *J Athl Train*. 2007;42(2):202-210; Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med*. 2006;34(7):1077-1083; Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2003-2004. *J Athl Train*. 2007;42(2):194-201; Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2(4):284-290.

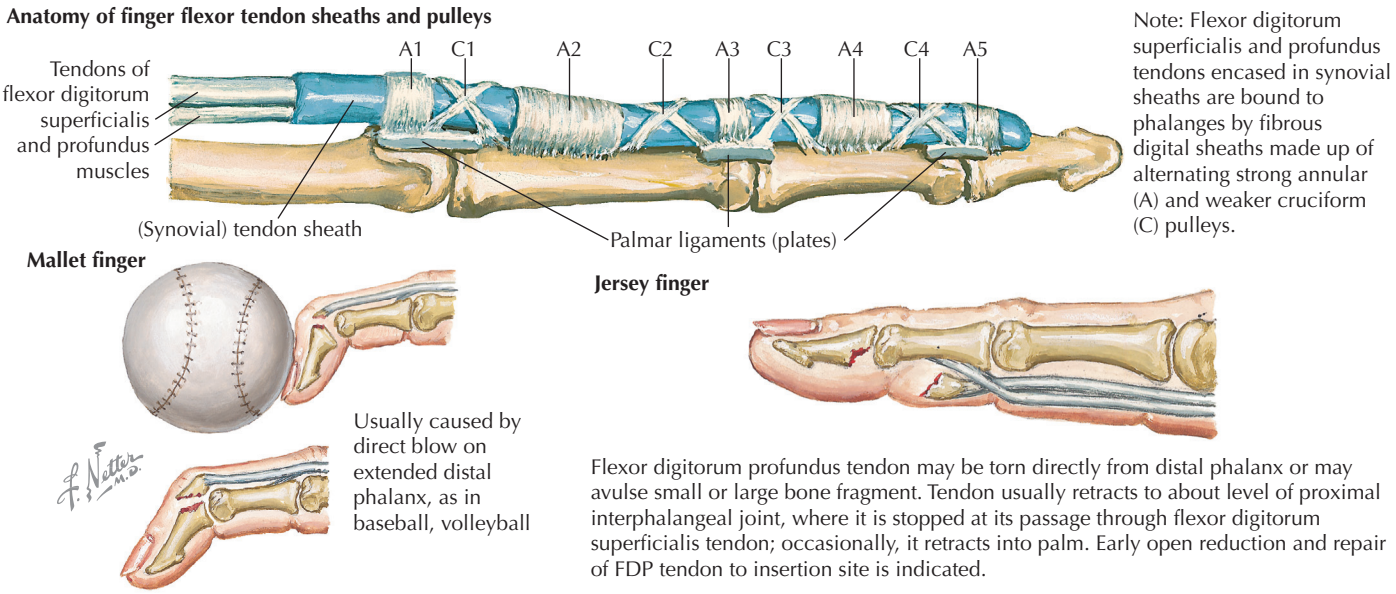



**Figure 72.1.** Finger anatomy and injuries.

origin of tendon on patella; differential in skeletally immature athletes includes Osgood–Schlatter disease (tibial tubercle apophysitis, see Fig. 72.2)

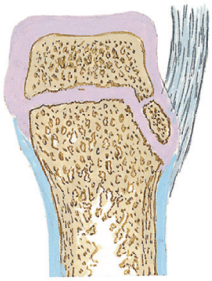
**Diagnostic considerations:** Differentiate from patellofemoral pain syndrome as both are common in basketball players (may occur together); Imaging:

- Well characterized by ultrasound (tendon thickening, intrasubstance tears, calcifications, and increased vascularity)
- Consider MRI for recalcitrant cases

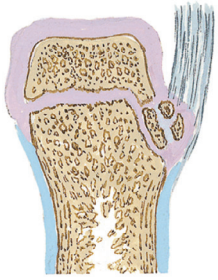
**Treatment:**

- Ice, rest, NSAIDs, and activity modification
- Taping or bracing (e.g., Cho-Pat strap) to offload the patellar tendon
- Quad/hamstring stretching and eccentric quad-strengthening exercises
- Injections
  - Consider PRP injections or prolotherapy to stimulate tendon repair if conservative management fails

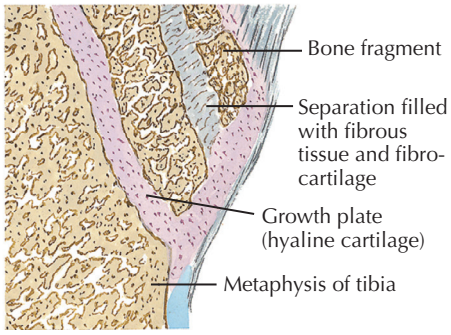
Clinical appearance. Prominence over tibial tuberosity due partly to soft tissue swelling and partly to avulsed fragments



Normal insertion of patellar ligament of ossifying tibial tuberosity

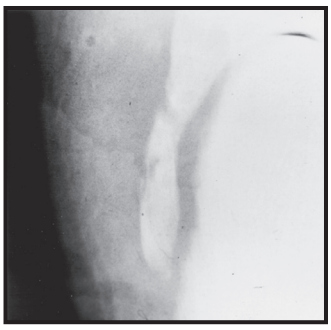


In Osgood-Schlatter disease, superficial portion of tuberosity pulled away, forming separate bone fragments




High-power magnification of involved area

Radiograph shows separation of superficial portion of tibial tuberosity.



Focal radiograph shows fragment at site of insertion of patellar ligament.

**Figure 72.2.** Osgood–Schlatter lesion.

- Corticosteroid injections contraindicated owing to risk of tendon rupture
- Ultrasound-guided percutaneous fenestration versus surgical debridement if no response to aforementioned therapies

## Ankle Injuries

**Description:** Most common site of injury in basketball at all levels (see Chapter 56, Ankle and Leg Injuries)

**Mechanism:** >90% inversion type injury resulting in sprain of lateral ankle ligaments (ATFL, CFL, and PTFL) (Fig. 72.3); common sites of associated injuries include peroneal tendons and fractures of the lateral malleolus or fifth metatarsal. Injuries to deltoid and anterior tibiofibular ligament may also occur. A majority of injuries occur on landing from jump and/or planting on another player's foot.

**Risk factors**: History of ankle sprain and shoes with air cells in heels both found to the increase risk of ankle sprain by >4 times the baseline risk; decreased ankle dorsiflexion strength and range of motion (ROM)

**History/examination**: Examine for swelling, tenderness, and laxity; perform anterior drawer and talar tilt tests. Rule out high ankle sprain with passive ER and syndesmosis squeeze test.

**Diagnostic considerations**: Radiographs to rule out fracture if indicated; consider stress views if concern for syndesmosis injury

**Treatment/return to play**: A vast majority of isolated ankle sprains can be nonsurgically treated. Higher-grade sprains may require a short period of weight-bearing immobilization in a walking boot or air cast. Key aspects of recovery include early mobilization, full ROM, strengthening, proprioceptive training, and peroneal muscle strengthening. Functional bracing or taping to stabilize the ankle when returning to playing basketball.

**Prevention:**

- Incidence of ankle sprains can be decreased by prophylactic ankle bracing or taping.
  - Study of American high school basketball players found that using a lace-up ankle brace reduced the incidence of acute ankle injuries by >3 times regardless of sex, age, level of competition, or BMI.
  - Studies have reported mixed results on efficacy of bracing versus taping.
- Multiple training programs have proven effective in decreasing the rate of ankle sprain in basketball players.
  - A minimum of 20 minutes/week of proprioceptive and balance exercises has been shown to significantly decrease rates of ankle injury.
  - The FIFA 11+ warm-up program significantly reduced overall injury rates in male elite basketball players, including ankle sprains.

### Achilles Tendon Rupture (See Chapter 56, Ankle and Leg Injuries)

**Description:** More commonly occurs in players in their third and fourth decades

**Mechanism:** Sudden, intense, and often unexpected eccentric or concentric contraction of ankle plantar flexors

**History/examination:** Patients may hear or feel a pop in posterior ankle and weakness of ankle plantar flexion. Positive Thompson test: absence of plantarflexion when calf is squeezed

**Diagnostic considerations:** MRI is the imaging choice, although ultrasound may be considered at the point of care.

**Treatment:** Choice of surgical versus nonsurgical management is patient dependent. A lower risk of re-rupture with surgery, but newer studies have suggested long-term differences may not be significant

## Foot Fractures

- Basketball players are at a high risk of foot fractures given the repetitive jumping and pivoting.
- Particularly true at elite levels as height, weight, and physicality of play increase
- Long season versus several other team sports increases the risk of stress fractures.

### Metatarsal Fractures

**Mechanism:** High-impact force on foot, can be acute or chronic (stress) fracture

**History/examination:** Metatarsal pain and focal tenderness to palpation, often with associated soft tissue swelling

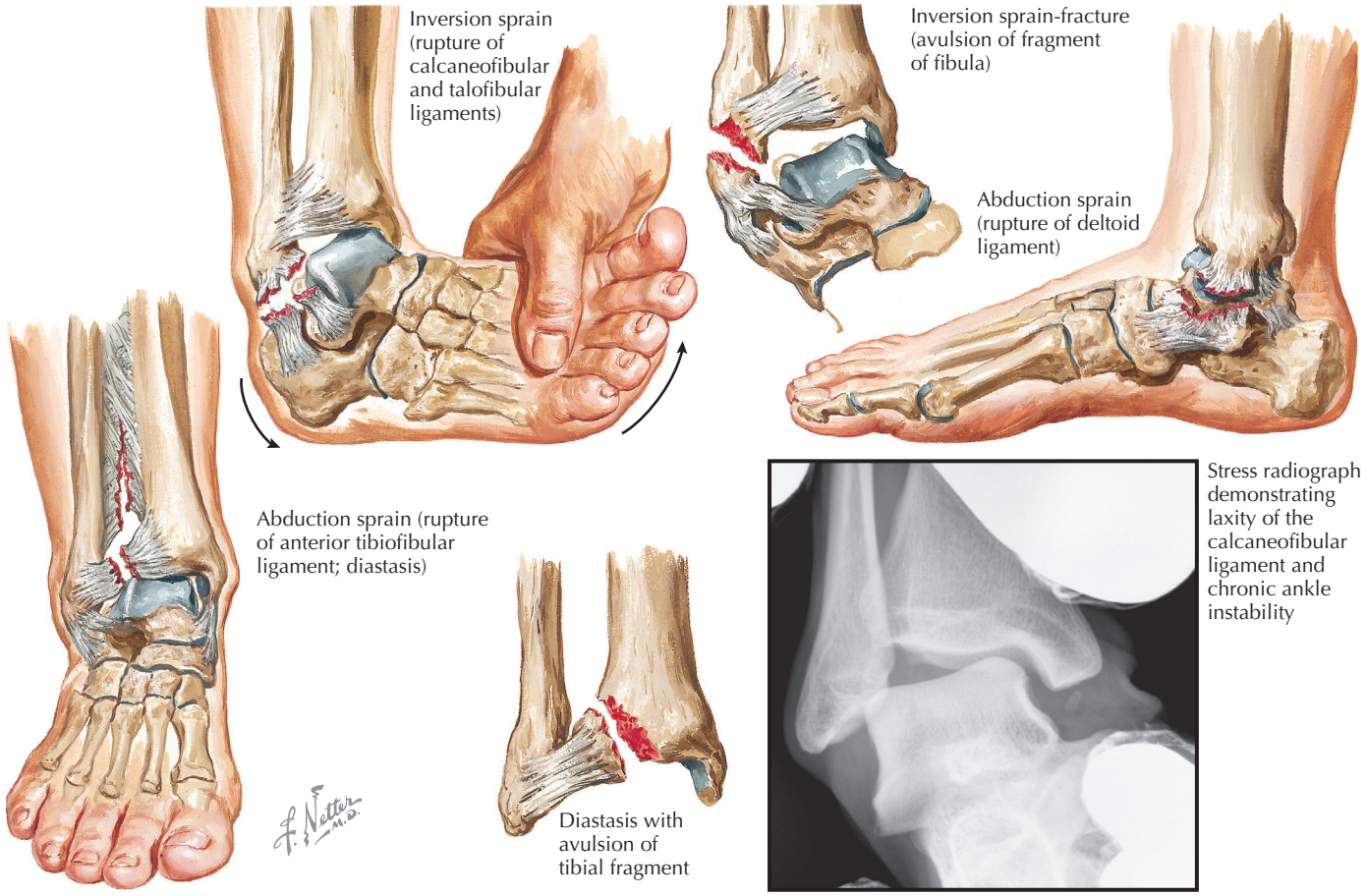



**Figure 72.3.** Ankle sprains and associated fractures.

**Diagnostic considerations:** Consider serial radiographs in suspected metatarsal stress fractures (can avoid more extensive studies as often see evidence for stress injury after 2–3 weeks); check MRI if suspicion for stress injury remains despite negative radiographs.

**Treatment:** Low-risk metatarsal stress fractures are routinely treated nonsurgically with partial weight-bearing immobilization. Low-grade injuries may be amenable to continued game participation with walking boot in between games, depending on the timing of the season.

### Jones Fracture

**Description:** Transverse diaphyseal fracture of the proximal fifth metatarsal; notorious owing to a high risk of nonunion given the vascular watershed area (Fig. 72.4)

**Mechanism:** Commonly an acute fracture caused by an impact on the foot in plantarflexion and/or inversion; may also be site of stress injury

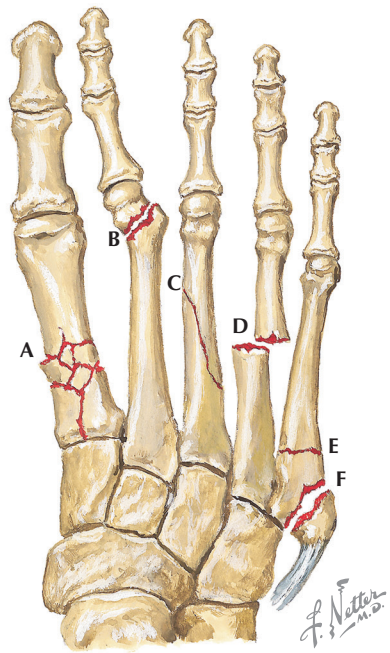
**History/examination:** Pain and tenderness at the base of the fifth metatarsal; check for associated swelling and ecchymosis

**Diagnostic considerations:** Diagnose using radiographs and consider CT if concern for delayed healing or nonunion

**Treatment:** Surgical fixation usually preferred over nonsurgical management, particularly in elite athletes; reduces the risk of nonunion and allows earlier return to weight-bearing. Review of Jones Fractures in NBA players over 19 seasons revealed that 85% of players returned to pre-injury levels of competition in the season following injury.

### Tarsal Navicular Stress Fractures

**Mechanism:** Navicular bone is susceptible to stress injury owing to high forces transmitted during high-impact activities and




**Types of fractures of metatarsal:** A. comminuted fracture, B. displaced neck fracture, C. oblique fracture, D. displaced transverse fracture, E. fracture of base of 5th metatarsal, F. avulsion of tuberosity of 5th metatarsal

**Figure 72.4.** Metatarsal fractures.

avascular watershed area in middle third; a high rate of nonunion

**History/examination:** Medial midfoot pain with tenderness over proximal dorsal portion of the navicular

**Diagnostic considerations:** Often missed diagnosis, a high index of suspicion required; low threshold for MRI or CT scan if radiograph negative

**Treatment:** Requires extended nonweight-bearing immobilization (at least 6 weeks), but surgical fixation is often required, particularly in elite athletes

### Medial Subtalar Dislocation ("Basketball Foot")

**Mechanism:** High impact on the plantarflexed foot, causing dislocation of talocalcaneal and talonavicular joints

**History/examination:** Bony midfoot deformity, foot locked in supination

**Diagnostic considerations:** Radiographs before and after reduction

**Treatment:** Immediate reduction and immobilization; >60% can be managed by closed reduction

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# VOLLEYBALL

# 73

Kevin Eerkes

## INTRODUCTION

- Volleyball is a popular sport played worldwide both recreationally and professionally.
- It requires quick and explosive movements.

## EPIDEMIOLOGY

- Most injuries occur to blockers and hitters as opposed to passers and setters.
  - Certain positions are associated with specific injuries (Table 73.1).
- Most injuries are related to jumping and landing.
  - A player landing from a jump near the net may accidentally come down on an opponent's foot.
- Acute injuries
  - Ankle and finger sprains make up a majority of acute injuries.
  - Most acute injuries occur near the net.
- Overuse injuries
  - More common than acute injuries
  - Patellar tendinitis is the most common.
  - Occur more often in elite players because they spend more time doing drills than recreational players

## GENERAL PRINCIPLES

### Terminology

- Positions (see Table 73.1)
- Serve types
  - Float serve
    - The server immediately retracts the arm after hitting the ball.
    - This causes the ball to "float" through the air because it has no spin, making it hard to return, like a knuckleball.
  - Jump serve
    - The server gets a running start and then jumps high into the air, with the serve taking place at the top of the jump.
    - Gives more velocity to the serve and a sharp downward trajectory
- Dig
  - Pass a ball that was spiked very hard
- Shank
  - When the ball goes in the wrong direction while attempting to pass

### Pertinent History

- Volleyball is a truly American game with origins back to the 1890s.
- Made its debut at the Olympic Games in 1964

### Equipment and Safety Issues

- Players often wear a long sleeve jersey to allow for safer sliding on the court during dives and slides.
- Elbow and knee pads can be worn (Fig. 73.1).
- Padded pants can help prevent contusions to the greater trochanters and iliac crests.
- Sand socks can be worn during beach volleyball to protect the feet.
- Calling the balls (i.e., yelling "mine") helps reduce chance of colliding with another player.

### Rules

- Players are generally not allowed to cross the center line as this can lead to injuries when a player comes down on an opponent's foot. The exception to the rule is doubles and beach volleyball.

## COMMON INJURIES AND MEDICAL ISSUES

### Foot and Ankle Injuries

#### Ankle Sprain

- Most common acute injury in volleyball, accounting for up to half of all acute injuries
- Often occurs when a spiker or blocker lands from a jump around the net on another player's foot
- Prevention
  - Wearing an ankle brace after an ankle sprain has been shown to reduce the incidence of recurrence in volleyball players.
  - Certain evidence suggests that a preseason plyometric exercises, similar to those used to prevent anterior cruciate ligament (ACL) injuries, help prevent ankle sprains.

#### Sand Toe

- Can occur during beach volleyball where shoes are not worn
- Hyperplantarflexion injury to the first metatarsophalangeal joint

### Wrist and Hand Injuries

#### Finger and Thumb Sprains, Fractures, and Dislocations

- When blocking, fingers are spread apart and vulnerable to injury from the ball or the opponent's hand (Fig. 73.2).
- If the fingers are below the ball while the player is attempting to block, they are prone to injury because of the downward trajectory of the ball onto the fingertips.
- Injuries may also occur when the fingers hit the net or another player during the follow-through phase of the swing.
- Injuries to the thumb, index, and middle fingers are particularly bothersome for overhead setters because they use those 3 fingers to set.
- Prevention
  - Appropriate blocking and hitting techniques may decrease the frequency of injury.
  - Keeping the fingers rigid during blocking can also help.

#### Wrist Fractures

- From falling on outstretched hand when losing balance while coming down from a jump or tripping

#### Wrist Tendinitis

- In overhead setters who frequently use wrists

#### de Quervain's Tenosynovitis

- When passing, the ball is supposed to hit the fleshy part of the forearm. If the ball instead repeatedly impacts the radial aspects of the wrists, not only does the ball shank, but tenosynovitis can develop as well.

**TABLE 73.1 INDOOR VOLLEYBALL**

| Position | Location on Court | Function | Injuries |
|---|---|---|---|
| Setter | Front court | Set ball to hitter | Wrist tendinitis<br>Finger injuries |
| Hitter (spiker) | Front court | Spike ball into opposing court | Ankle sprains<br>Shoulder instability/ impingement<br>Spondylolysis<br>Patellar tendinitis |
| Server (all players) | Back court | Serve ball | Shoulder instability/ impingement |
| Blocker | Front court | Block or alter ball hit by opponent | Finger injuries<br>Ankle sprains<br>Patellar tendinitis |
| Passer | Back court | Receive serve<br>Pass ball to setter<br>May need to dive for or "dig" ball | Contusions<br>Injuries of upper extremities<br>Patellofemoral syndrome<br>Low back pain |

**Figure 73.1.** Prevention of injuries. Knee pads can offer protection when digging balls. (Copyright © P. Zivnuska 2016.)

### Neuritis of the Superficial Branch of the Radial Nerve

- From repeated ball impact to the dorsoradial aspect of the distal forearms
- Decreased sensation and paresthesias over the dorsoradial hand
- Treatment involves improving the technique so that the ball impacts the appropriate location more proximal on the forearms.

Blockers are prone to finger injuries from the ball.

**Figure 73.2.** Finger and thumb sprains. (Copyright © P. Zivnuska 2016.)

## Shoulder Problems

### Shoulder Instability

- During the early acceleration phase of hitting and serving, the arm is in extreme extension and external rotation (Fig. 73.3). During these maneuvers, the humeral head is levered anteriorly in the glenoid, which can lead to shoulder pain.
- Rotator cuff impingement and tendinitis may occur.
- Jump serving places higher loads on the shoulder than the traditional overhead serve or float serve.
- Treatment involves strengthening of the rotator cuff.

### Internal Impingement

- When the humeral head translates anteriorly during hitting and serving, the articular side of the rotator cuff and labrum can become impinged at the posterosuperior aspect of the glenohumeral joint.
- Pain at the posterior shoulder and tearing of the cuff and labrum
- Treatment involves strengthening the rotator cuff and stretching the posterior capsule if tight.

### Glenohumeral Internal Rotation Deficit (GIRD)

- Seen in hitters and servers
- Have decreased internal rotation of the shoulder due to contracture of the posterior capsule; this results in obligatory posterosuperior shift in the humeral head during arm elevation.
- Associated with various shoulder problems such as internal impingement, external impingement, and labral tears.
- Treated with stretches of the posterior capsule (sleeper stretches)

Extreme external rotation and extension during hitting can cause shoulder problems due to anterior instability and overuse of the rotator cuff.

**Figure 73.3.** Shoulder injuries. (Copyright © P. Zivnuska 2016.)

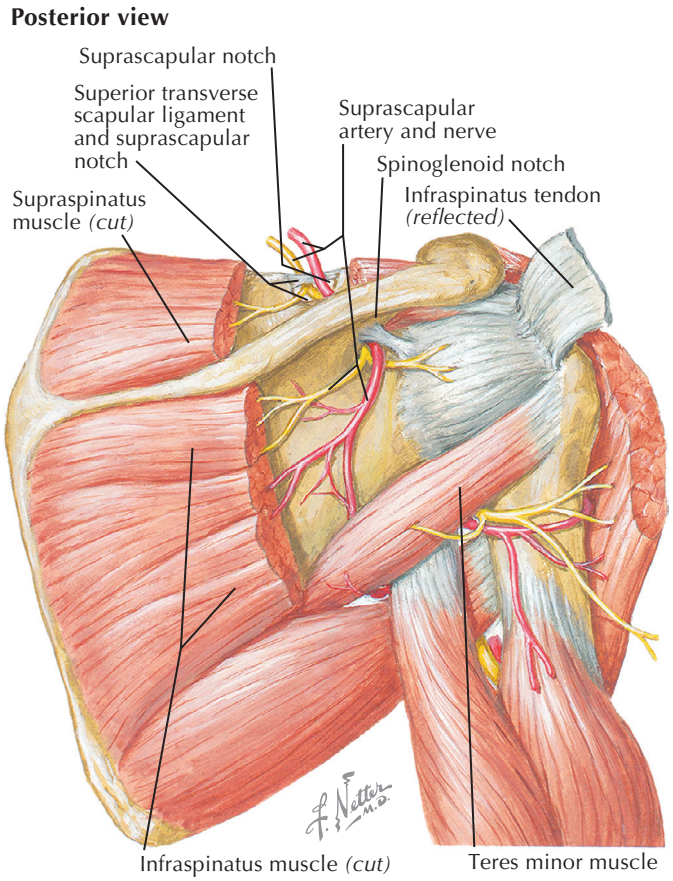


**Figure 73.4.** Suprascapular nerve traversing the spinoglenoid notch, where it can be entrapped.

## Suprascapular Neuropathy

**Description:** Irritation of the suprascapular nerve usually occurs at the suprascapular notch, but in volleyball players, it occurs more distally at the spinoglenoid notch (Fig. 73.4). This leads to atrophy of the infraspinatus muscle and weakness of external rotation.

**Mechanism of injury:** Thought to occur from traction and/or compression of the nerve during the extreme motions of the arm during the cocking or follow-through phases of the arm swing while serving and hitting; traction can also occur during float serves. This technique requires forceful eccentric contraction of the infraspinatus muscle to stop the arm, creating traction on the nerve.

**Incidence:** Occurs in the dominant upper extremity in 12%–30% of high-level volleyball players

**History:** May occasionally may cause posterior shoulder pain but usually asymptomatic and athletes still able to play at a high level; proposed reasons for why it does not usually bother the athlete:

- Nerve lesion is usually incomplete, so certain functions of the muscle remain.
- Teres minor muscle compensates (is innervated by a branch of the axillary nerve)
- External rotation strength is much less important for performance in volleyball than is internal rotation.

**Physical examination:** Varying degrees of atrophy of the infraspinatus muscle; weakness with external rotation; certain athletes may have point tenderness over the spinoglenoid notch

**Diagnostic considerations:** EMG and NCS can usually confirm the diagnosis but not necessary in most cases; if an athlete is not improving and is symptomatic, consider radiographs to check for a bony lesion compressing the nerve (Bennett lesion) or MRI to check a space-occupying lesion (ganglion) compressing the nerve; differential diagnosis of infraspinatus atrophy:

- Infraspinatus tendon tear
- Brachial neuritis affecting the suprascapular nerve

**Treatment** (if symptomatic):

- **Nonsurgical treatment**
  - Restricting overhead hitting
  - Nonsteroidal anti-inflammatory medications
  - Physical therapy to strengthen the rotator cuff and periscapular muscles, improve posture, and stretch posterior capsule, if tight
  - Repeat EMG at 6 months, if performed initially, to assess recovery
  - If patient is not improving or has significant initial disability, consider obtaining an MRI
- **Surgical treatment**
  - Possible indications
    - Significant pain and decreased performance despite 6 months of nonsurgical treatment
    - Space-occupying lesion compressing the nerve
  - Is rarely needed

- Surgical procedure involves exploring the nerve and releasing areas of compression

**Prognosis/return to play:** Most symptomatic cases respond to nonsurgical treatment but may take up to a year for maximal recovery; an exception is a space-occupying lesion compressing the nerve, which often will not resolve without surgery. While pain usually improves with nonsurgical or surgical treatment, muscle atrophy may not resolve. Most athletes are able to return to full function despite the persistent atrophy.

## Lumbosacral Spine Injuries

### Mechanical Low Back Pain

- Passers are susceptible because of repetitive lumbar flexion with arms held in front of the body (Fig. 73.5).
- Jumping positions are also susceptible because repeated landing places high forces on the spine.

### Spondylolysis

- A stress fracture at the pars interarticularis of the vertebra, most commonly of the fifth lumbar vertebra
- Occurs with repetitive hyperextension of the low back, most commonly associated with hitting (Fig. 73.6) but also with overhead setting and blocking

## Knee Injuries

### Patellar Tendinitis (Jumper's Knee)

- Most common overuse injury of the knee in volleyball
  - Affects approximately 50% of elite players
- Due to repetitive eccentric quadriceps contraction with jumping (blockers or hitters)

### Patellofemoral Syndrome

- Particularly common in volleyball positions that require jumping and squatting (hitting, blocking, and passing)

### Anterior Cruciate Ligament (ACL) Tear

- Occurs more frequently in female volleyball players than male
- Reconstruction is usually required if return to a sport such as volleyball is desired.
- Preseason proprioceptive and plyometric training programs can significantly reduce the incidence of ACL tears in women.

### Medial Collateral Ligament Tear

- From valgus force to the knee as a result of landing off balance

## Leg Injuries

### Tibial Stress Fracture

- May be found in positions requiring frequent jumping

### Gastrocnemius Muscle Strain

- Associated with an eccentric load to the muscle tendon unit

### Achilles Tendon Problems

- Acute Achilles rupture
  - Due to rapid, forceful eccentric loading of the tendon when landing from a jump or from sudden acceleration
- Achilles tendinitis

Repetitive flexion of the lumbosacral spine in under hand passing can lead to low back pain.

**Figure 73.5.** Lumbosacral spine injuries. (Copyright © P. Zivnuska 2016.)

**Figure 73.6.** Lumbar hyperextension with hitting.

  - Due to repetitive eccentric loads from jumping
- Achilles tendinosis
  - Degeneration and microtearing of the Achilles tendon at its insertion to calcaneus

### Concussion

- Can occur from impacting the head against the pole, the floor, or being hit by someone's arm

### Heat Illness

- Can occur with beach volleyball during hot conditions

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 74 BASEBALL

William G. Raasch

## INTRODUCTION

### History

- Baseball, the great American pastime, was first described by Abner Doubleday in 1839.
- The game evolved considerably in the early years, but it was with the advent of the overhand pitching motion in the 1880s that shoulder and elbow problems became a familiar part of the game.
- Although ballplayers are subject to usual sports-related strains, sprains, bumps, and bruises, it is in understanding the common shoulder and elbow throwing injuries that the baseball team physician is defined.

### Game Coverage

- A team physician is required at the professional level.
- Physician coverage is not required at the Little League, high school, or college level.
- Occasionally, a college will provide trainer coverage.

## THROWING BIOMECHANICS AND ASSOCIATED PATHOLOGY

- Throwing a baseball is all about the transfer of energy from the body to the ball.
- A smooth transition will maximize ball velocity while reducing the risk of injury.
- Throwing mechanics can be separated into five phases (Fig. 74.1).
  - Because certain body structures are susceptible to injury during each phase of throwing, determining the phase in which the injury or pain occurs may help make a diagnosis.

### Windup

- Varies from pitcher to pitcher
- Description:
  - Begins from the set position with the ball in the pitcher's glove
  - Arms drop and body rotates 90° (see Fig. 74.1)
  - The stride leg is elevated and flexed.
  - Provides rhythm and balance
- Injury risk: minimal

### Stride

- Description:
  - Begins when the supporting leg flexes and the body is lowered
  - The stride leg moves toward the plate.
  - The trunk remains back.
  - The stride length averages 75% of the body height with an offset of 0.4 cm (i.e., the lead foot lands almost directly in front of the back foot).
- Injury risk:
  - Stride too long: The athlete will be unable to rotate the hips, resulting in a loss of velocity. This break in the kinetic chain is known as "arm throwing," and the athlete may compensate by overloading the shoulder, resulting in a possible rotator cuff strain.
  - Stride is off line to the first base side (right-handed pitcher)—the torso will be ahead of the shoulder, resulting in "opening" too soon and stressing the anterior capsule; this may result in shoulder instability.
  - Stride is off line to the third base side (right-handed pitcher)—the pitcher will "throw across his body" with possible labral shearing.
  - Altered stride may also be a sign of hip contracture or hip labral pathology. A physical examination is required to assess the overall range of motion and rule out femoral acetabular impingement.

### Cocking

- Places the arm in maximum external rotation while abducted
- Description:
  - Begins when the stride foot makes contact with the mound
  - Hip rotation begins, followed by the trunk (speed of hip rotation correlates well with ball velocity).
  - Ends when the shoulder reaches maximum external rotation
  - External rotation in a professional pitcher may approach 180°.
- Injury risk:
  - When the shoulder is in maximum external rotation and 90° of abduction, internal impingement may occur: undersurface of the superior rotator cuff impinges against the posterior/superior labrum. This may result in undersurface tearing of the rotator cuff and posterior/superior labrum fraying.
  - Anterior capsule stretching may occur over time and result in increased anterior/inferior laxity and possible glenohumeral instability (see Fig. 74.1).

### Acceleration

- Generates ball velocity
- Description:
  - Begins with initiation of shoulder internal rotation
  - Ends with ball release
- Injury risk:
  - Primary movers and stabilizers of the shoulder are stressed by rapid acceleration.
  - Rotator cuff strain—primary stabilizer
  - Latissimus dorsi and teres major strain—internal rotation and adduction
  - Labral injury if humeral head does not remain centered during rapid acceleration
  - The elbow is placed under significant valgus stress—ulnar collateral ligament (UCL) is under tension while radiocapitellar articulation is under compression. Thus, UCL is at a risk of tearing and radiocapitellar articulation at a risk of osteochondral injury.
  - Valgus extension overload—as elbow extends under valgus load, the posteromedial aspect of the olecranon may impinge against the olecranon fossa, resulting in posteromedial elbow pain. Over time, spurring and degenerative changes may

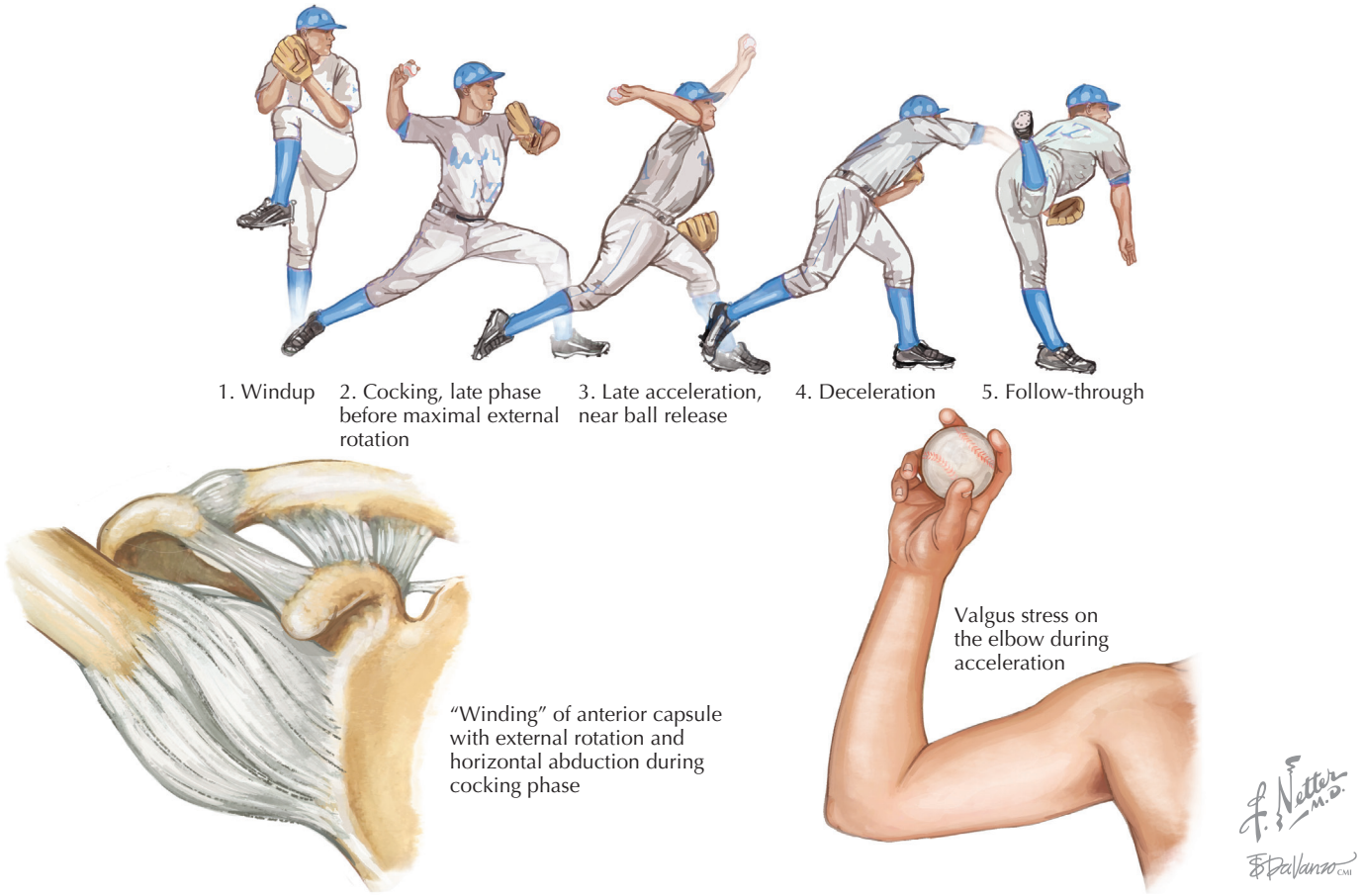


**Figure 74.1.** Phases of the overhead throw.

occur at the posteromedial ulnohumeral articulation (see Fig. 74.1).

## Deceleration

- Dissipation of energy as ball is released
- Distractive force at shoulder is equivalent to pitcher's body weight.
- Description:
  - Begins at ball release
  - Final elbow extension occurs
  - Final internal rotation of shoulder occurs
  - Ends when internal rotation velocity reaches zero
- Injury risk:
  - Superior labrum anterior to posterior (SLAP) lesion—traction injury to the superior labrum at insertion of the long head of biceps tendon
  - Bennett lesion—traction osteophyte of the posterior glenoid lip with thickening of posterior labrum and capsular attachment due to repetitive traction

## Follow-Through

- Allows for dissipation of energy
- Description:
  - Begins at the end of shoulder internal rotation
  - Ends when the trailing leg touches the ground
- Injury risk:
  - An appropriate follow-through reduces the risk of injury by gradual dissipation of the body's kinetic energy.

# COMMON BASEBALL THROWING INJURIES

- Overhead throwing motion results in a preponderance of upper extremity injuries secondary to the significant forces generated.
- Underhanded pitching motion is not associated with significant overuse injury.

## Shoulder

### Rotator Cuff Injury

**Description:** The rotator cuff is the key to a healthy throwing shoulder, centering and controlling the humeral head as the arm is accelerated. With weakness, the cuff may impinge against the acromial arch, the capsule may stretch or cause labral tear (Fig. 74.2). With extreme external rotation and abduction during cocking and early acceleration, undersurface tearing may occur. Rotator cuff injury is the primary differential diagnosis for throwing discomfort.

**Symptoms:** Pain often referred to lateral shoulder; posterior/superior shoulder pain with arm in the cocking position suggests internal impingement with possible undersurface cuff tear; shoulder may continue to ache after activity

**Diagnosis:**

- Pain and/or weakness with resisted external rotation with elbow at side
- Positive Jobe's sign: pain from rotator cuff isometric contracture
- Positive Hawkins sign: subacromial impingement of bursal side of cuff against acromial arch

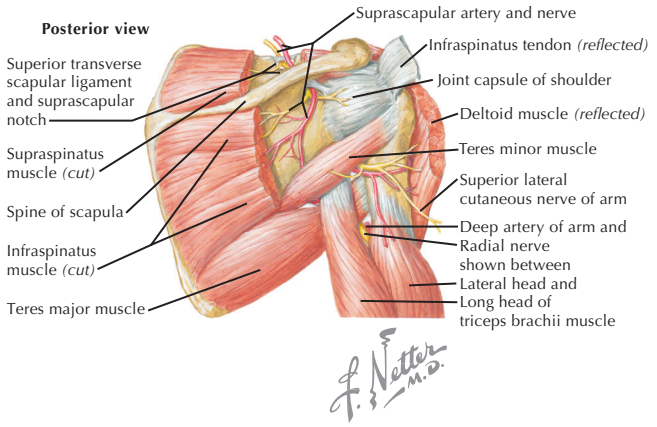


**Figure 74.2.** Rotator cuff anatomy.

O'Brien's Test

Shear Test

Milking maneuver

**Figure 74.3.** Elbow examination tests.

- Pain with extreme abduction/external rotation—internal impingement of articular surface of the cuff against posterior/superior labrum
- Magnetic resonance imaging (MRI) to define the extent of injury when nonsurgical management fails. MRI should be considered as a presurgical study. History and physical examination directs initial treatment. When MRI is advised, consider contrast to better define partial-thickness undersurface tears or associated labral pathology. The abducted and externally rotated (ABER) view of the shoulder is helpful to define posterior, superior labral, and undersurface cuff tears.

**Treatment:**

- Relative rest—varies from complete shutdown of all throwing activity to shifting the player's position (e.g., playing first base instead of pitching)
- Physical therapy
  - Rotator cuff strengthening
    - Thera-Bands with elbow at the side
    - Advance to pulleys with arm in throwing position (shoulder abducted 90° with arm internally rotating against resistance from 90° of external rotation to neutral position)
    - High repetitions (30) with single set
    - Plyometrics before trial of throwing
  - Scapular stabilization—must rule out scapular dyskinesis
  - Posterior capsular stretch—must rule out tight posterior capsule
- Cortisone injection—subacromial
  - Only after failure to respond to physical therapy
  - Must shut down for 5 days after injection
- Surgery if nonsurgical management fails
  - Arthroscopic debridement for partial thickness tears
  - Arthroscopic or open repair for partial tears >50%; poor outcomes for return to pitching when a rotator cuff requires a repair

## Labral Injury

**Description:** The superior/posterior labrum can be injured by impingement during the cocking phase or through distraction of the biceps anchor during deceleration (remember, the arm as well as the ball are in essence being thrown from the body and tension from the long head of the biceps can pull the labrum from its attachment).

**Symptoms:** Deep shoulder joint pain with throwing; typically not a problem with daily activities; pain is often positional and is not present at rest

**Diagnosis:** Positive provocative maneuvers with triggered pain located deep in the shoulder joint (Fig. 74.3):

- O'Brien's: Pain while resisting a downward force with the arm extended in the neutral position, horizontally adducted 30°, and thumb pointing down
- Shear: Pain with manipulation of the shoulder in 90° of abduction and maximal external rotation
- Rotation/compression: McMurray's of the shoulder—compress and rotate the shoulder in an attempt to pinch the labrum and trigger pain

**Treatment:**

- Physical therapy
  - Correct anything that may increase the chance of pinching the labrum
  - Rotator cuff strengthening
  - Scapular stabilization
  - Posterior capsular stretch (with loss of internal rotation)
- Arthroscopic labral debridement or repair when nonsurgical management fails

## Capsular Injury

CAPSULAR LAXITY

**Description:** The capsule may stretch anteriorly, resulting in anterior translation of the humeral head. Although the rotator cuff is the primary stabilizer, the capsule will provide static stability at extremes of motion—the cocking phase—and can provide proprioceptive feedback.

**Symptoms:** Sensation of shoulder slipping, loss of velocity and control, and tired arm (dead arm syndrome with subluxation)

**Diagnosis:** Must be made through history; laxity is confirmed by physical examination: load-and-shift maneuver demonstrates laxity and apprehension with translation. Crepitation during load-and-shift maneuver may indicate labral injury.

**Treatment:** Control laxity by improving rotator cuff function with rotator-cuff strengthening and proprioceptive training. Surgery with capsular plication for refractory cases with the

understanding that if the shoulder is made too tight, return to throwing is at a high risk.

GLENOHUMERAL INTERNAL ROTATION DEFICIT (GIRD)

**Description:** During the deceleration phase, the posterior capsule is tensioned. With repetitive trauma to the posterior capsule, thickening and contracture may occur. This is manifested by loss of internal rotation. With a tight posterior capsule, the humeral head translates anteriorly and superiorly, resulting in cuff impingement and possible superior labral injury. Check for a loss in internal rotation whenever rotator cuff impingement symptoms are present. A traction osteophyte arising from the posterior capsular attachment on the glenoid is known as a *Bennett lesion*.

**Symptoms:** Deep posterior shoulder pain; anterior lateral cuff pain secondary to impingement

**Diagnosis:** Examination reveals loss of internal rotation:

- Measurement is recorded with the arm abducted 90° in the scapular plane.
- Loss of ≥20° is typically symptomatic.
- Must account for the usual shift in rotation of the throwing shoulder with entire range of motion shifted in external rotation

**Treatment:** Physical therapy with stretching of the posterior capsule includes adduction of the arm across the chest and internal rotation with arm abducted 90° (Sleeper Stretch); surgical release in refractory cases—rarely required

### Scapular Dyskinesis

**Description:** The scapula is the anchor for the arm during throwing motions. Incorrect scapular position or rhythm may lead to shoulder injury.

**Symptoms:** Anterior lateral cuff impingement pain, deep anterior shoulder discomfort, and pain along posterior/medial border of the scapula

**Diagnosis:** Physical examination reveals the dyskinetic scapula.

- View patient from behind.
- Observe for differences in shoulder blade height and rotation.
- Observe for scapular winging when bringing the arm down from a forward elevated position.

**Treatment:** Physical therapy with scapular stabilization program; time required to correct the dyskinesis

### Latissimus Dorsi/Teres Major Strain

**Description:** Strain at the attachment site of the proximal third of the latissimus dorsi and teres major; may fail at the boney insertion as the tendons become joined; often misdiagnosed injury

**Symptoms:** Pain localized to posterior inferior shoulder; may vary from complete avulsion to minor strain

**Treatment:** Physical therapy

## Elbow

- Valgus moment is produced with throwing; the UCL is stretched and the radial capitellar articulation is compressed (Fig. 74.4)
- With extension of the elbow under valgus stress, the posterior medial ulnohumeral articulation gets compressed, which has been coined the "valgus extension overload syndrome" (see Fig. 74.1).

### Ulnar Collateral Ligament (UCL) Spain

**Description:** Valgus stress with throwing approaches strength of the ligament; may fail with a single throw or attenuate over time; common flexors protect the ligament

**Diagnosis:**

- History of an acute "pop" felt on medial side of the elbow associated with acute complete rupture; chronic medial-sided pain with throwing and gradual ligamentous degeneration

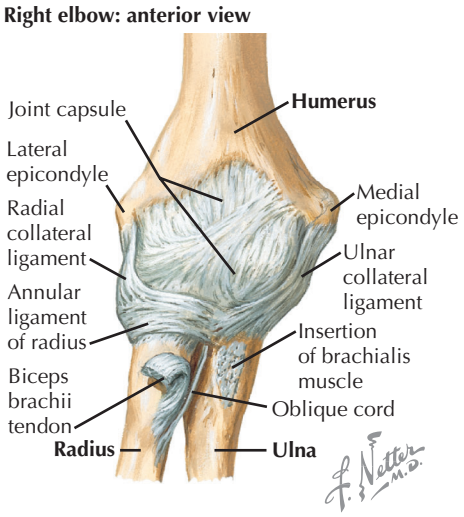


**Figure 74.4.** Anatomy of the elbow.

- Pain over UCL with palpation
- Pain, increased laxity, and/or soft endpoint with valgus stress of the elbow in 20°–30° of flexion
- Pain with "milking maneuver" (valgus stress applied with the elbow flexed 90° and forearm in maximum pronation) (see Fig. 74.4)
- With increased laxity, traction to ulnar nerve may occur, resulting in cubital tunnel syndrome—numbness in ulnar two digits of the hand
- MRI with contrast to confirm the injury

**Treatment:**

- Complete tear in a pitcher: UCL reconstruction (the "Tommy John" procedure)
- Partial tears:
  - Acute partial tear more likely to respond to nonsurgical management
  - Cessation of throwing for 6 weeks while emphasizing common flexor strengthening
  - Plyometrics before the throwing program
    - Plyometrics involves bouncing a weighted ball against an angled trampoline and catching the ball with the injured arm. This provides proprioceptive training while subjecting the soft tissue to a ballistic load in preparation for throwing.
  - Progressive throwing program

### Common Flexor Strain

**Description:** Dynamic medial stabilizer for the elbow; must differentiate from a UCL sprain

**Diagnosis:** Pain anterior and lateral to the medial epicondyle not over the UCL; pain with resisted wrist flexion/pronation; negative milking maneuver

**Treatment:** Activity restriction is important to prevent subsequent injury to the UCL. Pain with throwing should not be allowed. Physical therapy with gentle common wrist flexor strengthening as symptoms allow; transition to progressive throwing program following the initial course of plyometric exercises

### Articular Surface Injury

**Description:** With the elbow under a valgus stress while in extension, the posteromedial aspect of the olecranon will grind against the medial olecranon fossa; this can lead to arthritis with spurring. The load can be increased with UCL laxity. With valgus stress to the elbow, the radiocapitellar joint is under compression, which may lead to early articular wear.

**Diagnosis:**
- Posterior medial pain with throwing
- Discomfort and possible crepitation with valgus stress with extension
- MRI confirms the extent of chondral injury and rules out UCL injury

**Treatment:**
- Rest, NSAIDs, and cortisone injections
- Treat any underlying UCL laxity
- Arthroscopic debridement

### Olecranon Stress Fracture

**Description:** Repetitive loading of the elbow with pitching

**Diagnosis:**
- Discomfort along the medial aspect of the olecranon with throwing
- Discomfort is poorly localized with a negative milking maneuver.
- MRI to confirm diagnosis

**Treatment:** Rest with cessation of throwing, typically for 4–6 weeks; consider intramedullary screw fixation for failed or recurrent stress fracture

### Ulnar Nerve Neuropathy/Instability

**Description:** With valgus stress at the elbow, the ulnar nerve can develop traction neuritis at the cubital tunnel. UCL laxity may contribute to symptoms and injury to the UCL must be ruled out with ulnar nerve symptoms. Subluxation of the ulnar nerve may occur during the pitching motion. The pitcher will experience a "zinger" down the forearm with subluxation.

**Diagnosis:**
- Dysesthesias in the ulnar two digits
- Tinel's sign at the cubital tunnel
- Palpable subluxation of the ulnar nerve as it perches on the posterior aspect of the medial epicondyle with the elbow in maximum flexion
- Nerve conduction velocities may be used to confirm diagnosis.
- Must rule out UCL laxity as a source of traction on ulnar nerve

**Treatment:**
- NSAIDs
- Night splinting
- Ulnar nerve transposition when nonsurgical management fails
- Reconstruction of UCL when laxity results in a traction neuritis

## COMMON BASEBALL BATTING INJURIES

**Batter's shoulder:** Posterior subluxation of the lead shoulder while swinging the bat; typically occurs when the batter reaches out for a low-and-away pitch resulting in a traction injury to the posterior labral capsular structure; initial injury is treated with rest followed with rotator cuff strengthening and proprioceptive training emphasizing dynamic control. MRI evaluation is considered with failure of initial management and before surgical repair of the labrum, if symptoms persist.

**Anterior shoulder subluxation:** With batting swing follow-through, the lead arm may end in an extreme position of abduction and external rotation. This particularly occurs during a one-arm follow-through where the back hand releases the bat, placing all the decelerating force on a single shoulder, which may result in injury to the anterior labrum and capsule. Initial treatment consists of rest, rotator-cuff strengthening, and proprioceptive training. If nonsurgical treatment fails, MRI with subsequent arthroscopic labral repair may be indicated. Risk of re-injury may be reduced by keeping the back hand on the bat during follow-through.

**Contusion/fracture:** A baseball weighs 5 ounces; a major-league pitcher can throw the ball at up to 100 mph; when a player is hit by a pitch or fouls off, a ball injury may occur. Hand fractures occur from direct impact with a thrown ball. Foot fractures occur from an indirect impact from a ball fouled off the bat.

**Extensor carpi ulnaris subluxation:** Typically occurs when the wrist rolls over while swinging the bat (ulnar deviation with the forearm in pronation); pain over extensor carpi ulnaris tendon at distal ulna; subluxation may be reproduced with resisted forearm pronation while positioned in ulnar deviation. Acute injuries may respond to cast immobilization, but more often, surgical repair/reconstruction of the tendon sheath is necessary.

**Hook of the hamate fracture:** Fractures occur with swinging the bat as the hook makes contact with the nub of the bat; this may occur with a single swing or as a result of repetitive trauma resulting in a stress fracture. Pain is located over the hypothenar eminence. Hand radiographs, including the carpal-tunnel profile view, may provide diagnosis. CT confirms the diagnosis. MRI for stress fracture may be required. Excision is required with fracture.

**Digital neuroma at ulnar base of the thumb:** Neuroma develops secondary to repetitive compression and impact from a bat handle. Dysesthesias over the ulnar aspect of the thumb with radiating discomfort is experienced. A Tinel's sign is often present on examination, and palpable nodular thickening of the nerve may be present. Initial treatment comprises rest, NSAIDs, and additional padding of the batter's glove. Cortisone injections may be used when padding is ineffective. Cryotherapy and surgical transposition of the nerve are options when all previous treatment options have failed.

**Concussion, fractures and contusions:** From thrown or batted ball

## INJURIES UNIQUE TO YOUTH BASEBALL

### Overview

- Skeletally immature athletes may incur an injury to upper extremity growth plates instead of ligaments or capsule because these are often the weakest link. The body will fail at the weakest site, and in the Little Leaguer, the growth plate is often the weakest link
- When evaluating a growth plate, comparison views are mandatory, with the nonthrowing extremity as a control.
- In an effort to reduce the risk of throwing injuries, Little League baseball has instituted pitching limits based on pitch count, required rest, and age of the pitcher.

### Little League Pitching Limits

- Age 17–18 years: 105 pitches/day
- Age 13–16 years: 95 pitches/day
- Age 11–12 years: 85 pitches/day
- Age 9–10 years: 75 pitches/day
- Age 7–8 years: 50 pitches/day
- Exceptions: Pitchers may continue to pitch until the current batter in which the limit is reached (is put out or reaches base). A pitcher who delivers one or more pitches in a game cannot play the position of the catcher for the remainder of that day.

## REST REQUIREMENTS (Table 74.1)

### Common Injuries

#### Little Leaguer's Elbow (Medial Epicondyle Physeal Injury)

- UCL is stronger than epicondylar physis.
- May be acute or chronic injury. Acute injury represents avulsion fracture of the growth plate with displacement. Chronic injury will show a widened physis on radiographs.

**TABLE 74.1 LITTLE LEAGUE REST REQUIREMENTS**

| | |
|---|---|
| **Age ≤16 Years** | • If a player pitches ≥61 pitches/day, 3 calendar days of rest and a game must be observed.<br>• If a player pitches 41–60 pitches/day, 2 calendar days of rest and a game must be observed.<br>• If a player pitches 21–40 pitches/day, 1 calendar day of rest must be observed. |
| **Age 17–18 Years** | • If a player pitches ≥76 pitches/day, 3 calendar days of rest and a game must be observed.<br>• If a player pitches 51–75 pitches/day, 2 calendar days of rest and a game must be observed.<br>• If a player pitches 26–51 pitches/day, 1 calendar day of rest must be observed.<br>• If a player pitches 1–25 pitches/day, no rest is required. |

- Pain over medial epicondyle with pitching and palpation
- Medial pain with valgus stress and milking maneuver
- Medial laxity with valgus stress with avulsion fracture
- For widened growth plate, treat with relative rest
- For displaced fracture, treat with anatomic repair

### Little Leaguer's Shoulder (Proximal Humeral Physeal Injury)

- Pain over the proximal arm with throwing
- Comparison radiographs required for diagnosis
- Widening of proximal humeral growth plate on radiographs
- Treat with relative rest (pitching with pain may lead to fracture)
- 4–8 weeks rest typically required for healing

### Osteochondritis Dissecans of the Capitellum

- Osteonecrosis of the capitellum
- Lateral elbow pain with throwing
- Locking occurs with formation of loose bodies
- Diagnosis with radiographs
- MRI may be required for staging of lesion
- Treat with rest for intact lesions
- Excision or repair required for unstable lesions

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 75 SOFTBALL

Andrew William Gottschalk • Ryanne Elise Clarke

## INTRODUCTION

First conceived as a way to play baseball (see Chapter 73) indoors during months of harsh weather, softball has emerged over the decades as one of the most popular sports in the United States. Enjoyed by females and males of all ages and at all levels of competition, softball has proven itself distinct from its older cousin in terms of rules, biomechanical techniques, and injuries sustained during play.

## EPIDEMIOLOGY

### Incidence of Play

- Softball is played in over 100 countries worldwide.
- Approximately 40 million Americans play at least one game of softball each year.
- Softball is one of the most popular sports in the United States partly because of its myriad variants, including:
  - Organized grade school and high school softball leagues
  - Organized college softball leagues
  - Female and male professional softball teams
  - Recreational female, male, and mixed-gender team leagues for all ages
  - Fast- and slow-pitch rule variations
- The Amateur Softball Association of America (ASA) annually registers >245,000 softball teams.
  - 83,000 are female youth fast-pitch teams
- National College Athletic Association (NCAA) women's teams average 41.6 games per season and 49.6 practices per season

### Incidence of Injury

- On average, 1 person every 4 minutes is seen in an emergency department in the United States for a softball-related injury.

## GENERAL PRINCIPLES

### Terminology and Rules

#### Overview

- The rules of softball are similar to those of baseball.
- Notable differences: softball utilizes
  - A larger ball
  - A smaller playing field
  - Underhand pitching
  - Pitching area: pitchers stand in an 8-foot-diameter "pitcher's circle" that is flush with the field.
- As with baseball, almost every softball league requires helmets for batters and base runners.
- Mercy rules
  - NCAA softball: The game ends when one team wins by ≥8 runs after at least five completed innings.

#### Fast- Versus Slow-Pitch Softball

The major variations in softball play are with respect to how the ball is pitched.

FAST-PITCH SOFTBALL

- The primary variant used in competitive high school, college (including NCAA), and professional softball play.
- The dimensions of a regulation NCAA softball are rather specific:
  - $11\frac{7}{8}$–$12\frac{1}{4}$ inches in circumference
  - $6\frac{1}{2}$–7 ounces
- In fast-pitch softball, the ball is launched underhand in a 360-degree windmill motion 40–46 feet away from the home plate (vs. 60.5 feet in baseball).
- Distance depends on the league and player age.
- The ball advances in a trajectory that can have upward, downward, or side-to-side motion.
- As in baseball, a strike is called if the ball passes over the home plate in a zone between the batter's knees and chest.
- There is a 60-foot distance between softball bases.
- Leading off a base is not permitted in softball until after the ball has left the pitcher's hand.
- As in baseball, bunting and base stealing are permitted offensive strategies in fast-pitch softball.
- "Slap-hitting" is a permitted offensive strategy, unique to fast-pitch softball, wherein the batter starts at the back of the batter's box and—once the pitch is thrown—advances toward the pitcher before aiming a bunt or short "slap" swing at the ball.
- "Drag-bunting" is a permitted offensive strategy, also unique to fast-pitch softball, wherein a left-handed hitting batter will essentially start running to first base as s/he is in the process of striking the ball.
- There are seven innings of regulation play in an NCAA softball game.
- As in baseball, there are nine defensive players on the field at a time.

SLOW-PITCH SOFTBALL

- The main variant used in recreational softball league play in the United States (although certain recreational leagues do use fast-pitch)
- While most slow-pitch softball leagues utilize a standard-dimension fast-pitch softball, certain leagues use larger balls up to 16 inches in circumference.
- The ball is usually lobbed underhand in a "half-windmill" motion 50 feet from home base in an arc trajectory.
- The apex of the arc must be 6–12 feet high for the pitch to be legal.
- The ball is aimed so that it would land on home plate, a strike.
- Leading off a base is usually not permitted in slow-pitch softball.
- A player must stay on base until the hitter makes contact with the ball.
- Unlike fast-pitch softball, the offensive strategies of bunting and base stealing are usually not permitted in slow-pitch softball leagues.
- There are usually seven innings of play in a slow-pitch softball game.
- Unlike in fast-pitch softball and baseball, there are 10 defensive players on the field at a time.
  - The tenth position is often a "short fielder," a fourth outfield player who stands in the outfield behind the shortstop.
  - Alternatively, the defense will field both a right- and left-center fielder.

- Slow-pitch softball rules often allow for relatively heavier bats than those used in fast-pitch softball to help increase the power achieved when hitting a slower moving ball.

## History

### Beginnings

- The sport that would come to be known as "softball" was first played in 1887 in Chicago, Illinois, as an indoor variant of baseball by using a larger ball and a smaller field.
- According to tradition, the first game was played using a wadded-up boxing glove and a broomstick handle and was invented by George Hancock.
- The term "softball" was first coined in 1926 and was in widespread use by 1930.
- A slow-pitch softball tournament at the 1933 Chicago World's Fair ignited broader interest in the game.
- Moreover, in 1933, the ASA, the future parent of Team USA softball, was created.
- In 1934, softball rules were standardized in the United States by the Joint Rules Committee on Softball.
- Fast-pitch softball was the dominant form until the ASA officially recognized slow-pitch softball in 1953; thereafter, recreational slow-pitch league participation soon outpaced that of fast-pitch.

### Softball at the Olympics

- The International Olympic Committee (IOC) included women's fast-pitch softball in the 1996 Summer Olympics in Atlanta, Georgia, 4 years after baseball had debuted at the 1992 Summer Olympics in Barcelona, Spain.
- Team USA won the gold medal in 1996, 2000, and 2004 but took the silver behind Team Japan in 2008.
- Following several close votes, the IOC removed both softball and baseball from the Summer Olympics programs of 2012 and 2016, the first time Olympic sports had been removed since the exclusion of polo in 1936.
- Currently, there are ongoing efforts to resurrect softball and baseball as Olympic sports for the 2020 Summer Olympics in Tokyo, Japan.
- With the exclusion of softball from the Olympics, the ASA-sponsored World Cup of Softball, first played in 2005, has taken on greater significance, and is generally seen as the pinnacle of annual world softball competition.

# COMMON INJURIES AND MEDICAL PROBLEMS

## Overview

- Between 1994 and 2010, 2,107,823 patients aged 7–96 years were seen and treated in emergency departments of the United States for softball-related injuries.
- Injury rates are 1.6 times higher in games than in practices.
- Most game-related injuries occur during away games.
- The decreased intensity and increased time spent performing stationary drills in practice settings is considered responsible for the relatively fewer contact-related injuries seen in practices than in competitions.
- Preseason practice injury rates are more than double the regular-season practice injury rates.
  - Likely subsequent to off-season deconditioning
- The lower extremity is the most injured body part, which is followed by the upper extremity, head and neck injuries, trunk and back injuries, and other injuries.
- 6% of all softball injuries are head injuries, including concussions.
- 22% of all softball injuries cause ≥10 days of missed play.
  - The most common such injuries involve the knee and/or ankle.
  - The second most common such injuries are hand or finger fractures.
- Player position/activity plays a role in injury rates (from highest to lowest incidence of injuries): base runners, batters, pitchers, catchers, infielders, and outfielders.
- Injury rates are greater for female softball athletes (9.56 per 1000 players) than for male softball athletes (7.93 per 1000 players).
- Catastrophic injury in softball is extremely rare.
  - The National Center for Catastrophic Injury Research only reported 4 catastrophic softball-related injuries between 1982 and 2007.
    - One was a fatal blow to the head by a ball
    - Along with projectile-related (i.e., ball-related) injuries, head-first sliding is another mechanism for severe and/or catastrophic injury because the cervical vertebrae and spinal cord may be compromised.

## Mechanisms of Injuries

- The mechanism of softball injuries can be broken down into three categories: player-to-player contact, other contact, and noncontact.
- In games, "other contact" injuries are the most frequent and include player contact with balls (particularly hit-by-pitch injuries), fences, equipment, bases, and—most frequently—sliding injuries
- In practices, "noncontact" injuries are the most frequent and include overuse injuries, muscle strains, ligament sprains, and internal knee derangement.
  - Practices comprise more repetitive drills and stationary lower extremity plant-and-turn activities than typical game situations.

## Upper Extremity Injuries

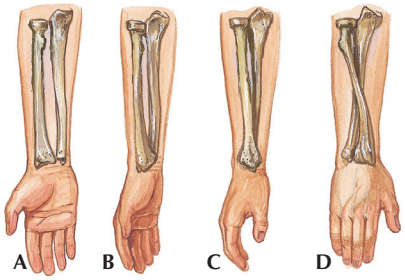
- Almost exclusively overuse injuries of the shoulder and elbow
- The shoulder injury rate for US high school softball was 1.00 injuries per 10,000 athlete exposures (AEs) (vs. 1.72 per 10,000 AEs for baseball)
- Approximately 1:20 United States high school softball shoulder injuries required surgery (vs. approximately 1:10 for baseball)
- Upper extremity injuries occur in pitchers far more often than in nonpitcher positions.
- In softball pitchers, the force acting on the arm in the direction of a launched ball (the "distraction force") can be equal to the pitcher's body weight.
- All levels of baseball have either required or recommended pitch counts/limitations, compared with softball where no governing body has recommended such limitations.
  - Without such regulation, it is possible (and permitted) that a softball pitcher may throw up to 1500–2000 pitches in a three-day, 10-game tournament.
- The windmill pitching technique leads to injuries that are unique to softball play (see below).

### Forearm Stress Injury and Fracture

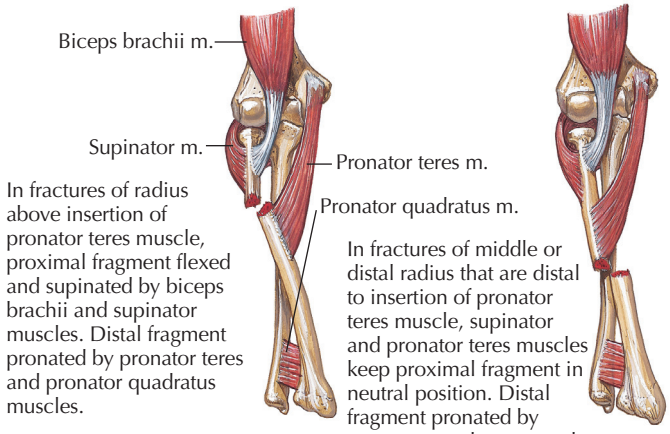
**Description:** The mid diaphysis/third of the ulna is at a risk of stress injury and fracture in sports that require athletes to forcefully pronate the forearm, such as softball windmill pitchers, table tennis players, tennis players who hit a two-handed backhand, and bowlers who apply significant spin to the ball (Fig. 75.1).

**History:** Nagging ulnar pain with pitching (particularly throwing curveballs and fastballs) that becomes more intense and persistent with unrested use.

**Physical examination:** Tenderness along the ulnar midshaft (volar > dorsal) and pain with resisted pronation




Tuberosity of radius useful indicator of degree of pronation or supination of radius
**A.** In full supination, tuberosity directed toward ulna
**B.** In about 40° supination, tuberosity primarily posterior
**C.** In neutral position, tuberosity directly posterior
**D.** In full pronation, tuberosity directed laterally



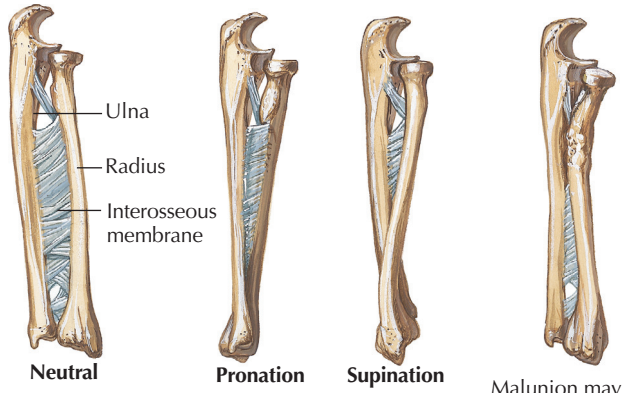




**Figure 75.1.** Biomechanical considerations in fracture of forearm bones.

**Diagnostic considerations**:
- Imaging: Radiography, MRI/bone scan/CT
- Other: CBC, serum vitamin D levels, and nutrition consultation (do not miss the female athlete triad)

**Treatment:**
- **Phase 1:** complete throwing rest (not just pitching), avoidance of upper body resistance/weight training, avoidance of batting, and appropriate caloric intake; consider bracing with wrist immobilization brace to prevent pronation. When the athlete is subjectively and objectively pain free, repeat imaging and progress to Phase 2
- **Phase 2:** physical therapy aimed at rotator-cuff strengthening, scapular stabilization, and core stability; repeat imaging if indicated by the most recent studies
- **Phase 3:** transition from physical therapy to a formal throwing program:

short toss→ long toss→ easy pitching→ harder pitching→ pitches with pronation→ competitive pitching

**Return to play**: 6–8 weeks for osseous healing, then progress as tolerated; severe cases can take up to 10 months after presentation to return to competitive pitching

**TABLE 75.1 MAX PITCHES PER GAME BY AGE**

| Age (years) | Max Pitches Per Game |
|---|---|
| 7–8 | 50 |
| 9–10 | 75 |
| 11–12 | 85 |
| 13–16 | 95 |
| 17–18 | 105 |

### Tendonitis of the Long Head of the Biceps

- Softball pitchers activate their biceps brachii in eccentric loading to a much greater extent than do baseball pitchers; this leads to a greater relative incidence of biceps tendinitis in softball pitchers (see Chapter 49: Shoulder Injuries).

### Ulnar Neuritis

- Repetitive stretching of the ulnar nerve at the medial elbow may occur in both softball and baseball pitchers.
- Softball pitchers may strike the medial elbow of the pitching arm against the lateral pelvis just before ball release, placing the ulnar nerve at a risk of injury (see Chapter 50: Elbow Injuries).

### Elbow Ulnar Collateral Ligament (UCL) Sprain and Rupture

- Repetitive overhead throwing may lead to UCL inflammation, sprain, or rupture.
- Softball windmill pitchers are at a lesser risk than their baseball counterparts because the windmill technique avoids the more severe medial elbow stresses encountered with several baseball pitching techniques (see Chapter 74: Baseball).

### Prevention of Upper Extremity Injuries

- Overuse injuries are almost entirely preventable (the best medicine is education).
- Inappropriate pitching techniques are often taught at youth levels and even in college. This causes injuries in young would-be elite players and often prevents these athletes from advancing to play softball at higher levels. Even at the professional level, the most devastating injuries are often related to poor mechanics resulting in overuse/chronic pathologies. Sports medicine physicians, expert coaches, and sports performance staff can work together with pitchers and throwers to ensure appropriate form, thereby preventing injuries.
- Insist on age-appropriate pitching mechanics and pitch counts (at the very least in youth and high school softball).
  - Several elite pitchers do not start pitching until they are in high school or even later in their careers.
  - Youth and high school softball should use Little League Baseball pitch counts (see Tables 75.1 and 75.2).
- Have athletes warm up before pitching or throwing:
  - Warming up muscle groups before sports participation likely reduces the risk of injury and may also improve performance.

**TABLE 75.2 NUMBER OF PITCHES IN A 24-HOUR PERIOD**

| Ages 7–16 Years | Ages 17–18 Years | Required Rest |
|---|---|---|
| ≥61 | ≥76 | 72 hours |
| 41–60 | 51–75 | 48 hours |
| 21–40 | 26–50 | 24 hours |
| 1–20 | 1–25 | None |

  - Stretching joints before sports participation likely does not reduce the risk of injury and may even increase the risk of injury and/or worsen performance (see below).
- Consider pre-activation exercises before sports participation:
  - Certain evidence suggests that pre-activation exercises such as depth jumps or weighted jumps acutely enhance the firing of Type II (i.e., fast-twitch) muscle fibers, thereby improving performance.
  - These pre-activation exercises should be used with caution. While they may increase early muscle/Type II fiber/fast-twitch muscle performance, there is an inevitable early introduction of fatigue to the muscle groups we hope to strengthen. In addition, given the ballistic/plyometric nature of these exercises, a risk of acute injury (e.g., ankle sprain) in inherent in their use.
- Joint laxity and stability should be taken into account.
  - Athletes' joints tend to be lax, as opposed to being tight.
    - Particularly true in female athletes and throwing athletes; however, softball players are often seen performing a stretch routine before a practice or game.
  - While muscle warm-up is appropriate before sports play, musculoskeletal stretching is—at best—unnecessary and—at worst—counterproductive to protecting joints from injury.
    - Instead, continued attention to joint stabilization through muscle strengthening, and when appropriate, bracing often assists in the protection of joints from injury.
- Encourage ongoing communication among the at-risk or injured athlete, the coaches, and the medical staff.

### Lower Extremity Injuries

- Upper extremity injuries tend to be more softball specific, but lower extremity injuries are more commonly encountered and are more sport generic (e.g., knee injuries and ankle sprains).
- Lower extremity injuries (42% of softball injuries) occur more frequently than upper extremity injuries (33%).
- Softball injuries: keeping a player out for >21 days usually affects the knee (31% of such injuries) or the ankle (19%).
  - Often common ankle sprains and knee internal derangements
- Ground reaction forces
  - A softball pitcher's leading lower extremity (the "stride leg") is subject to significant axial load forces, which may result in overuse or—less commonly—traumatic injuries.
  - Often involve the knee, and among knee injuries, meniscal irritation or tearing the most common

### Other Injuries

- Softball and baseball are the leading causes of sports-related facial trauma in the United States
  - 68% of craniomaxillofacial fractures in softball and baseball are caused by ball impact.

### Safe Softball Conditions

- Appropriate maintenance of competition and practice ball fields helps prevent acute knee, ankle, and fall-related injuries.
- Consistent, appropriate use of protective gear (e.g., batting helmets) helps prevent and minimize the dangers of projectile injuries.
- Consistent, appropriate use of equipment (e.g., breakaway bases) helps prevent injuries.
- Attention to appropriate playing techniques (e.g., sliding into base) and compliance with safety-based rules are fundamental, and often overlooked, cornerstones in the prevention of injuries.

## ACKNOWLEDGMENT

The authors would like to acknowledge the work of Jordan Blough, Ken Eriksen, John Guido, Michele Latimer, and Erica Taylor.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# 76 TENNIS

Alan H. Lee • Todd S. Ellenbecker • Marc R. Safran

## INTRODUCTION

- Over 18 million people in the United States (US) participate in tennis at least once a year, with an additional 14 million expressing interest in playing tennis.
- Five million people play tennis at least twice a month.
- More than 500,000 adolescents participate in tennis.
- Roughly 650,000 people play in the US at the competitive level.

## EPIDEMIOLOGY

- Estimates range from 0.04 to 20 injuries per 1000 hours of tennis played.
- Most injuries occur in the lower extremity (up to 67%), followed by the upper extremity and finally the trunk.
- A majority of injuries are sprains or repetitive trauma overload injuries.
- Acute injuries usually present in the lower extremity, whereas repetitive/chronic injuries tend to occur in the upper extremity and trunk.

## GENERAL PRINCIPLES

### Physiology

- Intermittent high-intensity exercise that requires aerobic and anaerobic fitness
- Tennis is a noncyclical anaerobic sport (10%–30%) with an aerobic recovery phase (70%–90%).
- Single rallies may only last 3–8 seconds, but complete matches may last for up to 3 hours.
- Over the course of a match, 300–500 bursts of effort may be expended.
- Tennis requires elements of quickness, endurance, strength, flexibility, reaction time/speed, agility, and coordination.
- Movements include sprinting, twisting, side-to-side running, sliding, jumping, lunging, and quick stops.
- On an average, the heart rate in singles tennis can be >160 beats per minute, >80% of the average maximal heart rate.
- Depending upon conditioning, age, gender, play intensity, hydration status, and environment, a player may lose 0.5–2.5 L of water per hour of play.
- Conditioning includes both aerobic and anaerobic fitness as well as progressive resistance strengthening of key muscle groups, including strengthening of the core and scapular stabilizers.

### Equipment/Facilities

**Racquet composition:** Change in manufacturing materials has resulted in racquets that are larger, lighter, stiffer, and more powerful than racquets of the past. Increased head size and increased stiffness may reduce arm vibration; however, to date, no studies have determined its effect on injury or provided any evidence on optimal racquet selection for injury prevention.

**Court surfaces:** Play a role in the types of injuries seen

**Clay surface:** Loose surface causes the ball to rapidly lose speed and bounce higher; allows increased time for the opponent to reach/return the ball; considered a "slow" surface; this allows for longer rallies and matches, resulting in overuse injuries. The forgiving surface is gentler on lower extremity joints, and certain studies have suggested that the risk of injury is lowest on clay courts. However, due to the loss of speed of the ball, the power to hit the ball hard must come from the kinetic chain, resulting in upper body and back overuse injuries as well. Further research is needed to establish whether playing tennis on clay courts results in fewer injuries, particularly in the lower extremity and trunk.

**Hard surface:** Concrete, coated asphalt; balls bounce low, giving hard-hitting players an advantage; considered a "fast" surface; harder impact to the lower extremities, with stress fractures of the lower extremities and other injuries, such as patellofemoral pain, more prevalent; sudden stops and starts make tennis toe and ankle sprains more common on this surface. Because a powerful serve is more important on this surface, injuries due to serving are more common.

**Grass surface:** Grass grown on hard, packed soil; balls tend to slide and bounce low, making returns difficult; favors the serve-and-volley player; considered the "fastest" surface; however, unevenness of the surface may result in slipping and sprains in the lower extremity.

**Indoor courts:** Allow for year-round play; usually have a hard court surface

## MECHANICS OF TENNIS

### Kinetic Chain

- The kinetic chain is the efficient transference of force from the ground to the racquet through the coordinated sequencing of the legs, hips, trunk, and upper extremity.
- Each segment transfers more energy than the previous one, resulting in maximal racquet acceleration.
- Fluid motion through the kinetic chain is essential to generate a powerful swing and minimize the risk of injury.

### Strokes

- 75% of play involves the forehand and serve.

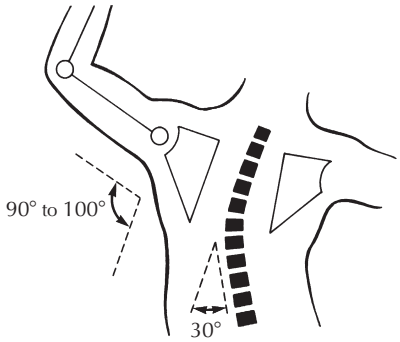
#### The Serve

- Considered the most important stroke of the game and is also the most commonly associated with injury
- The service motion puts a significant stress on the lower extremities, spine, abdomen, and shoulder, although certain phases of the serve are more apt to cause injury than others.
- Four phases: wind-up, cocking, acceleration, and follow-through (Fig. 76.1)

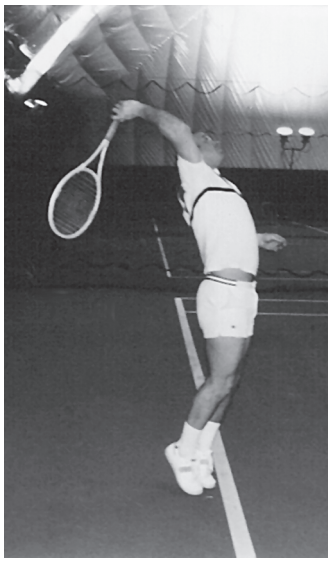
FOUR PHASES OF SERVE

**Wind-up:** From the standstill (ready) position, this phase is the initiation of the serving motion and ends with the toss of the ball by the contralateral extremity (when the ball leaves the hand). The lower extremities prepare for the build-up of power that occurs in the cocking phase as the knees and hips bend. The hips and back rotate toward the dominant extremity (see Fig. 76.1).

**Cocking:** From the release of the ball for the toss, through the point where body motion stops moving backwards, toward the




This view of service wind-up illustrates the addition of 30° lateral thoracic tilt to the 90° to 100° of scapulohumeral abduction.



Back and neck hyperextension should be avoided during the service motion.

Service ball toss in front of service line ensures hyperextension is avoided.

**Figure 76.1.** Phases of serve.

dominant extremity side; the arm holding the racquet appears to be in maximal external rotation (see Fig. 76.1) and is abducted 90°–100°. This phase is characterized by the building up of power. Energy is stored by prestretching of the muscles. Subject to injury:

- The anterior shoulder capsule is tensioned to its physiologic limit with the shoulder in maximum external rotation.
- The glenoid labrum helps prevent shoulder subluxation during external rotation and translation.
- With abduction, horizontal abduction, and external rotation of the humerus, internal impingement may occur.
- Muscles of the lower extremity during extension from a flexed (loaded) position
- Intervertebral disc, pars interarticularis with hyperextension/rotation of the spine
- Wrist extensors

**Acceleration:** This phase is characterized by the body moving forward and upward as the dominant shoulder appears to be in even greater (maximal) external rotation initially and internally rotates to ball impact. High muscular activity is noted with peak activity before ball impact (see Fig. 76.1) as energy in transferred along the kinetic chain. Internal rotation of the humerus is responsible for 40% of the racquet speed at impact. Fluid motion through the kinetic chain is crucial. Injury to one segment will lead to a loss of power and place another segment at a risk of injury. 54% of the energy used in the tennis serve comes from the lower extremity and trunk. Subject to injury:

- Muscular overload of the rectus abdominus, obliques, hip adductors and rotators, rotator cuff, glenohumeral internal rotators and adductors, elbow extensors, and wrist flexors may occur.
- Superior labrum with extreme abduction and internal rotation of the humerus
- Ulnar lateral collateral ligament (LCL) and the flexor–pronator muscles of the elbow secondary to valgus stress
- Extensor carpi ulnaris tendon at the wrist with hypersupinated and ulnarly deviated wrist (for topspin/slice)

**Follow-through:** Ball impact through completion of the stroke; activation of shoulder musculature is required to decelerate the humerus to maintain glenohumeral stability. Long-axis rotation through the arm via internal rotation of the shoulder and forearm pronation helps in the dissipation of forces. Subject to injury:

- The posterior shoulder muscles contract eccentrically to slow the internal rotation of the shoulder.
- The rotator cuff acts to maintain the humeral head within the glenoid.
- The biceps function to slow forearm pronation and elbow extension and assist in stabilizing the glenohumeral joint.
- The posterior capsule is placed under tension as it counters distraction forces.
- Scapular motion combined with contraction of the infraspinatus muscle places the suprascapular nerve, located in the spinoglenoid notch, at a risk of injury.

#### KINEMATICS

- Racquet speeds reach a peak velocity of 62–83 minutes/hour.
- Ball velocities reach 83–153 minutes/hour.
- The aforementioned speeds are achieved in 0.2–0.3 seconds from the end of the cocking phase until ball contact.
- Shoulder rotates internally at 1100°–1700°/second.
- Elbow flexes to 120° during late cocking and extends to 15°–20° of flexion at ball impact, resulting in an extension velocity of 900°–1000°/second.
- Forearm pronation has been recorded at 350°–900°/second before ball impact and has been documented to increase to 1300°/second, 0.1 seconds after impact.
- Wrist speeds approach 1000°/second, 0.1 seconds before ball impact, ROM of wrist during a serve is 90°–100°.

### Ground Strokes

- Forehand and backhand
- Each has three phases: preparation, acceleration, and follow-through
- Each may be hit open stance (feet aligned parallel to the net/baseline) or closed stance (feet aligned perpendicular to the net/baseline).

#### BACKHAND

- The backhand groundstroke may be performed one- or two-handed. The one-handed backhand stroke allows the player to have a better reach and the ability to slice the ball more effectively. The two-handed backhand stroke requires less arm strength but requires more trunk rotation and may result in greater power and accuracy and is considered easier to learn with fewer moving segments to control.
- There is an increased incidence of lateral elbow pain in novice players in the backhand stroke, particularly the one-handed

backhand. This is attributed to not using the trunk and shoulder musculature appropriately, placing more stress across the elbow joint, and from hitting the ball with the wrist in flexion (versus neutral or extension). Players should strive to hit the ball in front of their body to reduce injury to the elbow.

- Using a two-handed backhand reduces the risk of elbow injury owing to a greater need for trunk rotation to hit the backhand while maintaining appropriate elbow and wrist position.
- Wrist pain is also common with the two-handed backhand stroke as players "flick" the racquet at ball contact. Appropriate technique, taping of the wrists, and strengthening exercises will reduce injury to the wrist during this stroke. In particular, the nondominant wrist is more often injured in players who use a two-handed backhand in the preparation phase as the wrist is in an extended, ulnarly deviated, and supinated position.

FOREHAND

- Players may hit the forehand shot with a Western, semi-Western, or Eastern grip.
  - The Western and semi-Western grip allows greater production of topspin and is more suitable for play on clay or slow, hard courts. However, this style places more of a valgus stress on the elbow.
  - The Eastern grip allows the player to slice the ball and is utilized on grass or carpet surfaces by serve- and volley-type players.
- The forehand shot has three phases: preparation, acceleration, and follow-through.

## COMMON INJURIES

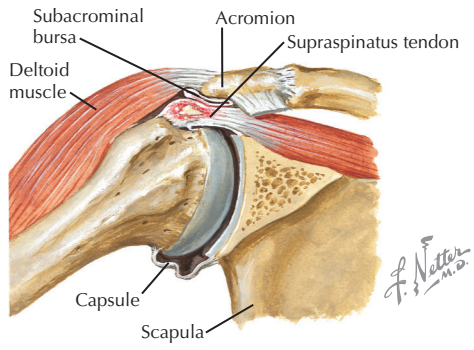
### Upper Extremity

#### Shoulder

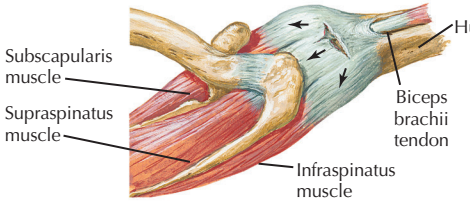
- 35% of junior tennis players complained of shoulder pain at some point in time.
- Over 50% of older players and elite athletes note shoulder pain at some point in their career. A majority of their shoulder pain is caused by impingement and instability.

**King Kong arm:** Drooping and hypertrophy of the musculature of the shoulder girdle of the dominant upper extremity from repetitive use. This is attributed to eccentric stretching of the posterior shoulder and scapular stabilizers.

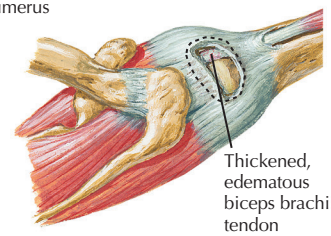
**Rotator cuff inflammation:** One of the more common causes of shoulder pain, attributed to the chronic, repetitive swinging of the racquet. Rotator-cuff inflammation in young players is more often owing to instability, whereas posterior capsular tightness, impingement, and instability are factors in older players. Suprascapular nerve entrapment, particularly at the spinoglenoid notch, may result in atrophy of the infraspinatus muscle, causing weakness in external rotation. However, several players may be asymptomatic. This can occur in approximately 40% of professional tennis players. Decreased strength of the scapular stabilizers and external rotators and decreased flexibility with internal rotation is associated with shoulder instability and is a common finding in elite tennis players with shoulder pathology. Impingement of the rotator cuff between the acromion and humeral head is more likely to occur with serves, overhead shots, and high volleys versus ground strokes (Fig. 76.2). Players will



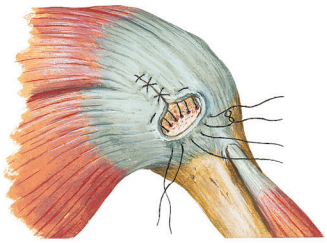

Abduction of arm causes repeated impingement of greater tubercle of humerus on acromion, leading to degeneration and inflammation of supraspinatus tendon, secondary inflammation of bursa, and pain on abduction of arm. Calcific deposits in the supraspinatus tendon may progress to acute calcific tendinitis and sudden onset of severe pain.



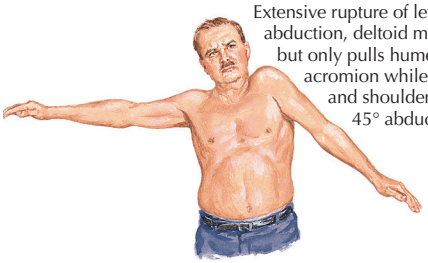

Acute rupture (superior view). Often associated with splitting tear parallel to tendon fibers. Further retraction results in crescentic defect as shown at right.




Retracted tear, commonly found in surgery. *Broken line* indicates extent of debridement of degenerated tendon for repair.

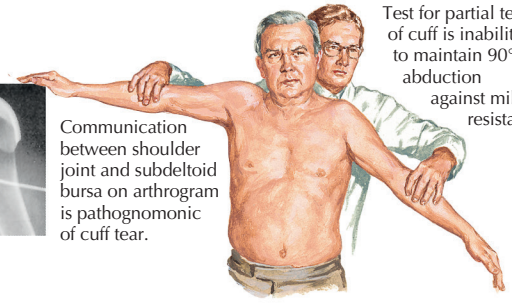


Repair. If freshened edges of tear cannot be brought together, notch is created in humerus just beneath articular surface to allow attachment of tendon through drill holes in bone, using strong sutures.



Extensive rupture of left cuff. To bring about abduction, deltoid muscle contracts strongly but only pulls humerus upward toward acromion while scapula rotates and shoulder girdle is elevated. 45° abduction is thus possible.

Communication between shoulder joint and subdeltoid bursa on arthrogram is pathognomonic of cuff tear.



Test for partial tear of cuff is inability to maintain 90° abduction against mild resistance.

**Figure 76.2.** Rotator cuff injuries. Having the racket hand high above the shoulder level, such as to serve or hit an overhead, puts the subacromial space and rotator cuff at a risk of impingement.

present with subacromial and referred pain to the lateral arm; they may claim that their arm "feels dead" during play. Persistent inflammation may lead to tears of the rotator cuff (see Fig. 76.2).

- **Rehabilitation:** Avoid activities that may further aggravate shoulder symptoms/pathology. Strengthen weak muscles; external rotators tend to be weaker than internal rotators. Increased rotator cuff strength is associated with increased velocity of serve. Assess scapular motion with forward flexion and abduction, and correct periscapular dyskinesis with scapular stabilization exercises. Maintaining flexibility of the posterior shoulder is important for prevention of injury. Common stretches recommended to achieve this include the sleeper stretch and the cross-arm adduction stretch.

**Biceps tendinitis:** Overuse injury, leading to pain in the front of the shoulder with activities that flex the arm at the elbow, rotate the forearm, and accelerate/decelerate the arm such as hitting a high topspin forehand. Activity modifications and anti-inflammatory medications with eventual strengthening of the muscles of the shoulder, elbow, and scapula are recommended to prevent persistent tendinitis or tendon rupture; must rule out superior labrum, anterior to posterior (SLAP) lesions.

**Superior labrum, anterior to posterior (SLAP) lesions:** Two theories regarding the etiology of SLAP lesions in tennis players:

- Tightness of the posterior inferior capsule leads to peeling off of the posterior superior labrum when the arm is abducted 90° and externally rotated.
- Tension placed on the biceps tendon with repeated internal and external rotation of the shoulder leads to avulsion of the labrum from the superior glenoid.

**Osteoarthritis (OA):** In a recent study of senior elite tennis players with no prior shoulder surgery or trauma, there was a statistically significant increase in mild osteoarthritis (OA) of the glenohumeral joint in the player's dominant shoulder compared with age-matched controls. Increased instability because of high demands placed on the shoulder during tennis play may be the underlying cause for the development of OA.

## Elbow

- Strengthening biceps, triceps, and wrist flexors/extensors (as they cross the elbow) will improve control over the elbow, helping to reduce the risk of injury.
- Full pronation of the arm after impact will reduce excessive load and stress on the elbow.

**Lateral epicondylitis (tennis elbow):** 50% of recreational players sustain injuries to the origin of the extensor carpi radialis brevis (ECRB) tendon (and occasionally the extensor digitorum communis [EDC]) at the lateral epicondyle (Fig. 76.3). Microtears in the tendon lead to the formation of granulation tissue and adhesions, which cause pain.

- Risk factors: age >30 years, inappropriate grip size, tight strings, use of inappropriate equipment, incorrect technique (wrist-flexed backhand), inadequate conditioning, and practice >2 hours/day
- Inappropriate technique: wrist-flexed backhand, premature trunk rotation, and leading with the elbow during the backhand; correction of technique in addition to rest, rehabilitation, counterforce bracing, and anti-inflammatory medications is recommended.

**Medial epicondylitis (golfer's elbow):** Less common than lateral epicondylitis in the general tennis playing population but more common in professional tennis players (see Fig. 76.3); usually due to strain at the origin of the common flexor tendon at the medial epicondyle. Competitive players are prone to this injury from repeated wrist flexion from overhead serves or from pronation stress associated with placing topspin on the ball. Degenerative changes can be seen in flexor carpi radialis (FCR), palmaris longus (PL), flexor digitorum superficialis (FDS), and flexor carpi ulnaris (FCU).

**Medial and lateral elbow tendinosis:** Respond better to physical rehabilitation although steroid injections may help by reducing the pain while rehabilitating; platelet-rich plasma (PRP) injection may result in improved clinical outcomes. Suspect radial nerve entrapment in a player with refractory tennis elbow.

**Epicondylitis (tennis elbow).** Exquisite tenderness over lateral or medial epicondyle of humerus

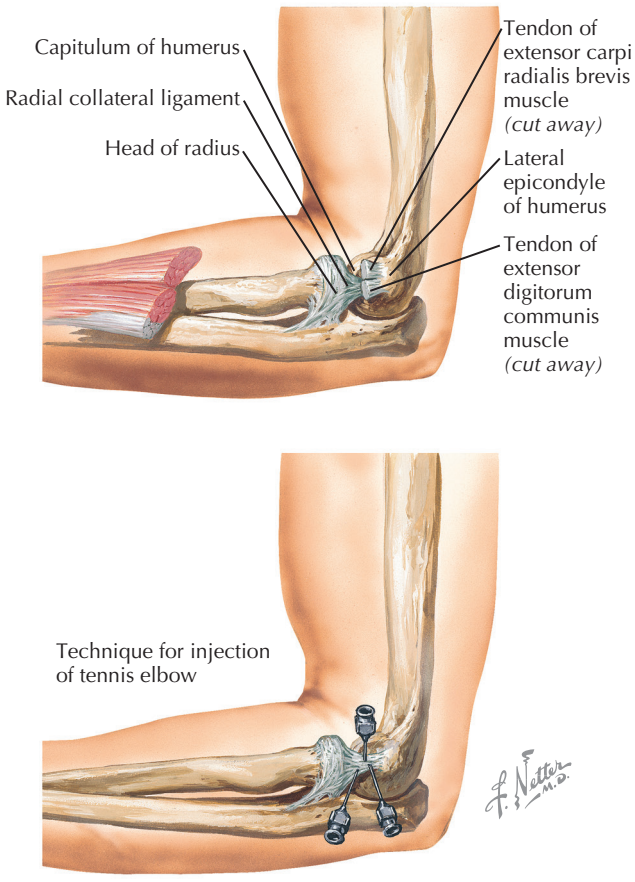



**Figure 76.3.** Tennis elbow. A degenerative process of the elbow. Lateral epicondylitis is seen in recreational tennis players aged 30–50 years, who play 3–5 times/week and, more often, have a 1-handed backhand with poor mechanics. Racket factors have also been implicated. Medial epicondylitis is seen more commonly than tennis elbow in high-level tennis players. Factors associated with this overuse injury include hitting numerous topspin on the forehand and the wrist snap in serving.

**Posterior impingement syndrome of the elbow:** Impaction of olecranon into the olecranon fossa of the humerus with repetitive, forceful extension of the elbow. Pain and tenderness occur about the elbow with repeated bony/soft tissue impingement. Activity modification to avoid leaning on the elbow and hitting the ball with the arm in full extension (e.g., forehand with fully extended arm) is recommended. Players should strengthen the biceps, triceps, and wrist flexors/extensors. Recalcitrant cases may be treated with excision of the impinging osteophytes, usually via an arthroscopic approach.

**Ulnar collateral ligament (UCL) sprain:** Over time, repetitive valgus stress to the elbow may stretch the UCL. This may lead to increased contact and cartilage wear on the lateral side of the elbow (between the radial head and the capitellum) and to ulnar neuritis. A completely ruptured UCL is relatively rare in tennis players and would generally warrant surgical intervention of the dominant arm. Players may avoid this injury by using the appropriate technique. Avoid hitting the Western forehand with the arm fully extended. Exercises to strengthen the biceps, triceps, wrist extensors/flexors are recommended.

### Wrist/Hand

**DeQuervain's stenosing tenosynovitis:** Irritation to the abductor pollicis brevis and extensor pollicis brevis rubbing over the radial styloid with excessive ulnar deviation of the hand during grasping and swinging of the racquet (see Fig. 76.3).

**Triangular fibrocartilage complex (TFCC) tears:** Painful or painless clicking noted on the ulnar side of the wrist due to tear of the TFCC; seen in either the dominant or nondominant wrist of a player with a two-handed backhand; presents with pain with ulnar or radial deviation and pain in the ulnar fovea; increased association with ulnar positive variance (increased length of the ulna relative to the radius). Prevention includes decreasing twisting motion at the wrist, functional bracing, and wrist-strengthening exercises. When treated acutely with nonsurgical management, refractory cases may require arthroscopic debridement and/or repair.

**Hamate fracture:** Rare injury due to impaction of the butt end of the racquet handle into the hook of the hamate (Fig. 76.5); treatment may require excision of the fracture fragment. Wrist ganglion cysts may cause pain, predominantly in the dorsum of the wrist. They are treated by drainage, compression, anti-inflammatory medications, splinting, and surgery; these may recur.

**Extensor carpi radialis brevis (ECRB)/longus, flexor carpi ulnaris tendinitis:** Result from poor form, overstretching of the muscles during serve, and eccentric muscle contraction to stabilize the wrist during off-center shots.

**Wrist sprains:** Acute over-stretching or tearing of one or more ligaments of the wrist because of an unexpected twist, bend, or impact to the wrist. Taping, bracing, and splinting of the wrist should be performed until the player is completely pain free.

**Recurrent dislocating extensor carpi ulnaris tendon:** Tears or stretching of the extensor retinaculum allow for subluxation of the tendon in and out of its normal groove. This commonly occurs when players hit a forehand shot with the wrist in ulnar deviation and supination, imparting a slice to the ball. Symptoms include a painful snapping sensation over the dorsum of the wrist with rotation of the forearm and wrist. Recommended treatment includes immobilization of the wrist for 6 weeks in supination. If nonsurgical treatment fails, surgery may be necessary to repair the retinaculum. Prevention of injury by strengthening exercises of the wrist extensors and application of support braces, tape, or elastic bandages.

**Neurovascular injury:** May include ulnar nerve (cubital tunnel syndrome or entrapment at Guyon's canal), median nerve (pronator teres syndrome and carpal tunnel syndrome), radial nerve entrapment (radial tunnel syndrome), suprascapular nerve injury, or injury to palmar arteries

## Lower Extremities

**Muscle strains:** Partial muscle tears or pulls affecting the quadriceps, hamstrings, adductors, gastrocnemius, and soleus are common. Injury most commonly occurs at the muscle–tendon (myotendinous) junction. Muscles that span two joints are more susceptible to injury (hamstrings, gastrocnemius, and quadriceps).

**Hip injuries:** External rotation and hyperextension of the hip has been suggested to cause tearing of the anterior acetabular labrum (Fig. 76.6). The prevalence of such injuries is greater in players who hit with an open stance stroke. In addition, labral tears occur more commonly in players with hip dysplasia and femoroacetabular impingement. To confirm a diagnosis, MRI arthrogram (with anesthetic injection) is the diagnostic tool of choice. Surgical treatment via hip arthroscopy is advocated for treatment of labral injuries (see Fig. 76.6). Elite tennis players may have asymmetric hypertrophy of the iliopsoas (with the nondominant leg being larger) and may thus develop groin pain from iliopsoas tendinitis. In addition, iliopsoas tightness may result in flexion contracture of the hip, for which a stretching program may be therapeutic.

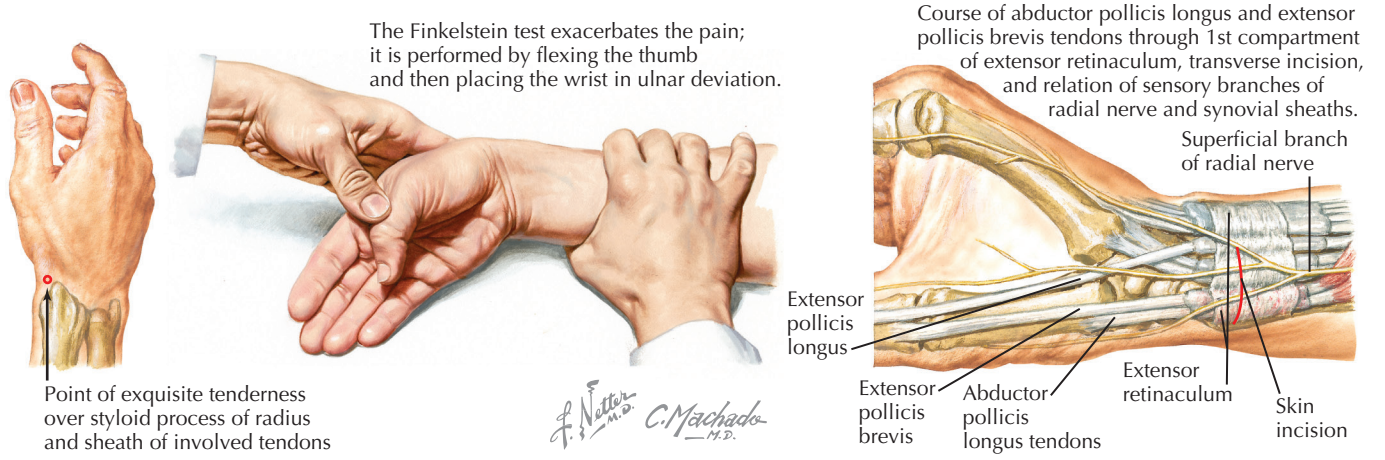



**Figure 76.4.** De Quervain's tenosynovitis. Common in tennis because of the motions required to hit a ball with some spin as well as the grasping necessary to hold the racket.

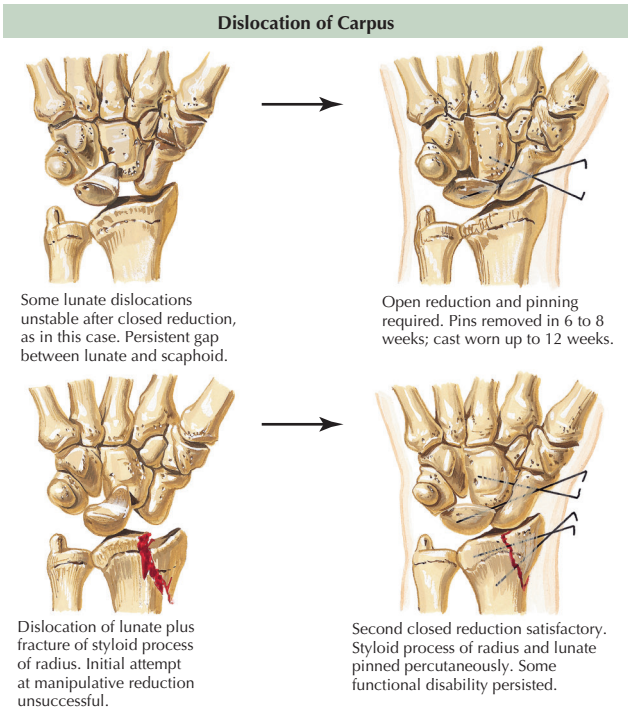
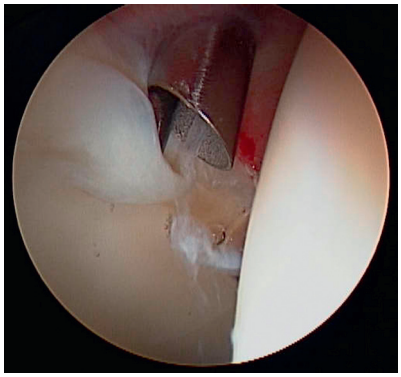



**Figure 76.5.** Wrist injuries.



Hip lateral tears are becoming more common due to the open stance used in hitting the ball. The external rotation and extension of the hip result in increased stresses to the labrum.

**Figure 76.6.** Arthroscopic picture of labral tear.

**Knee injuries:** Common in tennis due to the side-to-side, pivoting, twisting, jumping, bending, rapid acceleration, and deceleration nature of the game; most common knee injuries include patellofemoral syndrome, patellar tendinitis, meniscal injuries, and bursitis. In a survey of the USTA national team, 19% of all injuries were knee injuries. Of these, 70% were traumatic and 30% were from overuse. Anterior cruciate ligament (ACL) injuries occur in between 10% and 13% of tennis players with knee injuries. LCL and medial meniscus pathology occurs more frequently in tennis players than in other sports. An uncommon injury noted more commonly in tennis is popliteus tendinitis.

**Tennis leg:** Incomplete/complete rupture of the medial head of the gastrocnemius. Injury is often incurred with a forceful contraction of the gastrocnemius when the knee is extended and the foot is dorsiflexed. Players aged 35–50 years are at an increased risk of such an injury.

**Medial tibial stress syndrome (shin splints):** Periostitis along the posterior medial border of the distal 1/3rd region of the tibia from repetitive shock to the lower extremity. Muscles that normally absorb the shock in leg fatigue, transferring the energy to the adjacent periosteum/bone. Rest, arch supports/orthotics, taping, and anti-inflammatory medications may alleviate symptoms.

**Achilles tendinopathy/rupture:** Tendinosis usually caused by overuse; rupture is more commonly seen in players aged over 40 years and is associated with movements that require a quick burst of speed. Physical therapy (particularly eccentric loading exercises) is often useful for treatment of tendinopathy. Rupture may be treated either surgically or with casting.

**Ankle sprains:** The most common acute injury in tennis and account for 20%–25% of all injuries. Inversion injuries predominate and result in ATFL and CFL sprains. Appropriate footwear, taping, and bracing may help prevent such injuries. Physical therapy, including proprioceptive training, is also beneficial.

**Plantar fascitis:** An overuse injury that occurs due to repetitive forefoot push-offs during volleys and other repetitive on-court movements; treatment includes activity modification, nonsteroidal anti-inflammatory medications, and evaluation of heel and medial arch support. Night splints that stretch the plantar fascia may also help (Fig. 76.7). Change in footwear or use of an insert may be required.

**Posterior tibial tendinitis:** Pain is noted over the medial aspect of the ankle, accentuated by activities that involve pushing off, including jumping and quick starts; prevention with use of a medial heel wedge, arch supports/orthotics, and stretching.

**Peroneal tendinitis:** Frequently encountered in tennis players due to pivoting and rapid change in direction; players should use tennis shoes appropriate for the surface of the court. Stabilize the ankle with braces, elastic bandaging, tape, or high-level athletic shoes. Strengthen peroneal muscles by eversion against resistance as well as proprioceptive training.

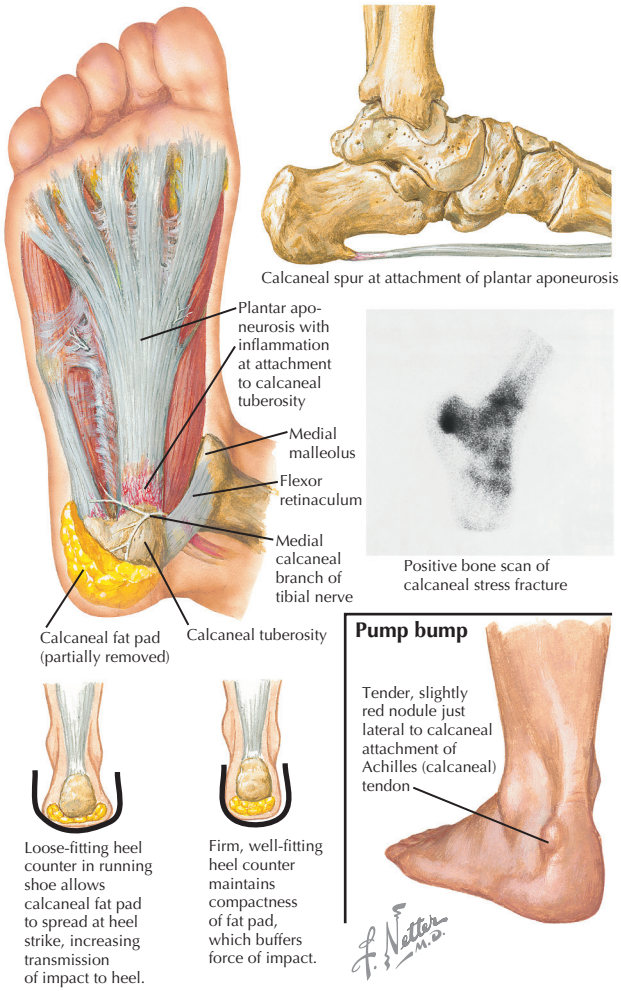



**Figure 76.7.** Plantar fasciitis. Associated with tightness of the calf muscles, and being on the toes for much of play; the plantar fascia may become inflamed, degenerative, and even tear.

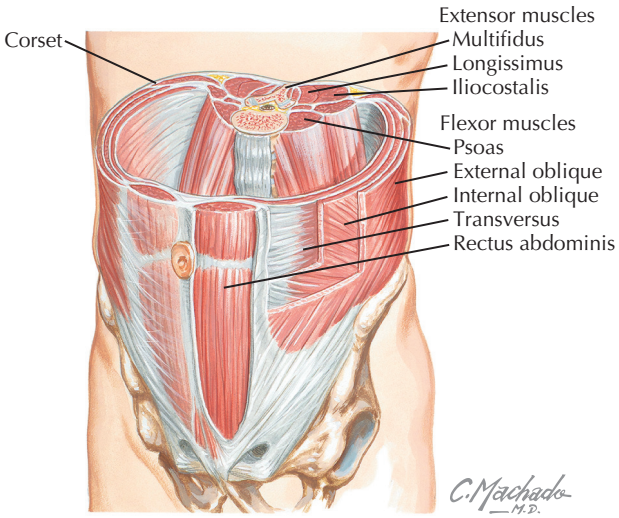



**Figure 76.8.** Muscles of anterior abdominal wall. Abdominal muscle injuries are common in tennis as a result of the stretching and then contraction of the muscles to hit the serve and the increased use of the trunk muscles to generate force with the open stance ground strokes common in today's style of play.

**Tennis toe:** Repetitive abutment of toes against the shoe may lead to a subungual hematoma. Players should wear appropriately sized shoes and keep their toenails cut short.

## Trunk

**Low back pain (LBP):** Common in tennis players of all ages; 38% of professional, 47% of elite junior female, and 31% of elite junior male tennis players have missed a tournament secondary to LBP.

**Lumbar strain:** Onset of symptoms often correlated to a change in intensity/duration of play. Repetitive trunk rotation and hyperextension place the erector spinae and multifidus muscles at a risk of injury. When the player serves the ball, tossing the ball slightly ahead of the service line, and use of lower extremities to launch into the service will decrease the amount of hyperextension of the back and neck.

**Herniated disc:** (player serving, hyperextending and rotating the back with upward force of the lumbar spine by knee and hip extension, and a disc with an annular tear/protrusion of disc contents) Repetitive hyperextension and rotational forces applied to the lumbar spine, particularly during the serve, subject the annulus to microtrauma that could lead to a tear. Correction of form and biomechanics is important to prevent a recurrent injury.

**Facet syndrome:** Repetitive hyperextension, rotation of the trunk, or a combination of both may lead to cartilage wear of the facet joints. Additional load may be placed on the facet when there is loss of disc height, as seen with degeneration of vertebral discs. Players should avoid hyperextension of the lumbar spine to prevent acute facet impingement and chronic facet arthropathy.

**Rib stress fracture:** Repetitive contraction of muscles that have origins or insertions onto the ribs place excessive stress on the ribs and may lead to a stress fracture. Ribs susceptible to stress fracture in the tennis player include the first and fourth–ninth ribs, with the fourth and sixth being most commonly affected. This is because of action of the serratus anterior and external obliques. Players will note pain with deep inspiration, coughing, and overhead activities. Rib fractures are commonly missed on plain radiograph. Bone scan or MRI will confirm the diagnosis. Rib fractures heal with time: 4–6 weeks. Activity modification is recommended during this time.

**Spondylolysis:** Due to repetitive hyperextension of the lumbar spine; prevention is similar to that for lumbar strains, as mentioned earlier.

**Abdominal muscle strains:** Partial muscle tear or pull of the abdominal musculature (Fig. 76.8); nondominant rectus abdominis is predominately affected. The internal and external oblique muscles are also prone to strains. During the cocking phase of the serve, the player is hyperextending his or her back, placing the abdominal musculature on greatest stretch. In the acceleration phase that ensues, the abdominal musculature is forcefully contracted, placing these at a risk of injury. Treatment is ice and rest. Avoid massage over the affected area to minimize the risk of development of myositis ossificans. Prevention focuses on improving core strength and using the appropriate technique.

## Adolescents

- Muscle strains from overuse are the predominant injuries in young players, although the physis is the weakest link and uniquely susceptible in skeletally immature players.

- Injuries to the lower extremity are twice as common as injuries to the spine and upper extremity.
- Injuries to the foot, leg, and wrist prevail in female adolescent players, whereas injuries to the ankle, groin, hand, abdomen, and back prevail in male adolescent players.
- Overall predominance of injury pattern: Strains > Inflammation > Sprain

### Physeal Injuries

**Wrist epiphysitis:** Repeated hyperextension and rotation of the wrist causing inflammation of the distal radius epiphysis; this is commonly seen in adolescent players who attempt to put topspin on the ball. Premature closure of the growth plate is a potential complication of this process. Treatment strategies range from activity modification and wrist immobilization to surgery for treatment of an associated fracture or for premature physeal closure. Players with wrist epiphysitis should avoid push-ups and flatten strokes, avoiding excessive topspin.

**Traction to the apophysis at the greater/lesser tuberosity of the humerus:** Akin to a little leaguer's shoulder; rest and activity modification are the mainstays of treatment. Upon return to play, the player should start with ground strokes only. High volleys and serves should be gradually incorporated.

**Humeral medial epicondyle apophysitis (adolescent medial tennis elbow):** Overuse injury resulting from repetitive muscular contractions of the forearm and wrist flexors during forehands and serves. Players may sense a decrease in their ability to serve at full speed and to fully straighten the elbow. Use of a racquet with vibratory dampening characteristics, an oversized/light/stiff head, flexible shaft, a large cushioned grip (i.e., comfortable to the player), and low tensioned strings of gut or high-quality coreless mutifilament synthetic strings is recommended. Activity modification with limitations on the intensity of conditioning/play and amount of serving/overhead play/throwing/heavy lifting is encouraged.

**Osgood–Schlatter disease:** Tibial tubercle apophysitis; shoe wear modification for increased shock absorption and stability, stretching of the quadriceps and hamstring musculature to decrease the tension of the muscles pulling on the patellar tendon, training on soft surfaces (clay or sandy surfaces), and use of a patellar tendon strap (Fig. 76.9).

**Sever's disease:** Calcaneal apophysitis, most common cause of heel pain in adolescent players; prevention and treatment entail appropriate stretching, use of a heel support that provides cushioning, shock absorption, and decreased tension on the Achilles tendon.

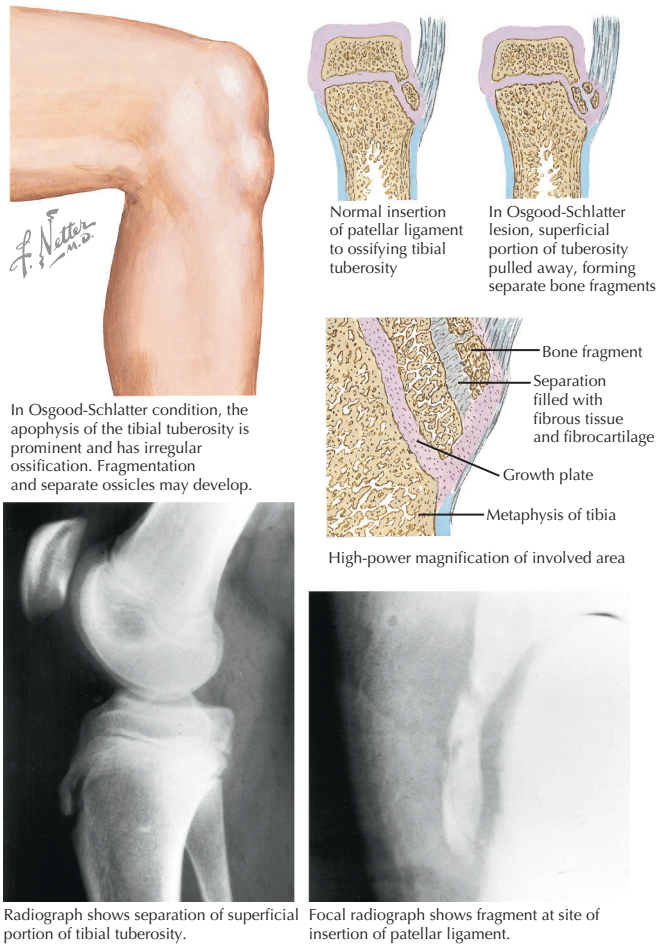



**Figure 76.9.** Osgood–Schlatter lesion. Patellofemoral problems are common in tennis as a result of repeated bending of knees (ready position, serving, and hitting low balls), lunging for the ball, and sudden stops and starts. In young players, Osgood–Schlatter disease, inflammation of the growth plate where the patellar tendon inserts, is common, whereas in adults, patellar tendinitis or patellofemoral overload may occur.

## SUMMARY

- Tennis is a popular sport—worldwide, both genders, all ages
- Tennis requires both aerobic and anaerobic fitness.
- Tennis may result in a variety of unique injuries.
- Lower-extremity injuries predominate, although shoulder, elbow, and back injuries also occur commonly.

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Available at www.ExpertConsult.com.

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# 77 ALPINE SKIING

Matthew R. Gammons • Daniel C. Cole

## GENERAL PRINCIPLES

### Background

- Alpine skiing is a popular sport worldwide, with approximately >200 million participants per year.
- High speeds, variable terrain, and weather conditions, combined with equipment, can create a significant opportunity for getting injured.
- Equipment changes have changed the nature of injuries, but overall rates have not significantly decreased in the past 15–20 years.
- Lower extremity injuries are the most common, but upper extremity injuries are also frequent.
- Head injuries and chest wall/abdominal trauma are also of great concern because these injuries can be life threatening.
- Medical issues include cold exposure, sun exposure, altitude issues, and general travel-related problems.

### Levels of Competition

- Alpine ski racing at its highest level is governed by Fédération Internationale de Ski (FIS).
- The U.S. Ski Association is the governing body in the United States (US).
- The US national team is divided into four groups: A to D. Elite skiers are in the A team, with athletes ranked according to skill into the other divisions, down to the development team, or the D team.
- Junior levels are divided by age groups (U21, U19, U16, U14, U10, and U8).
- Often, there is overlap between collegiate levels, high school levels, and junior race clubs.
- Levels U14–U21 may compete locally, regionally, and nationally, with the best skiers competing internationally. U10 and younger usually compete locally and occasionally regionally.
- There are various master's race associations, and recreational skiers can compete in the National Standard Race (NASTAR). The NASTAR is a program wherein recreational skiers of all ages and abilities can test their skills on courses set up at resorts across the country. Times and scores are compared under a universal handicapping system similar to that used in golf.

### Events

#### Speed Events

- Downhill is the fastest of the events, with speeds reaching 90 mph.
- Skiers are allowed practice runs and course inspections.
- Super G combines downhill with giant slalom.
- Gates in Super G races are farther apart than those in the giant slalom, and speeds are slightly less than those in downhill.
- Full-speed practice runs are not allowed, and only a small amount of inspection time is permitted; this requires the skiers to constantly adjust to the terrain.

#### Technical Events

- Giant slalom involves a course between Super G and slalom that requires more technical turns than Super G.
- Slalom is the most technical of events and involves short arc turns and contact with the poles marking the course.

#### Combined Events

- Classic combined races usually involve one downhill run and a slalom run; occasionally, it may combine a single slalom with a Super G race.

### Event Coverage

- Planning and communication are the keys. Communicate with ski patrol before the event or training to learn established protocols and establish a chain of command in the event of an injury.
- Several ski areas are a significant distance away from medical or trauma facilities; this, along with winter weather, often mandates that stabilizing care and pain control be provided while transport is arranged.
- Medical personnel must be proficient in alpine skiing because courses are often steep and icy and should generally station themselves high up on the course so that they have access to the entire run.
- Radios allow communication between the medical team and spotters on the course; these should be tested ahead of the training or race because most two-way radios are site-to-site and may have trouble transmitting in certain areas. Cell phone service is also inconsistent in several mountainous areas and should be investigated before the competition.
- Most injuries are best taken care of off the mountain; hence, if possible, a majority of the care should be delayed until the athlete can be transported to lodge or medical facilities.

### Epidemiology

- Although rates of skiing injuries have declined in the last three decades, most of this decline occurred in the late 1970s and early 1980s and rates have been stable over the past 25 years.
- Reported injury rates vary from 2 to 5 per 1000 skier days.
- Ratio of lower extremity to upper extremity injury is approximately 2:1.
- Location of most common lower extremity injury has changed from ankle/tibia to knee; this is likely because of equipment changes, including stiffer boot materials and the advent of release bindings.
- Rates and severity of injury are similar with snowboarding, although snowboarders have a higher rate of upper extremity injuries.
- Catastrophic injuries are rare: 0.01 per 1000 skier days.

### Equipment

- Major equipment changes occurred in the late 1970s and early 1980s; this included a move from leather to plastic boots and releasable bindings. Additional equipment changes occurred in the mid 1990s with the introduction of shaped skis.
- A common misconception is that modern equipment protects the knees when it is, in fact, designed to prevent ankle and tibia fractures. Although this appears to have been effective in reducing the number of injuries, it has caused a dramatic increase in knee injuries.
- Standard binding release is based on the skier characteristics such as skill level, boot size, height, and weight: most commonly

adjusted based on the Deutches Institu für Normung (DIN) standard.
- Binding mechanism may not release at slow fall speeds because torque requirement is not met.
- Expert skiers and racers will often ski at DIN settings higher than recommended to prevent prerelease.
- Lower extremity injuries, excluding knee sprains, are often associated with inappropriate equipment adjustment.
- Shaped skis have become the mainstay in all disciplines except downhill. These skies are shorter than the tradition ski and have increased side-cut. Although shaped skis are the most common type of ski used, it is unclear if these skies have had any effects on injuries at the recreational level. Regulation changes limiting the side-cut radius for all World Cup disciplines except slalom were instituted in the 2012/2013 season. Early data suggest a small but statistically significant decrease in overall injury rates.

## Training and Physiology

- Skiing requires a good base of lower extremity and core strengthening and aerobic fitness to tolerate training loads.
- Large eccentric and isometric loads are placed on the lower extremity; hence, strength training should focus on these areas.
- A majority of any ski race event occurs at the anaerobic threshold, hence, cardiovascular training regimens should focus on increasing this tolerance.
- The competitive season of elite skiers involves altitude and significant travel; therefore, training should be frequently adjusted to maximize performance while minimizing the risk of overtraining the athlete.

# SPECIFIC INJURIES

## Lower Extremity

### Knee Ligament Injuries (See Also Chapter 55, Knee Injuries)

- Not unique to skiing but the mechanism of injury differs because of the ski/binding/boot interface (Fig. 77.1)
- Injuries to the medial collateral ligament (MCL) are the most commonly reported, but anterior cruciate ligament (ACL) injuries have dramatically increased and now account for almost 20% of all skiing injuries.
- Injuries among elite skiers are reported at 8.5 per 100 skier seasons.
- Combination injuries (ACL/MCL) are relatively common as well.
- Increased risk of ACL injury in elite female skiers compared to male although the effect is not as dramatic as is seen in field sports
- There appears to be no relationship between knee sprains and binding malfunction. ACL injuries in female recreational skiers have been reported to occur with slow speed falls and no binding release; this does reflect binding malfunction but indicates that torque release requirements, set to protect leg and ankle, may not be met during this type of fall.
- Common mechanisms for knee ligament injuries (see Fig. 77.1)
  - Valgus external rotation: occurs when ski edge catches, forcing the leg into abduction and external rotation; this is the most common mechanism for MCL injuries
  - The boot-induced anterior drawer occurs when the rear of the ski contacts the snow first after jump. The ski then levers the boot forward, creating the anterior drawer movement.
  - The phantom foot: occurs when the skier's weight is posterior, and the skier's hips drop below the level of the flexed knees; weight is on the downhill ski and, as the skier attempts to recover, increased force to the downhill ski results in greater edge pressure. This causes an abrupt internal rotation force on the downhill knee. It was previously believed that this was the most cause of knee ligament injuries; however, more recent studies have challenged this theory.
- Prevention is focused on education, avoidance of high-risk positions, and equipment adjustment. A small-scale study demonstrated a decrease in knee injuries with education on not trying to recover from a fall into the hill (phantom foot). It is unclear what effect these interventions may have on elite skiers, but recent equipment regulations appear to have helped decrease injury rates.

### Fractures

- Although fractures of the lower extremity have decreased, there are still two fractures commonly seen in skiers: spiral and tibial plateau.
- Spiral fractures of the tibia are common in children but are also seen in adults. These are caused by external rotation forces on the ski and have been associated with binding dysfunction (see Fig. 77.1).
- Incidence of tibial plateau fractures, most commonly of the lateral plateau, has increased along with knee ligament injuries.
- The increase of these fractures along with knee injuries likely represents a similar mechanism of injury—valgus/external rotation—as for knee ligament injuries.

## Upper Extremity

- The shoulder, hand, and wrist constitute a majority of upper extremity injuries.
- The shoulder accounts for the largest overall number of upper extremity injuries in skiing. The most common shoulder injuries are listed in Table 77.1.
- Skier's thumb, also known as *gamekeeper's thumb*, is the most common single injury of the upper extremity, accounting for 8% of all injuries.
- Sprains of the ulnar collateral ligament (UCL) occur with a fall onto an outstretched hand with a pole in the palm (see Fig. 77.1).
  - This causes a radial stress on the ligament.
  - Treatment involves protection and occasionally surgical fixation.
  - The risk of UCL injuries may be reduced by having poles with breakaway straps or using a pole without straps.
  - A modified thumb spica splint molded to a ski pole can allow continued skiing participation (see Fig. 77.1).

## Trauma

### Head Injuries (See Also Chapter 45, Head Injuries)

- Account for a small portion of total injuries but for a majority of severe injuries
- Concussions are relatively common, particularly in the adolescent age group.
- Helmets are mandatory in most competitions, but their role in prevention remains unclear. Several studies have shown a

**TABLE 77.1 COMMON SHOULDER INJURIES IN SKIING**

| | |
|---|---|
| Rotator cuff injuries | 24% |
| Anterior dislocation/subluxation | 22% |
| Acromioclavicular separations | 20% |
| Clavicle fractures | 11% |

Data from Koehle MS, Lloyd-Smith R, Taunton JE. Alpine ski injuries and their prevention. *Sports Med.* 2002;32(12):785-793.

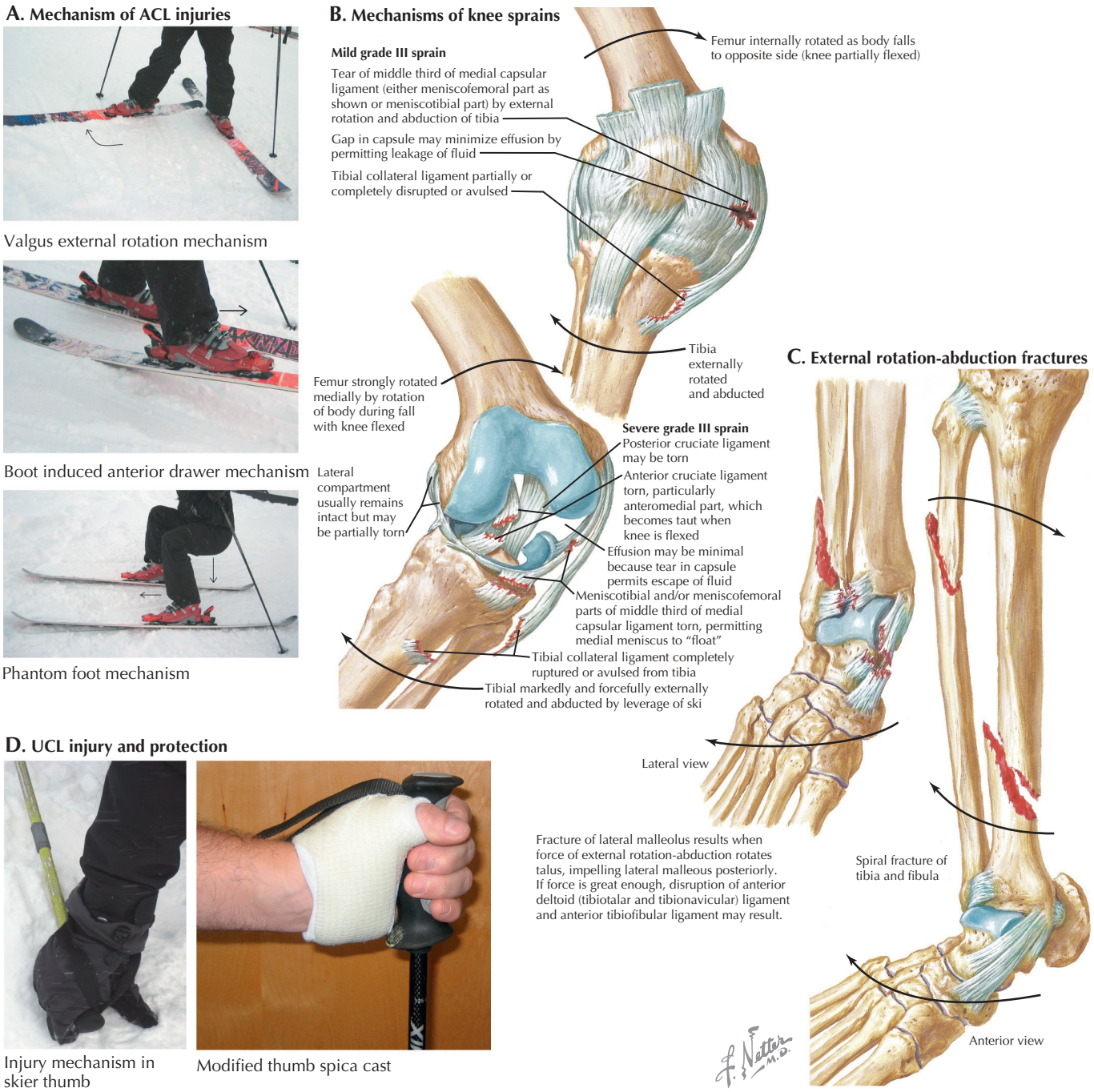


**Figure 77.1.** Skiing injuries.

decrease in the risk of head injuries in helmet wearers. However, recent studies have not shown a decrease in severe traumatic brain injuries despite increased helmet use. Several confounding variables, including rates of helmet use, terrain, weather, conditions, and skill level, make it difficult to assess what overall protective effect helmets may have in skiing.

## Spinal Injuries

- Severe spinal injuries are rare in alpine skiing: 0.001–0.004 per 1000 skier days.
- Spinal injury rates in snowboarding are higher than in skiing, and the most common injury in both is a wedge compression fracture.
- Slope-side treatment of suspected spinal injuries can be difficult.
- Ski patrol members are usually the best personnel to accomplish boarding and spine stabilization on mountains.

## Thoracoabdominal Trauma

- Minor chest-wall, back, and abdominal trauma is relatively common in skiing and is generally not life threatening.
- However, because of the high speeds involved, physicians should always be aware of the possibility of severe trauma, particularly in cases of collisions.
- Severe injuries include aortic rupture and intra-abdominal organ trauma.

### Medical Problems

- Medical problems inherent to skiing include cold-related injuries and altitude illness (see Chapter 22: Exercise in the Cold and Cold Injuries).
- Elite athletes will often log several thousands of miles during the year for both competitions and training camps.
- International travel is common, and general precautions should be taken to prevent travel-related illnesses such as traveler's diarrhea.
- Jet lag and fatigue can also be significant problems and are best managed on a case-by-case basis.
- Because of the cold weather climates, skiers often overlook the potential for sun-related skin problems.
- A combination of altitude and reflection off the snow can increase ultraviolet (UV) exposure to extreme levels.
- Education of both coaches and athletes on sun protection is important to help prevent acute sun exposure injuries and the potential for skin cancer.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 78 CROSS-COUNTRY SKIING

David D. Cosca

## GENERAL PRINCIPLES

- Cross-country or nordic skiing is multifaceted and can be pursued either as a simple recreational outdoor activity or a vigorous competitive endurance sport.
- Cross-country skiing serves as an excellent means to develop and maintain cardiovascular fitness; most large muscle groups of the upper and lower body are used in a smooth, rhythmic, low-impact manner.
- Injury rates are typically lower than those seen in alpine skiing and running.
- It is very popular in Scandinavian countries and is moderately popular in the northeastern and western mountain regions of the United States.

## CROSS-COUNTRY SKIING VARIATIONS

### Trail/Track Skiing

- Skiers use machine-groomed trails.
- Trails are compacted and rolled with heavy sleds pulled by snowcats or snowmobiles, leaving a set of parallel ski tracks and an 8-by-10-foot wide and open lane for ski skating.
- Groomed trails are standard at cross-country ski resorts. Several communities set trails on snow-covered golf courses or bike trails for local citizen use, and some are lighted for night skiing.
- Trail skiing is suitable for a wide range of individuals, young and old, regardless of prior experience. Novices simply "ski-walk," employing a shuffling-like technique on the snow. More experienced skiers use tracks with a diagonal stride (classic) technique or groomed lanes with a ski-skate (freestyle) technique.
- Competitive ski racing and training require machine-groomed trails.

### Backcountry Skiing/Ski Touring

- No groomed trail is required.
- Participants choose their own route (i.e., bushwhacking). Certain enthusiasts pursue a midday trek in the woods and a picnic lunch, while others plan a multiday snow camping tour, and still others seek out backcountry slopes to climb and then descend using a telemark turn technique.
- A telemark turn is one wherein the skier thrusts the outside (or downhill) ski forward, assuming a lunge position with the front knee at approximately 90 degrees of flexion and the back knee in a kneeling position. The turn is carved while holding this position.
- Backcountry skiers typically choose skis with metal edges and relatively heavy, supportive boots.

### Chair Lift–Facilitated Telemark Skiing

- Could be considered a variant of alpine skiing; participants opt for a chair lift at a ski area to repeatedly transport them to the top of a slope and then descend while making telemark turns (in contrast to the parallel turns of alpine skiers).
- Skiers use stiff plastic boots, wide metal-edged skis with a significant side cut, and strong three-pin or riveted toe cable bindings that still allow the heel to lift freely off of the ski. The three-pin and most other cable bindings are not designed to release during a fall, although newer binding designs do release.
- Skilled telemark skiers often reach speeds similar to alpine skiers and are subject to similar injuries.

## COMPETITIVE CROSS-COUNTRY SKIING

### Race Events

- There has been a recent trend toward using audience-friendly formats such as mass-start, sprint, relay, and pursuit (a race that involves switching skis and styles midway through the race).
- Venues for major ski races (e.g., the 2002 Winter Olympic venue at Soldier Hollow, Utah) have been designed to allow better spectator viewing, with large sections of the course visible from the stands.
- World Cup and Olympic race events include (distance format for women and men, respectively) 1 km sprint, 2 × 1 km team sprint, 10 km/15 km individual start, 15 km/30 km pursuit, 30 km/50 km mass start, and 4 × 5 km/4 × 10 km relay.
- Citizen race distances vary from 5 to 55 km. Certain races are specifically designated as classical technique only, whereas most use a freestyle format.
- The largest event in the United States is the American Birkebeiner in northern Wisconsin (52 km, with >7000 participants).

### Equipment

#### Diagonal Stride (Classic)

**Skis:** Double camber, with central area for kick wax (waxless skis have a "fish scale" pattern imprinted on the ski base); when the skier's weight is evenly distributed between skis, the central kick zone should not contact the snow, thus allowing maximal glide. When the ski is aggressively weighted, the kick zone is engaged in the snow, thus allowing a push-off, or "kick." Skis are typically 20–25 cm greater than the skier's height, but the skier's weight should also be considered when choosing ski length and ski flex.

**Boots:** Lightweight, relatively low cut, flexible sole

**Bindings:** Currently, there are two predominant systems: New Nordic Norm (NNN) and Salomon Nordic System (SNS). Both are lightweight and engage the ski boot at the toe alone, allowing the heel to lift freely off of the ski while striding. The two systems primarily differ by the ridges on the binding plate that fit into corresponding slots on the boot's sole.

**Poles:** Carbon fiber (preferred by elite skiers) or aluminum, with variable grip and strap systems. Pole length extends to a height between the skier's armpit and top of shoulder.

#### Ski Skating (Freestyle)

**Skis:** Typically 10–15 cm shorter than classic skis, with more torsional rigidity to accommodate the forces generated during skate push-off; ski design continues to evolve, particularly in relation to side cut and ski tip shape. Racing ski bases have specified "grinds" to enhance glide wax absorption and maximize glide when matched to specific snow conditions.

**Boots:** Skate boots are cut higher, with a stiffer upper boot that is hinged at the ankle, and provide more lateral support than classic boots. Skate boots also have a more rigid sole.

**Bindings:** Similar to classic bindings, but with a more rigid toe plate; the SNS Pilot system has a second attachment point to the boot under the toe that operates as a spring-loaded hinged plate and provides slightly more control over the ski.

**Poles:** Carbon fiber (best) or aluminum, with variable grip and strap systems, slightly longer than classic poles; typical height of a skate pole is skier's midchin.

### Ski Base Preparation

**Ski glide:** Considerable effort is directed at maximizing ski glide during cross-country ski competitions. Most elite skiers carry several different pairs of skis with variable base compositions and stone ground patterns, or "grinds," each best suited to a particular snow condition. A "rill" pattern may also be pressed onto the ski base to facilitate channeling of melting snow and reduce friction on particularly warm and humid days.

**Glide wax:** Typically applied to the entire length of the base of skate skis and to the tips and tails of classic skis; layers of wax are melted in with an iron, scraped nearly clean between layers, and then the final layer is brushed and polished after it is ironed and scraped. Modern, high-performance ski waxes contain a variable percentage of fluorocarbon, typically using a higher percentage for warmer, higher-humidity conditions. 100% fluorocarbon wax, though expensive, is often used as the final layer on a fully prepped race ski, even at the citizen race level.

**Kick wax:** Applied to the center section of classic skis to provide grip for forward propulsion; waxes are specifically formulated for different temperature ranges—harder for colder snow, softer for warmer snow—and can be rubbed on like a crayon, then melted with an iron. Certain conditions (icy or warm and wet) call for Klister wax—a sticky, glue-like paste squeezed from a tube.

### Clothing

**Fabric and fit:** Cross-country skiing is a highly aerobic sport. Race participants typically wear light, form-fitting, synthetic clothing, although newer merino wool and silk alternatives are available.

**Layers:** Layered clothing is a key strategy to avoid either overheating or excessive cooling that might otherwise be caused by variations in exertion or variations in weather, particularly during training or casual skiing.

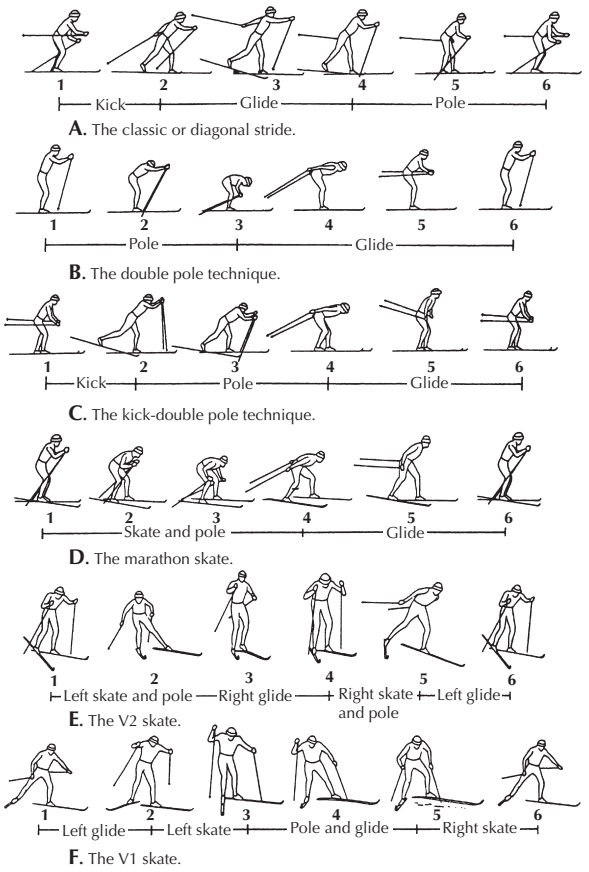



**Figure 78.1.** Cross-country skiing techniques. (From Elmquist LG, Johnson R, Kaplan MJ, Renstrom PA. Alpine skiing. In: Fu FH, Stone DA, eds. *Sports injuries*. Baltimore: Lippincott, Williams, & Wilkins; 1994:481-500.)

## CROSS-COUNTRY SKI TECHNIQUES

- Ski racers use both classic and skating techniques in competitions (Fig. 78.1).
- Both require a dynamic body position, with forward lean and flexion at the waist.
- Both techniques rely on aggressive poling to generate forward propulsion, and therefore, both require good upper body and core strength.
- The technique of ski skating has evolved during the past two decades, with the result that skate skiing technique can be up to 25% faster than classical technique under certain conditions.

### Classic Technique

**Diagonal stride (classic):** The skier keeps both skis in the groomed track, directed straight ahead, and obtains thrust by weighting a ski, engaging the kick wax (or fish scales) for grip and pushing off from the kick zone onto the other ski as it glides forward (see Fig. 78.1). The pole is planted on the opposite side of the kick ski, similar to the arm swing during running, and the body assumes a "diagonal" position relative to the snow.

**Double pole:** Both poles are simultaneously planted, while the skier vigorously flexes at the waist, leaving the elbows bent, and thrusts downward and backward onto the poles as the skis glide forward in the track (see Fig. 78.1).

**Double pole with kick:** Both poles are simultaneously planted as described earlier, but a single leg kick is made as the poles swing forward (see Fig. 78.1).

### Ski Skating Technique

**Description:** Skate skiing requires the skier to push one ski outward with the ski slightly angled, applying force to the inside edge much like an ice skater. Complete weight transfer onto the gliding ski is essential for fast and efficient skiing. Ski skating has different techniques for different terrain, much like changing gears on a bicycle.

**V1 skate:** The technique is asymmetric, with poling accompanying skating on one side but not the other. Double pole with arms in slightly offset position, simultaneously planted with every other step onto the glide ski, and the pole thrust timed with skating push off (see Fig. 78.1); typically used for hill climbing, where poling contributes more than half of the propulsive force; requires good timing, cadence, and body position

**V2 skate:** A symmetric technique with a double pole on every skating push-off, timed just before the step onto the gliding ski; used on flatland or gentle uphill (see Fig. 78.1); requires complete weight transfer, good balance, upper body strength, and aerobic fitness

**V2 alternate skate:** An asymmetric technique with a double pole on every other skating push-off; timing similar to V2 skate, as opposed to V1 skate; used on flatland or downhill at relatively high speed

**Open field skate:** Skate without poles; used on downhill at higher speeds

## TRAINING FOR CROSS-COUNTRY SKI RACING

### Aerobic Capacity of Cross-Country Ski Racers

- Elite-level cross-country ski racers are among the most aerobically fit athletes in the world.
- Olympic gold medal-winning male ski racers have $\dot{V}O_2$ max levels that range 85–92 mL/kg per minute.
- World-class female ski racers have mean $\dot{V}O_2$ max levels in the mid 70 mL/kg per minute range.
- Elite-level masters citizen ski racers in the United States in the 50- to 60-year-old age group have been shown to have $\dot{V}O_2$ max levels in the low-to-mid 60 mL/kg per minute range for men and low-to-mid 50 mL/kg per minute range for women (unpublished data from the University of California–Davis Sports Performance Lab).

### Endurance Training

- Exercise training, in season and out, must be specifically targeted to maximize both aerobic fitness and skiing efficiency (technique) in order to improve race performance. Roller skiing is the most sport-specific off-season training activity, although cycling, ski bounding, and uphill ski walking are also good cross-training modalities.
- Most elite coaches recommend that 70%–90% of the total training volume be dedicated to a mix of long-distance, low- and medium-intensity training at levels below the respiratory compensation threshold (variably referred to as "maximal lactate steady state" or "anaerobic threshold"). The remaining training volume should consist of a progressive incorporation of fast distance training at intensities near race pace (slightly above threshold and with close attention to good technique) and short bouts of high-intensity training to improve $\dot{V}O_2$ max.
- Cardiopulmonary exercise testing on a treadmill using expiratory gas measurements can be used to measure aerobic fitness ($\dot{V}O_2$ max) and ventilatory thresholds and provide specific heart-rate training zones. Lactate threshold testing can be performed either on roller skis or on the snow and can also provide accurate heart-rate training zones.
- Ski racing does require anaerobic efforts during sustained hill climbs and end-of-race sprints, and therefore, skiers should include training to improve this capability.
- Upper body and core strength are key contributors to ski race performance; therefore, sufficient effort must be directed at improving these parameters during the off season. Core strengthening may also reduce the incidence of back pain, which is a relatively common complaint among ski racers.

### Blood Doping

- Three medal winners in the 2002 Winter Olympic cross-country skiing events tested positive for darbepoetin, and seven skiers were disqualified in the 2006 Olympics.
- Skiing is an endurance sport, much like cycling, where the benefits of an elevated erythrocyte mass brought on by use of EPO or autologous blood transfusion can result in a significant competitive advantage.
- Drug testing at the world-class level is aggressively maintained, with fewer disqualifying results in the past decade.

## ENVIRONMENTAL CONCERNS

- Terrain for cross-country skiing ranges from well-groomed flatland skiing in open fields to backcountry alpine wilderness, where hills, obstacles, and uneven snow surfaces factor into play.
- Participants are subject to the effects of altitude, humidity, sun exposure, temperature, and wind chill, each of which can vary during any particular outing.
- In certain backcountry areas, avalanche is a concern.
- Skier awareness to snow conditions, weather, and potential hazards are foremost in the avoidance of environment-related health hazards.

### Sun Injury

**Description:** Up to 85% of ultraviolet (UV) waves reflect off the snow surface, which magnifies the effect of sun on exposed areas of skin and unprotected eyes. Perspiration may decrease the efficacy of sunblock lotions, although "sweat-proof" preparations are available and highly recommended.

### Cold Injury

**Frostnip:** Freezing of superficial layers of skin causing burning pain and erythema that is completely reversed with rewarming

**Frostbite:** Ranges from first-degree injury to epidermis only and no resultant tissue loss to deep penetrating fourth-degree wounds involving subcutaneous tissues, muscle, and tendon

**Corneal freezing:** Affected by wind chill, ground speed, temperature, and humidity; use of goggles or sunglasses is recommended, although evidence for primary prevention is lacking.

**Hypothermia:** Cooling of core temperature with resultant physiological changes, ranging from shivering, tachypnea, and poor judgment to stupor, bradycardia, and death

**Risk factors:** Exposed or inadequately protected skin, wet or inappropriately fitting clothing, altitude, humidity, and wind chill constitute the inherent risk factors for cold injury in skiing. Cold injury is more common in recreational or inexperienced skiers. Prevention strategies include wearing appropriately sized clothing in removable layers; using a base-layer garment designed to "wick" moisture away from skin is recommended. Synthetic fabrics such as polypropylene or naturals such as wool or silk are suitable options for next-to-skin wear. Appropriate nutrition and avoidance of alcohol and dehydration are highly recommended (see Chapter 22: Exercise in the Cold and Cold Injuries).

### Altitude

#### Acute Mountain Illness (AMS)

- Estimated to occur in approximately 20%–25% of individuals with recent ascent above 2500 meters (8000 feet) but is relatively infrequent in nordic skiing, given that most ski areas are at lower elevations
- Acute mountain illness (AMS) may develop in skiers at the lower elevations encountered at ski areas or race events, particularly if the skiers live at low elevation and travel quickly to higher elevations.
- Symptoms in such athletes are usually mild and self-limited. Altitude illness is less common in elite athletes due to experience, acclimatization, and training.
- Backcountry skiers, particularly those who enjoy mountaineering, are at a higher risk of more severe AMS.
- Only a fraction of participants (0.01%–0.05%) develop high-altitude cerebral edema (HACE) or experience high-altitude pulmonary edema (HAPE). Severe symptoms are best treated with rapid descent (see Chapter 23: High-Altitude Training and Competition, for details on diagnosis, treatment, and prevention of altitude illness).

## EPIDEMIOLOGY OF INJURY AND ILLNESS

- Frequency difficult to measure; skiers may not report minor injuries or illness, and certain cross-country skiers seek out remote locations that may lead to a delay or even failure to present for medical care.

- A prospective case study reported an injury rate of 0.72 per 1000 skier days in cross-country skiers compared to an injury rate of 3.4–7.4 per 1000 skier days in alpine counterparts.
- A 2012 study in the Czech Republic reported an injury rate of 0.1 per 1000 skier days over 7 years.
- Most injuries occur during descents.
- Injuries and illness occurring in competitive skiers are common, yet often minor in nature.
- Complaints addressed during or after competition include muscle fatigue and soreness, tendonitis, and minor ligament injuries.
- Medical complaints such as dehydration, cold injury, gastrointestinal problems, and bronchospasm are also common.
- Exercise-induced bronchospasm (EIB) is estimated to occur in >30% of participants; EIB more common in cross-country skiing than any other winter sport.
- Cross-country skiing is a winter sport that is pursued when the risk of viral upper respiratory illness is at its highest. The intense nature of endurance training may also lead to a diminished immune response.
- A common cold may strike just before a highly anticipated race event, negating months of training and resulting in a disappointing race result.
- During race season, skiers should use all common sense strategies to minimize the risk of cold and flu: good hand washing, avoidance of close contact with infectious individuals, a flu shot, and optimization of immune function with adequate sleep and good nutrition.
- If ill, skiers should appropriately reduce their training intensity.

## MUSCULOSKELETAL INJURY

- Lower extremity injuries are consistently reported as more common than upper extremity injuries (approximately 55% vs. 35%).
- Injury type and relative frequency as described in a published meta-analysis are sprains (40%), fractures (27%), contusions (16%), lacerations (9%), dislocations (6%), others (1%).
- Injury frequency and type are significantly influenced by skier experience and terrain conditions.
- Competitive athletes tend toward overuse injuries, whereas less-experienced skiers are at a higher risk of traumatic injuries.
- Impact injuries from falls are most common and are predisposed by icy surfaces and obstacles buried in snow.

## OVERUSE INJURIES

**Upper extremity:** Common injuries to all skiing styles include de Quervain's tendonitis; wrist extensor tenosynovitis; rotator cuff, bicipital, and triceps tendonitis; and medial and lateral epicondylitis. The longer poles used in **skating** predispose skiers to **triceps tendonitis.**

**Lower extremity:** Strains to the hip adductors, internal rotators, and flexors are common, with **adductor strain** being particularly predisposed by **skating** technique. Injuries common to running sports are similarly found in Nordic athletes and include patellofemoral pain, patellar tendonitis, medial tibial stress syndrome, stress fracture, Achilles tendonitis, plantar fasciitis and rupture, sesamoiditis, and hallux rigidus (skier's toe) (Fig. 78.2).

**Trunk and spine:** Low-back pain is a common complaint in competitive skiers, likely related to the aggressive forward-flexed skiing posture. Studies report variable frequencies between classical and skating techniques, with strongly conflicting data. Inappropriate technique, inexperience, and training errors are thought to be contributing factors regardless of the skiing style.

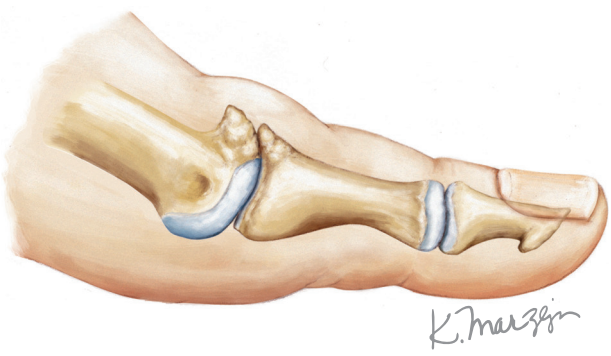
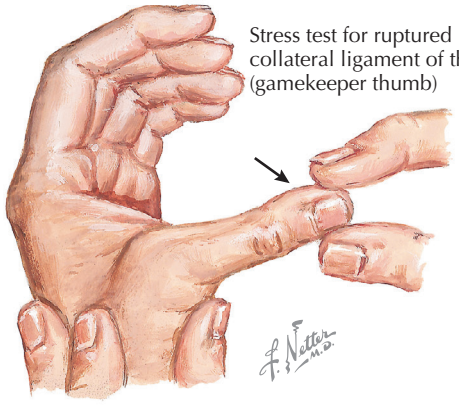
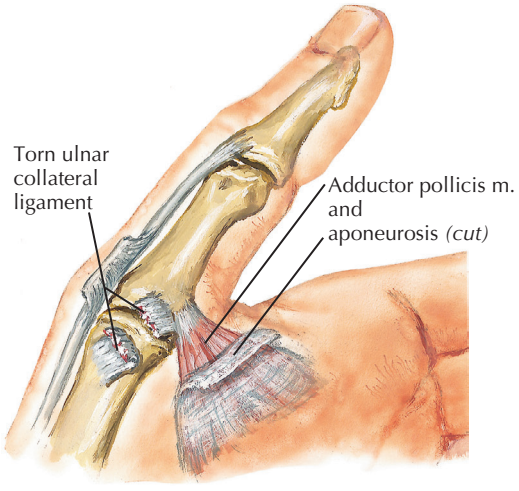


**Figure 78.2.** Skier's toe.




Ruptured ulnar collateral ligament of metacarpophalangeal joint of thumb.

**Figure 78.3.** Skier's thumb.

## TRAUMATIC INJURIES

**Upper extremity:** Mechanism for distal upper extremity injuries is most often a fall onto outstretched hand (FOOSH) or direct trauma and can result in ulnar collateral ligament sprain of the metacarpophalangeal (MCP) joint of the thumb **(skier's thumb),** or distal radius, carpal metacarpal, and phalanx fractures (Fig. 78.3). Proximal upper extremity injuries, often caused by direct shoulder trauma, include fracture of the clavicle, shoulder subluxation and dislocation, and acromioclavicular (AC) joint sprain or separation.

**Lower extremity:** Lower extremity traumatic injuries mimic those found in most high-velocity sports, and the risk is confounded by bulky equipment that may get caught in snow or obstacles, thereby acting as a lever and magnifying forces. Examples include femur fractures, knee sprains (medial collateral ligament [MCL] and anterior cruciate ligament [ACL]), meniscal tear, patellar dislocation, tibial and fibular fractures, ankle sprains, ankle fractures, calf strain, and plantar fascia rupture.

**Trunk and head:** Soft tissue trauma may cause contusions and lacerations. Direct blows to the head can result in mild traumatic brain injury (concussion). Current data suggest increasing use of helmets in snow sports, but current studies focus on alpine skiing and snowboarding. One report suggests a reduction in head injuries in helmeted alpine skiers when traveling at lower velocities, which might thus infer protection for nordic skiers, particularly backcountry skiers and telemark skiers accessing ski lifts.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# SNOWBOARDING

# 79

Thomas R. Sachtleben • Rebecca Ann Myers

## GENERAL PRINCIPLES

### Overview

- Snowboarding was popularized in the 1980s and became an Olympic sport in 1998.
- Snowboarding is one of the fastest growing sports worldwide, and there are >6 million riders in the United States.
- Snowboarders ride on slopes shared with skiers at winter resorts as well as on terrain parks, on half-pipes, and in the backcountry.

### Equipment

- Standard equipment includes a snowboard, bindings, and snowboard boots (Fig. 79.1).
- Snowboard bindings and boots fix the feet to the board and transfer energy forces to the board.
- Snowboards are made of fiberglass with a wood or foam core.
- Most riders wear soft boots, which are comfortable and allow increased movement.
- Hard boots are also available, which have a hard plastic shell similar to ski boots and are designed for increased control and precision of movements.
- Additional safety equipment includes helmets, wrist guards, goggles as well as hip and knee pads.

### Events

- Alpine-style races (parallel or giant slalom)
- Half-/super-pipe
- Snowboard cross—multiple riders race simultaneously through a course of ramps and jumps
- Big-air events—riders jump for maximum height with aerial maneuvers
- Slopestyle—snowboarders race through an obstacle course full of rails and tables

### Biomechanical Principles

- Both feet are positioned nearly perpendicular to the long axis of the board and direction of movement (Fig. 79.2). This prevents the board from acting independently as a lever and applying torque on the knee, which occurs in skiing.
- Riders move with one shoulder and leg leading the way down the slope (see Fig. 79.2). This creates a partial blind side, increasing the risk of collision. Catching the toe or heel edge of the board on snow can cause falls forward onto riders' hands and knees or backward onto the occiput and sacrum, respectively. Approximately 75% of lower extremity injuries involve the lead foot.

### Injury Patterns

- The overall injury rate is approximately 5 per 1000 snowboarder days.
- Falls are the most common mechanism of injury, followed by jumps/landings and collisions.
- Collisions with stationary objects or other skiers/snowboarders account for only about 10% of injuries; 4%–8% of snowboarding injuries involve chairlifts.
- Flexible snowboard boots provide less support than ski boots, making snowboarders more susceptible to ankle injuries.
- Compared with skiing, upper extremity injuries, particularly in novice riders and children, are more prevalent.
- Wrist injuries are 10 times more common in snowboarders than in skiers because of frequent falls backward onto an outstretched arm and hyperextended wrist.
- Head injury rates are three times higher in snowboarders than in skiers. Head and spinal injuries are common and are related to the popularity of aerial acrobatics and jumping.
- Snowboarders are 2.5 times more likely to sustain a fracture than skiers.

### Risk Factors

- It is estimated that approximately half of all injuries occur in beginner snowboarders and that >50% of injured beginners have never received formal instruction.
- Fast skill advancement and progression to aerials and jumping put numerous riders at risk of serious injury. During competitions, snowboard cross has the highest risk of severe injuries.
- Snowboarding often appeals to those seeking risky behavior. Recreational drug use, alcohol, sleep deprivation, using personal music players, as well as a rider's perception of these risk factors, can contribute to injury.
- Additional risk factors include riding on hard, icy, or slushy terrain as well as poor visibility/inclement weather.

## INJURIES AND MEDICAL PROBLEMS

### Upper Extremity Injuries

- Snowboarders reach out with their arms to aid in balance and to brace falls, thus making upper extremity injuries very common. Backward falls cause twice as many fractures as forward falls.
- The shoulder is vulnerable, particularly in advanced riders (acromioclavicular joint separations, shoulder subluxations/dislocations, and clavicle fractures).
- Elbow fractures and dislocations occur frequently, particularly in children.
- Early detection of forearm intraosseous membrane injuries is essential as delayed diagnoses lead to poor outcomes.

### Wrist Injuries

- Wrist injuries occur frequently, particularly in children and beginners; one-fourth of all snowboarding injuries involve the wrist, and approximately 75% of these are fractures.
- Distal radius fractures are particularly common, with approximately two-thirds being intra-articular or comminuted fractures requiring surgical intervention.
- Wrists are often used for speed control as well as pivoting and trick maneuvers. A majority of wrist fractures sustained by

**Figure 79.1.** Snowboarding equipment.

novice riders are a result of falls, whereas most wrist fractures seen in advanced riders are caused by jumping.

## Head Injuries

- Head injuries encountered in snowboarding include skull fractures, concussions, cerebral contusions, diffuse axonal injury, subdural (most common), epidural, and intracerebral hematomas.
- The incidence of traumatic brain injuries among adolescents is increasing.
- Traumatic brain injury is the leading cause of severe injury and death among snowboarders.
- Collisions with stationary objects (e.g., trees or lift poles) account for approximately half of head injuries.
- Acute subdural hematomas are more common in beginners, while fractures, epidural hematomas, and neurologic injuries are seen more often in intermediate and expert riders.

## Spinal Injuries

- Spinal injuries constitute 2%–4% of all snowboarding injuries and are a major cause of permanent disability.
- Most spinal injuries involve compression, burst, or transverse process fractures.
- Half-pipe and "big-air" events are associated with an increased frequency of spinal injuries. Axial loads are transmitted to the spine when landing aerials and jumping in a flexed position. Spinal cord injuries usually occur at the thoracolumbar junction and involve an anterior fracture/dislocation.

## Chest/Abdominal Injuries

- Snowboarders usually sustain injuries to the chest, including rib fractures and pneumothorax, as a result of collisions or falls while jumping.

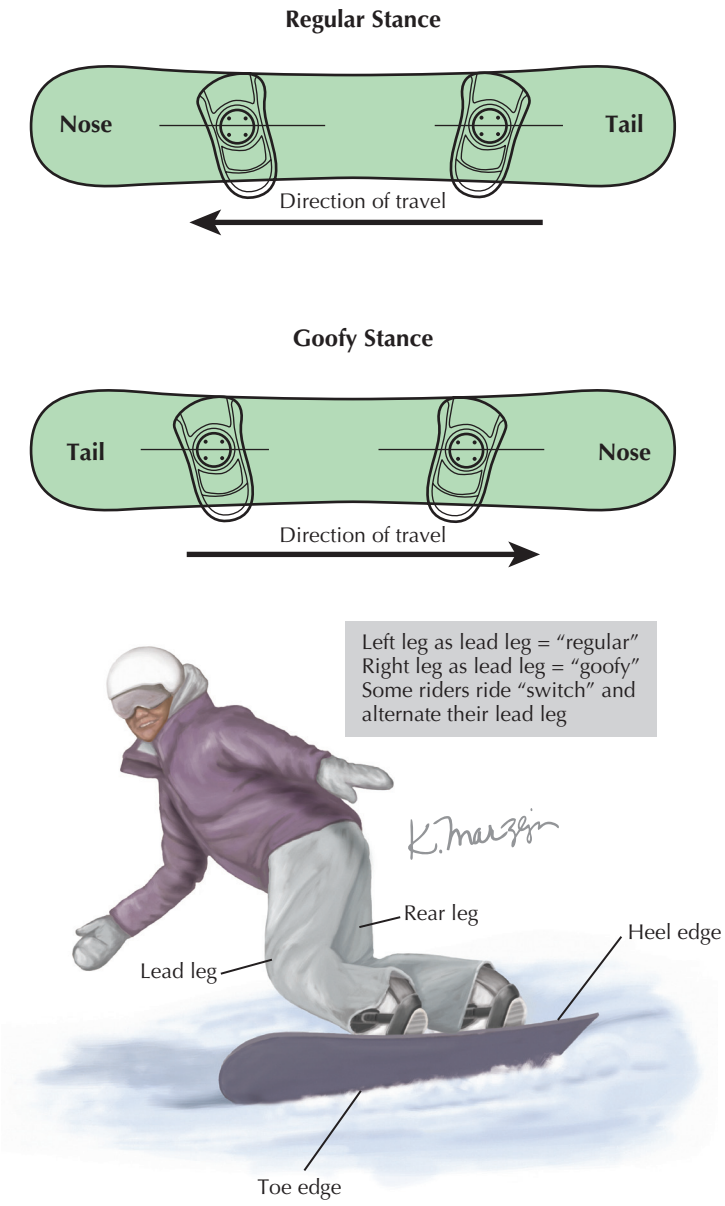


**Figure 79.2.** Snowboard stance.

- Blunt abdominal trauma, including liver lacerations, renal contusions, and splenic injuries, are also common in snowboarders, usually after falling from great heights while jumping.
- The typical mechanism of splenic injury is a snowboarder's own flexed and adducted lead elbow being thrust into his or her abdomen upon hitting the ground with an outstretched hand.
- Contrast-enhanced computed tomography (CT) is best for rapid and accurate diagnosis of intra-abdominal injuries, and a follow-up CT is important when clinical examinations suggest possible delayed splenic rupture. These injuries can be life threatening, and plain radiographs detect an ominous sign such as pneumopertioneum in only 30% of patients with visceral rupture. Ultrasound can be used when CT is not available or in patients who are hemodynamically unstable.
- Abdominal injuries may be initially overlooked because of concomitant distracting upper extremity injuries, head trauma, or when riders are under the influence of alcohol and/or drugs.

## Lower Extremity Injuries

- Ankle injuries make up approximately 15%–20% of all snowboarding injuries, with half of these being fractures, primarily involving the lateral process of the talus (LPT).
  - Approximately one-third of ankle fractures in snowboarders involve the LPT.
  - This fracture is frequently seen in riders wearing soft boots that allow increased ankle flexibility.
- Snowboarders are also at a risk of malleolar bursitis and pseudotumor of the ankle.
- Malleolar bursitis develops as a result of repetitive friction from stiff snowboard boots, while pseudotumors occur due to compression of soft tissues between the lateral malleolus and snowboard boot.
- Only 6% of snowboarding injuries involve the knee. Anterior cruciate ligament (ACL) injuries are infrequent and are primarily seen in advanced snowboarders landing flat after a jump or riding in the terrain park. Novice riders are at a risk of ACL injury while getting on and off lifts because this action requires one leg free for self-propulsion.
- The incidence of hip dislocations in snowboarders is five times higher than in skiers.

### Fracture of Lateral Process of Talus (LPT)

**Description:** This particular injury is often referred to as "snowboarder's ankle" because it is relatively unique to snowboarding. This fracture occurs 15 times more frequently in snowboarders than in the general population. The LPT is a large, wedge-shaped prominence that articulates with both the distal fibula and the posterior calcaneal facet (Fig. 79.3A). It is important for hinge and rotatory movements and has multiple ligamentous attachments.

**Mechanism of injury:** LPT fractures usually occur as a result of sudden dorsiflexion and hindfoot inversion with axial loading, although external rotation is thought to be a key component. Axial loading in this position, along with shearing forces transferred from the calcaneus, often occurs with landing after jumps.

**Presentation:** LPT fractures have similar presentation as lateral ankle sprains. Persistent pain with weight bearing, nonresolving pain, and severely limited range of motion should invoke a high index of suspicion for talar injury.

**Examination:** Swelling is anterolateral, with maximal tenderness inferior to the tip of the lateral malleolus. Significant pain is usually noted with attempts at weight bearing. The LPT stress test is performed by applying a dorsiflexed and everted stress to the foot while compressing the ankle in a cephalad direction and holding the lower leg in external rotation (see Fig. 79.3B, Videos 79.1, 79.2). The LPT is compressed against the posterior calcaneal facet with this maneuver.

**Differential diagnosis:** Similar clinical presentations are seen with a lateral ligamentous sprain, subluxed peroneal tendon, lateral compartment syndrome, subtalar joint sprain, and Salter–Harris type 1 fracture of the lateral malleolus.

**Diagnostics:** The best radiographs for visualization of LPT fractures are the mortise view with 10–20 degrees of inversion or the Broden view. A posterior subtalar effusion should raise suspicion for a LPT fracture. However, the diagnosis of approximately 50% of LPT fractures is delayed because it is not visualized well on standard radiographs (Fig. 79.3C). CT is often necessary to delineate fracture patterns (Fig. 79.3D). Consider advanced imaging in patients with positive Ottawa ankle rules and/or pain at the anterior talofibular ligament (ATFL) in snowboarders. LPT fractures are classified into three widely recognized patterns (Fig. 79.3E): Type 1 fractures involve a small avulsion without extension to the talofibular articulation. Type 2 fractures involve a single large fragment (extending from the talofibular joint to the subtalar joint). Type 3 fractures are comminuted.

**Treatment:** The management of LPT fractures is often challenging. Maintaining appropriate anatomic alignment is paramount to the management of LPT fractures. Type 1 fractures and nondisplaced type 2 fractures (<2 mm or extra-articular) are generally treated nonsurgically with 4–6 weeks of nonweight-bearing cast immobilization. After 2 additional weeks of a weight-bearing cast boot, patients should begin physical therapy to prevent stiff subtalar and tibiotalar joints. Surgical debridement of the LPT may be considered if symptoms persist >6 months. In type 2 fractures (particularly >2 mm or displaced), open reduction and internal fixation (ORIF) has been shown to reduce long-term morbidities (Fig. 79.3F). Excision of small, displaced fragments is usually necessary with type 3 fractures as patients treated with immobilization alone frequently have persistent symptoms. In displaced type 2 and all type 3 fractures, several authors recommend attempting closed reduction, with subsequent cast immobilization. If appropriate reduction is unsuccessful, secondary treatment with ORIF is generally required, followed by 4–6 weeks of nonweight-bearing (Fig. 79.3G). Delayed diagnosis of LPT fractures can lead to nonunion, avascular necrosis, and early degeneration of the subtalar joint. Surgical removal of multiple fragments and/or posttraumatic arthritis may result in instability and ultimately a subtalar arthrodesis. These potential complications can cause significant disability in a young, active population. Fortunately, early diagnosis and aggressive management typically result in good outcomes, with 80% of patients who sustained a LPT fracture returning to their previous sport level.

## Other Risk and Prevention Measures

### Environmental Risks

- Appropriate prevention and protection is necessary to prevent dehydration, hypothermia, and frostbite.
- Protection with sunscreen is particularly important because of high-altitude ultraviolet light exposure.
- Prevention of altitude illness and prompt recognition of symptoms is essential (see Chapter 22: Exercise in the Cold and Cold Injuries).

## Backcountry

- Backcountry use by snowboarders will continue to grow as riders seek more varied terrain and better snow conditions in unpatrolled areas.
- Avalanches claim the lives of numerous snowboarders each year, usually from suffocation and/or blunt trauma.
- Deep snow immersion can also cause asphyxiation as a result of a fall into a deep snowbank or tree well. Tree wells are deep depressions of loose snow that form around the base of a tree with low branches. Snowboarders who fall headfirst into a tree well or deep snowbank can be easily trapped and suffocate in as little as 15 minutes. These fatalities, also known as *nonavalanche-related snow-immersion deaths (NARSIDs)*, have become an increasingly recognized fatality pattern in snowboarders. Certain backcountry riders use strapless, releasable ("click-in") bindings, or hand-accessible release cords to both bindings in an effort to get their board off quickly in emergent situations.
- Skill, knowledge, good judgment, and appropriate safety gear are crucial for safe travel in the backcountry. Essential gear includes an avalanche transceiver, shovel, and probe as well as appropriate avalanche training. Avalanche airbags (self-deployed bladders of air that can be used to keep avalanche victims closer to the surface) are gaining popularity, and certain riders use a device called an *AvaLung* that uses a shoulder sling, valve box, and tubing that allows fresh air intake and $CO_2$ displacement when buried. Backcountry riders should never ride alone.

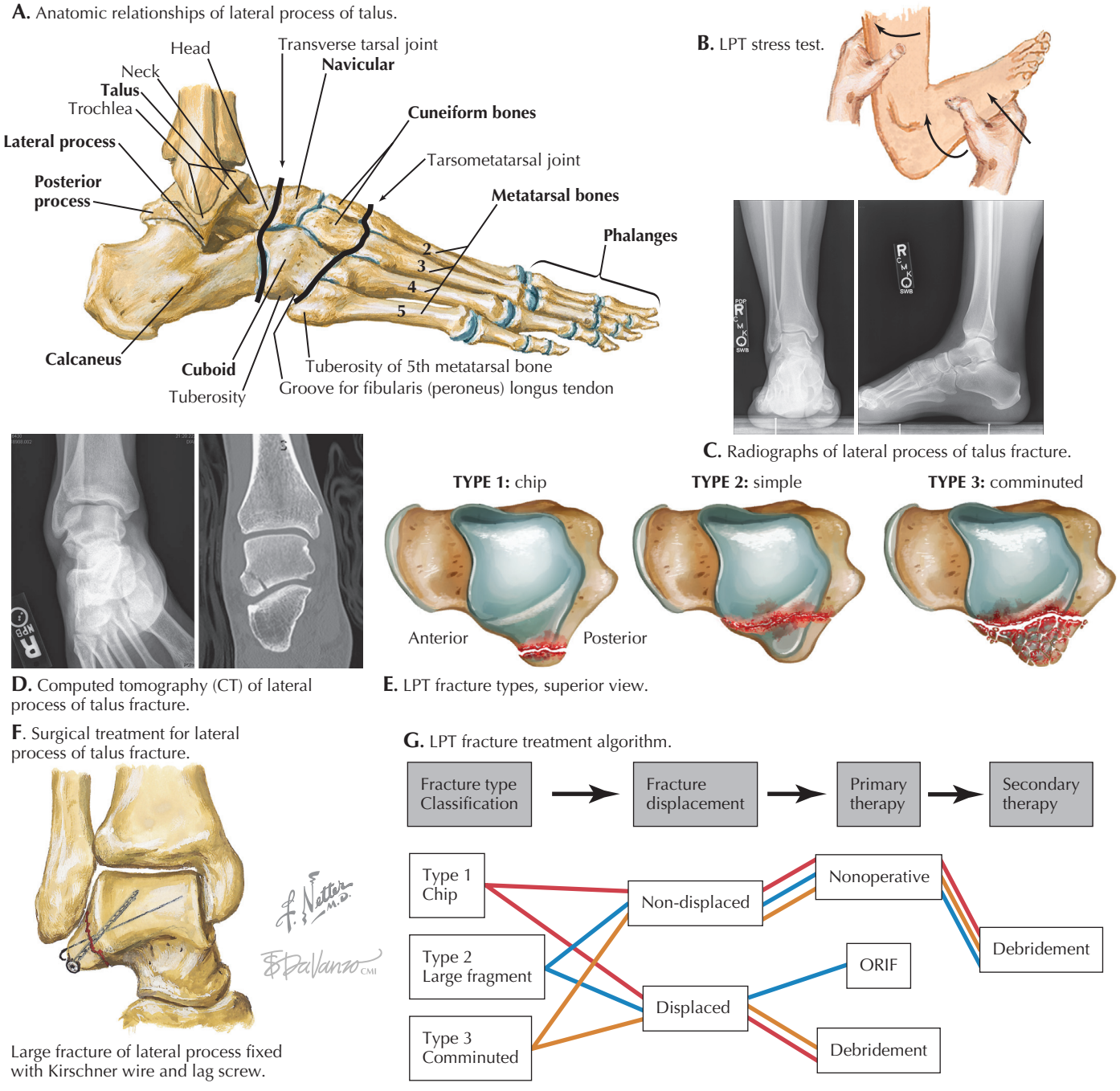


**Figure 79.3.** Fracture of lateral process of talus. (Modified from Valderrabano V, Perren T, Ryf C, Rillmann P, Hintermann B. Snowboarder's talus fracture: treatment outcome of 20 cases after 3.5 years. *Am J Sports Med.* 2005;33[6]:871-880.)

## Injury Prevention

- Effective education and risk awareness is paramount, particularly for young riders and beginners.
- Helmet use is recommended and has steadily increased over the past 15 years; however, studies designed to evaluate the effectiveness of helmet use in reducing injury have reported mixed findings.
  - Helmets reduce impact forces and can reduce head injuries such as skull fractures, but most do not markedly reduce acceleration/deceleration and rotational force injuries.
  - Education regarding the limitations of helmets is important, yet they are still strongly encouraged for all snowboarders. Helmets should fit appropriately. There is no increased risk of cervical spine injury in riders who wear helmets.
- Wrist guards are beneficial in preventing serious wrist injuries, particularly in children and beginner riders; unfortunately, only <20% of riders wear them.
  - Risk of injury is approximately three times higher in those who do not wear wrist-protection devices.
  - Wrist guards made of high-density plastic are recommended, and some are incorporated into the gloves.

  - They must be flexible enough to absorb as much energy as possible in order to avoid forces at the proximal and distal ends of the brace, which can increase the risk of fracture.
  - Despite the concern that the use of wrist protectors may transfer injury to other forearm locations, studies have not shown this to be true.
- Further research is needed to evaluate the potential benefits of core strength and neuromuscular training, as well as cardiovascular fitness, in injury prevention.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- Video 79.1, Anatomical Relationships of LPT Fracture
- Video 79.2, LPT Stress Test Demonstration

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 80 ICE HOCKEY

Robert Johnson

## HOCKEY ORGANIZATION AND PARTICIPATION

- **USA Hockey,** located in Colorado Springs, Colorado, is the national governing body for ice hockey in the United States (US) and the official representative of US Olympic Committee and International Ice Hockey Federation. USA Hockey works in conjunction with the National Hockey League (NHL) and the National Collegiate Athletic Association (NCAA).
- During 1968–1969, 3800 teams were registered with USA Hockey. During 1993–1994, 21,150 teams with approximately 340,000 players were registered. Between 2005 and 2006, over 442,000 players and 58,000 coaches were registered. For 2014–2015, USA Hockey reported over 530,000 male registrants. Of these, about 36,000 are boys aged ≤18 years. According to the National Federation of High School Associations (NFSHA), 1603 boys' teams representing 17 states and almost 36,000 players participated in interscholastic hockey.
- Women's hockey continues to grow in popularity. The number of female hockey players registered in 2014–2015 was almost 70,000. Of these, almost 52,000 were ≤18 years. NFHSA data show 615 girls' high school hockey teams representing 12 states with almost 19,000 participants. Between 1995 and 1996, 38 women's teams competed in an interscholastic competition held in Minnesota. During 2014–2015, 245 teams competed. A first state high school tournament for girls was held in Minnesota in 1995. Despite the growth in women's hockey, in a few states, girls are still competing in boys' youth hockey teams.
- The NCAA has 36 women's teams competing in Division I and 62 in Division III. For men, the NCAA reports 61 teams competing in Division I and 72 in Division III. There are no Division II ice hockey teams.
- The Canadian Hockey Association, or Hockey Canada, represents the governing body for amateur hockey in Canada. They have over 600,000 registered players. Of these, approximately 522,000 are males.
- **Age range of organized competition: 5 to >50 years**
- **Age group divisions** are determined by birth date as of August 31 of each year (Table 80.1).
- **Regarded by most as the fastest competitive team sport**

## GAME OF ICE HOCKEY

### Structure

- **Professional, college, adult:** three 20-minute periods
- **High school:** three 17-minute periods
- **Youth:** three 12- to 15-minute periods

### Team Composition

- Eighteen players and two goalkeepers (usual position distribution). Six players compete at one time: three forwards, two defensemen, and one goalie.
- **Goalkeeper (goalie):** The player who tends the goal to catch or deflect the puck and prevent the opponent from scoring
- **Forwards (left wing, center, and right wing):** Offensive-minded players who attack the opponent with an intent to score a goal; also assist defensemen in protecting their goal
- **Defensemen (left and right):** Primary responsibility is to protect their goal and the goalie to prevent the opponent from advancing to the net to score
- A **substitution** may occur during play ("on the fly") or during time stoppages for violations, goals, or penalties.

### Rink

- Should be 200 by 100 feet; smallest recommended dimensions are 185 by 85 feet
- Should be surrounded by wooden or fiberglass boards 40–48 inches high with a yellow or light-colored kickplate at bottom; it is recommended that a safety glass or another protective screen encircle the rink.
- The goal should have dimensions of 4 feet high by 6 feet wide and should have metal goalposts and a crossbar and net surrounding the metal framework.

### Special Equipment

- The **puck** is vulcanized rubber, 1 inch thick and 3 inches in diameter, weighing 5.5–6 ounces.
- **Hockey stick**
  - Forwards and defensemen: the blade is usually made of wood (the shaft may be made of other materials) with the shaft <63 inches, blade <12.5-inches long by 2–3-inches wide, and curve not exceeding 0.75 inches
  - Goalie: wood shaft <63 inches with the blade <15.5-inches long by 3.5-inches wide, and curve not exceeding 0.75 inches

### Skills

- The skill in **skating** involves three factors: angle of propulsion (angle formed by the skate blade in the direction of the skate), angle of forward inclination (body lean), and length of the stride.
- **Shooting**
  - **Types of shots: standing wrist shot** (sweeping action with the stick terminating in wrist snap and follow-through), **skating wrist shot** (similar to a standing wrist shot except that a player has forward momentum while skating; most accurate), **standing slap shot** (the stick and blade are brought back a variable distance, followed by a vigorous forward motion, and "slapping" at the puck like a golf swing; least accurate), and **skating slap shot** (greatest velocity)
  - **Maximal velocity** is the result of the strength of arm and shoulder muscles and full trunk rotation.
- **Passing**
- **Stick handling:** Ability to advance the puck while maneuvering on ice
- **Checking:** Intentional contact with an opponent who is in possession of the puck, using the hip or shoulder; a player may check from the side, diagonally or frontally, approaching with no more than two skating strides. In 2011, USA Hockey changed the age at which checking could be introduced from

**TABLE 80.1 AGE GROUP DIVISIONS DETERMINED BY USA HOCKEY**

| Level | Boys (Years) | Girls (Years) |
|---|---|---|
| Mites | ≤8 | |
| Squirts | ≤10 | 6–12 |
| Pee Wees | ≤12 | 13–15 |
| Bantams | ≤14 | |
| Junior Midgets | ≤19 | 16–19 |
| Junior | ≤19 | |
| Senior | >19 | >19 |

PeeWee to Bantam based on injury data showing decreased injury and concussion rates at the PeeWee level.

- **Goal tending:** The goalkeeper (goalie) tends the goal and is protected by special equipment and pads to catch or deflect the puck from the goal.

## Safety and Protection

### Protective Equipment

- Goalie: helmet, mask, throat protector, chest protector, cup, thick padded shin guards, blocker (worn on one hand), trapper (device to catch the puck, worn on the opposite hand), skates that are unique to protect the goal and goalie
- Forward and defense: helmet, shoulder pads, elbow pads, padded gloves, cup, breezers (padded hockey pants to protect the sacrum, coccyx, and pelvis), shin guards, and skates
- **Face masks**
  - Full face masks required at youth and high school levels in 1975; Eastern Collegiate Athletic Conference mandated use in 1977; NCAA required use in 1980; helmets required in NHL; visors (half face shields) were required for all players new to the NHL as of the 2013–2014 season. For players entering the NHL before 2013–2014, face masks (visors) remain optional.
  - **Effects of full and half face shields** (college level, Canada):
    - Full shield: 61.6% had at least one injury
    - Half shield: 63.2% had at least one injury
    - Risk of facial, dental injury: 2.3 times greater with half shield
    - Risk of concussion: Concussion rates are higher in those wearing the half shield compared to those wearing the full face mask.

### Rules to Protect Players

- Penalties: 2 minute (minor), 5 minute (major), 10 minute (major), or a combination
  - Offending player must sit in a designated penalty box and his or her team must play with one less player on ice ("short-handed"). If two penalties are assessed against the team, it must play two players short. Team never has to play more than two players short.
  - For 10-minute penalties, offending team does not have to play shorthanded. They lose services of that player for that time interval.
  - Single or multiple game disqualifications may be assessed, depending on severity of infraction.
- Goaltender protection: no unnecessary body contact with goalie; the "crease" is the goalie-protected area in front of goal where opposing players cannot enter without a puck. If a goaltender loses his helmet, the play is immediately stopped.
- Common penalties enforced for protection of players:
  - **Cross-checking:** using shaft of stick with both hands to check opponent
  - **Hooking:** using blade of stick on opponent's body to block or impede opponent's progress
  - **Slashing:** striking or attempting to strike opponent by swinging stick
  - **Spearing:** poking or attempting to poke opponent with blade of stick
  - **Interference:** impeding progress of opponent not in control of the puck
  - **Charging:** using more than two skating strides to check the opponent
  - **Checking from behind**
- Officiating: Three to four officials enforce rules, assess penalties, and award goals

## PHYSIOLOGY OF ICE HOCKEY

### Skating Stride

- **Three phases:** Glide during single-support propulsion; during single-support propulsion; and during double support
- **Propulsion:** When extending knee joint in skating thrust, quadriceps develop largest contractile force. Hamstrings and gastrocnemius stabilize knee during weight shift and push-off.
- **Stride rate is related to skating velocity.** Stride length is unrelated except in young hockey players.
- Faster skaters show better timing in push-off mechanics with resultant push-off in the direction perpendicular to the skating direction. Elite skaters sustain the gliding phase longer.
- In players aged 8–15 years, increases in velocity are accompanied by increases in stride length and no significant change in the stride rate.
- To accelerate quickly, players should attempt full extension of hip, knee, and ankle.
- With fatigue, decrease in skating velocity is caused by decreased stride rate (slower leg extension and longer glide phase) and excessive forward lean.
- Typical game skating behavior is a complex activity involving repeated accelerations, decelerations, turning, and stopping. Complicating skating behavior are upper body activities of stick handling, shooting, passing, and checking.

### Physical Characteristics of Hockey Players

- **Professional players** are taller and heavier on average than college and junior players.
- **Defensemen** are taller and heavier than forwards.
- **Body composition (% fat)**
  - Junior: 8.6%–13.6%
  - College: 8.6%–10.7%
  - Professional: 9.7%–14.2%
  - Forwards and defensemen have equal body compositions.
  - Goalies, on average, have higher body composition than forwards and defensemen.

### Energy Expenditure

Physiology has been primarily studied in adult, elite hockey players, which underscores uncertainty of applying this science to youth hockey.

#### Game

- Shifts
  - One shift averages 45–90 seconds, with an average of 2–3 play stoppages/shift, lasting an average of 27 seconds.
  - Average playing time/shift is 40 seconds with recovery of 225 seconds between shifts.

  - One shift plus recovery averages work capacity of 32 mL/kg/minute (66% of $\dot{V}O_2$ max).
  - Average player plays 14–21 shifts each game with an average playing time of 21–28 minutes/game (based on usual practice of alternating three "lines").
- Energy requirement estimated at two-thirds anaerobic metabolism and one-third aerobic metabolism. On-ice energy requirements of college players is estimated at 70%–80% $\dot{V}O_2$ max and youth hockey players is estimated in excess of 80% $\dot{V}O_2$ max.
- On-ice heart rate averages 152 beats/minute.

### Time–Motion Analysis

- Adult elite players average 6400–7200 meters/game (3.9–4.4 miles/game)
- Forwards demonstrate more anaerobic activity than other positions; aerobic system primarily used for recovery
- Defensemen have longer playing time (33%), more shifts (17%), and longer playing time/shift (21%) but less recovery time between shifts (35%); defensemen average approximately 62% of skating velocity of forwards
- Goalie's quick, explosive movements of short duration with rest periods of submaximal activity use primarily adenosine triphosphate-phosphocreatine (ATP-PC) energy system
- However, few physiologic studies of youth hockey (older age groups) had similar findings.
- Adult recreational hockey players tend to stay on ice much longer per shift.
- Time–motion analyses are based on use of alternating three lines. In adult recreational leagues, only two lines may be used. At collegiate and professional levels, four lines may be used.
- Heart-rate telemetry estimates on-ice intensity averaging 70%–80% $\dot{V}O_2$ max during a 60-minute stop-time game. For 30 minutes of each game, players' $\dot{V}O_2$ max exceeds 90%. Adult recreational players average heart-rate intensity in excess of 70%.

### Muscle Glycogen Stores (Energy Source)

- Glycogen stores decline by an average of 60% for forwards and defensemen after one game.
- All muscle fibers (types I, IIa, and IIb) contribute glycogen; type I depletes (contributes) most.
- Two-fold increase in plasma free fatty acids suggests mild glycogen-sparing effects in the muscle.
- Consecutive-day games usually do not allow complete repletion of glycogen stores (based on diet as desired).

### Lactate Accumulation

- Because anaerobic glycolysis is a major energy contributor, lactate accumulates over course of the game (8- to 10-fold increase). Because approximately 10 minutes are required to remove lactate from exercising muscle, there is inadequate time between shifts for full recovery. Result is mild metabolic acidosis.
- Lactate values usually higher in first and second periods; forwards and defensemen have similar levels
- Levels actually lower than predicted because each shift is interrupted by average of two to three play stoppages, averaging 27 seconds; this usually allows approximately 60%–65% of phosphocreatine to be resynthesized before the next shift.

### Muscle Fiber Type

- Wide range of fiber composition
- No difference from general population; no position-to-position variation

### Anaerobic Power and Endurance

- Forwards, defensemen, and goalies have similar results in peak power and endurance.
- Similar results occurred when younger, less-experienced players were tested.

### Aerobic Endurance

- Although hockey is largely anaerobic, improving aerobic capacity reduces fatigue and may enhance performance. Involvement of anaerobic system may depend on efficiency of the aerobic system.
- $\dot{V}O_2$ max ranges 52–62 mL/kg/minute. Maximum aerobic capacities of youth hockey players are similar to those of adult players when adjusted for size and weight.
- NHL players have shown consistent increase in aerobic capacities over past 15 years presumably because of more effective off- and in-season conditioning strategies.

### Muscle Strength and Endurance

- Professional players were stronger than amateurs based on each of the six tests used for comparison.
- Comparing defensemen and forwards at similar levels, data relative to body weight showed these to be equal.
- Compared with other sports, hockey players obtained high levels for total and relative leg force. Only elite canoeists and athletes from power events scored higher (Finnish study).

### Flexibility

- Forwards and defensemen have similar flexibility.
- Goalies have significantly better flexibility (key element for that position).
- In general, flexibility of hockey players exceeds that of other elite athletes in wrist, hip, knee, and ankle.
- Other elite athletes exceed hockey players in flexibility on neck rotation, all shoulder and elbow actions, trunk extension-flexion, and lateral flexion.

### Fatigue

- Hockey players at risk of fatigue; activities of ice skating require use of all major muscle groups. Hockey has heavy metabolic demands for energy *and* removal of waste products of energy metabolism.
- Studies of fatigue in hockey show failure of return of maximal muscle contractions to pre-exercise levels at 24 hours. Loss of ability to generate maximal force affects athlete's ability to perform peak-force activities to accelerate, stop, and turn.

### Detraining

- On-ice practice and game play may not provide sufficient stimulus to maintain or improve fitness among hockey players.
- Studies suggest that additional aerobic activities may be necessary during competitive seasons.

### Practical Application of Training Studies

- Programs that have **failed** to improve skating speed: leg squats using weights; pushing partner as technique of resistance skating; speed skating with instruction; skating with ankle weights
- Six-week preseason training program consisting of continuous running, stair running, flexibility, and strength training resulted in 11% increases in $\dot{V}O_2$ max. During subsequent season, gains in oxygen consumption were lost in absence of any specific in-season aerobic training program.
- Hockey training stimulates improvement in cardiovascular conditioning similar to that of continuous training programs in untrained players. In fit elite players, there were no improvements in cardiovascular fitness over course of the season.
- Hockey practice observations show 20 minutes of actual skating during 60-minute practice. Heart rate monitoring, however, provided sufficient stimulus for aerobic training effects.

- Anaerobic endurance improved over course of the season by approximately 16% but not associated with increases in glycolytic enzymes
- Muscular fatigue over 6-day routine of practices and games showed decrements in maximal voluntary muscle contractions, implying fatigue. Levels decreased through first 3 days, then reached plateau at a level lower than the baseline. After hockey practice, muscle output remains diminished over the practice–game cycle.

### Nutrition of Hockey Players

- Dietary composition: protein, 14%–20.5%; carbohydrate, 38%–44%; and fat, 34%–43%
- Average daily intake is 2800–4900 calories.

### Environmental Factors

- Ice arenas usually have lower ambient temperatures than other athletic settings, which minimize the risk of heat-related injury.
- Hockey protective equipment reduces the ability to dissipate heat.
- Despite hydration between periods and shifts, hockey players lose 2–3 kg body weight through sweat each game.

### Physiologic Studies and Their Implications for Shift Length

- Shorter shifts result in higher contribution of phosphocreatine and oxidative phosphorylation to ATP (energy source) turnover, reducing contribution of anaerobic glycolysis, which reduces consumption of muscle glycogen.
- Shorter shifts result in less lactate accumulation in exercising muscles. Lactate accumulation causes muscles to be inefficient and fatigue more readily. If lactate levels are lower, lactate clears more quickly, and muscles recover more quickly.

## INJURIES IN ICE HOCKEY

### Epidemiology

- **Incidence and rate:** In NHL, most recent data 2011–2012 season; injury incidence density 39.4 (per 1000 hours actual ice time) compared to 2007–2008, injury incidence density, 68 (Table 80.2)
  - NHL defensemen injured more often than forwards; goalies have greater time loss with injury than other positions.
- Collegiate injuries (Table 80.3)
  - Youth hockey injuries (Canada, 15-year study; Tables 80.4 and 80.5)

**TABLE 80.2 NHL INCIDENCE RATE AT SITE OF INJURY AND MECHANISM**

| Injury Site | Incidence Rate |
|---|---|
| Head | 17% |
| Thigh | 14% |
| Knee | 13% |
| Shoulder | 12% |
| Ankle | 6% |
| Foot | 6% |
| Pelvis/low back | 5% |
| **Injury Mechanism** | **Incidence Rate** |
| Checking | 28.2% |
| Incidental contact | 14.3% |
| Puck | 13.5% |

- Age group differences
  - Age 11–14 years: 1 injury/100 hours playing time
  - Age 15–18 years: 1 injury/16 hours playing time
  - Age 19–21 years: 1 injury/11 hours playing time
  - Professional: 1 injury/7 hours playing time
  - In Finland, youth hockey injuries occurred with incidence similar to youth soccer and alpine skiing.
  - Under the age of 12 years, injury was infrequent. Beyond 12 years, injuries increased evenly over older age groups.
- Average injury risk of *all* sports is 1.37%. Hockey has an average incidence of 2.71% compared with an average risk of 3.95% in soccer.
- One study at elite level showed 5% of all injuries were related to fighting.
- **Catastrophic injury rate:** 2.55 per 100,000 compared with football rate of 0.68 per 100,000; rules infractions related to 17% of all catastrophic injuries
- **Injuries per player per year:** youth hockey, 0.02; professional, 3.0

**TABLE 80.3 COLLEGIATE INJURIES (BASED ON DATA FROM 2000/01 TO 2006/07 SEASONS)**

| | Men (injury rate reported per 1000 athlete exposures) | Women (injury rate reported per 1000 athlete exposures)* |
|---|---|---|
| Type | Game: 18.7<br>Practice: 2.23 | Game 12.1<br>Practice 2.9 |
| Position | Goalie 2.7<br>Defense 5.0<br>Forward 5.1 | Goalie 14.0%<br>Defense 41.2%<br>Forward 44.7% |
| Period | First 15.1<br>Second 15.1<br>Third 11.2 | N/A |
| Location | Home 11.9<br>Away 15.6 | N/A |
| Site | Knee/leg 22%<br>Head 19%<br>Shoulder 15%<br>Foot/ankle 12%<br>Hip/groin 9%<br>Back/spine 9%<br>Wrist/hand 7%<br>Other 7% | **Game/practice†**<br>Head/neck 25.4/16.2<br>Upper extremity 30.3/22.2<br>Trunk/back 11.4/26.4<br>Lower extremity 31.8/31.1<br>Other 1.1/4.2 |

*Checking is not permitted in women's hockey. Injuries are the result of "incidental" contact (collisions with other players, the boards, the ice, and the goal), the stick, and the puck. Fifty percent of injuries occur as a result of collisions.
†Concussion is the most common injury reported in both practices and games.
*N/A,* Statistics not available.

**TABLE 80.4 YOUTH INJURIES**

| Years | Female (%) | Male (%) |
|---|---|---|
| Age 7–8 | 2.8 | 2.9 |
| Age 9–10 | 9.3 | 8.6 |
| Age 11–12 | 21.2 | 22.1 |
| Age 13–14 | 34.7 | 35.6 |
| Age 15–17 | 32 | 30.8 |

### TABLE 80.5 COLLEGIATE INJURIES BY INJURY SITE, MECHANISM, AND TYPE

| Injury Site | Female (%) | Male (%) |
|---|---|---|
| Upper extremity | 39.2 | 45.2 |
| Head/neck | 28.4 | 25.1 |
| Lower extremity | 23.2 | 21.4 |
| Abdomen/thorax | 25.3 | 17.8 |
| **Injury Mechanism** | **Female (%)** | **Male (%)** |
| Collision | 28.6 | 24.6 |
| Checking | 25.7 (illegal) | 42.8 |
| Falls | 25.3 | 14.8 |
| Other (puck, stick) | 20.4 | 17.8 |
| **Injury Type** | **Female (%)** | **Male (%)** |
| Soft tissue | 39.8 | 32.6 |
| Sprains/strains | 21.1 | 17.6 |
| Fractures | 18.2 | 27.1 |

### TABLE 80.6 PRACTICE VERSUS GAME (INJURIES/1000 PLAYERS): COLLEGE INJURY COMPARISON

| Sport | Practice | Game | Total |
|---|---|---|---|
| Men's basketball | 5.1 | 9.5 | 6.0 |
| Men's gymnastics | 4.7 | 14.8 | 8.9 |
| Wrestling | 6.8 | 28.1 | 8.9 |
| Hockey | 2.23 | 18.7 | 5.0 |

- **Descriptive injury data**
  - 24%–45% of all injuries occur during practice (Table 80.6).
  - **55%–76% of all injuries occur during games:** first period, 20.5%–31%; second period, 30%–38%; and third period, 28%–46.2%
  - In youth hockey, the rate of injury in games is four to six times the incidence in practices. The higher game rates occur at older, more competitive levels. Injury rates increased at ages when checking is introduced. House leagues had lower injury rates than the equivalent travel (more competitive) leagues.
  - **Injury incidence—time in period:** 0–7 minutes, 12.5%; 7–15 minutes, 40.6%; and 15–20 minutes, 46.9%
  - **Acute versus overuse:** acute, traumatic, 80%; overuse, 13.5%–20%
  - **Location on ice:** 40% in the defensive zone, 35% in the offensive zone, 25% in the neutral zone
  - **Injury by position:**
    - Defensemen (107.8 per 1000 game hours): 55% minor, 30% moderate, 15% severe
    - Forwards (71.8 per 1000 game hours): 73% minor, 21% moderate, 6% severe
    - Goalies (39.2 per 1000 game hours): 83% minor, 17% moderate, 0% severe
  - Male and female injury rates are similar, in spite of "no-check" rules in women's hockey.

### TABLE 80.7 MECHANISM OF INJURY IN ELITE ATHLETES

| Mechanism | Beiner et al. 1973 (%) | Lorentzon 1988 (%) |
|---|---|---|
| Stick | 25 | 11.8 |
| Puck | 17 | 14.5 |
| Collision* | 17 | 57.9 |
| Skate | 6 | 2.6 |
| Other | 36 | 13.2 |

*33% injuries in adult hockey with 14% of collisions unintentional.

### TABLE 80.8 MECHANISM OF INJURY IN COLLEGE ATHLETES

| Mechanism | % |
|---|---|
| Legal check | 44.6 |
| Accidental collision | 28.6 |
| Illegal stick check | 12.2 |
| Fighting | 6.5 |
| Illegal check | 5.8 |
| Noncontact | 2.2 |

### TABLE 80.9 MECHANISM OF INJURY IN YOUTH (SMALL STUDIES)

| Mechanism | % |
|---|---|
| Collision* | 50-86 |
| Puck | 14.3 |
| Overuse | 14.3 |
| Stick | 7.0 |
| Skate | 7.0 |

*10% unintentional collision
In one study, illegal checks and violations caused 66% of injuries, but penalties were assessed only 14% of the time.

- **Injury potential**
  - **Collisions** with players, boards, and goalposts
  - **Skating velocity** (examples): senior amateur players, 30 mph (48 km/hour); PeeWee (age 12–13 years), 20 mph (32 km/hour)
  - **Sliding velocity** (after a fall): 15 mph (24 km/hour)
  - **Hockey puck** (6 ounces [170 gm] hard rubber) shooting velocity: professional, 120 mph (192 km/hour); senior recreational, 90 mph (144 km/hour); and PeeWee (ages 12–13 years), 50 mph (80 km/hour)

    Maximal impact force of the puck at its terminal velocity is 1250 pounds (567.5 kg). Hockey masks deform at puck speeds of 50 mph (80 km/hour).
  - **Hockey stick** velocity is measured at 100–200 km/hour during shooting.
  - **Hockey skates** often cause lacerations from sharp, steel blades.
  - **Nonimpact forces:** vertical reaction force during skating stride is 1.5–2.5 times body weight compared with 3–4 times body weight in runners; posterior push force during skating measured at 150 pounds.
- **Mechanism of injury** (Tables 80.7 to 80.9)

- **Anatomic sites** (Table 80.10)
- **Type** (Tables 80.11 and 80.12)
- **Severity**
  - **Minor (>7 days' absence):** 61%–73% (46% of all minor injuries caused by body checks)
  - **Moderate (8–30 days' absence):** 19%–22%
  - **Severe (>30 days' absence):** 8% (75% of all severe injuries caused by body checks)

**TABLE 80.10 ANATOMIC SITE OF INJURIES**

| Site | % |
|---|---|
| **Professional*** | |
| Head, scalp, and face | 28.1–52.9 |
| Eye | 2.6 |
| Shoulder | 5.6–21.9 |
| Hand | 2.1–10.5 |
| Thigh (groin) | 15.3–35.7 |
| Knee | 11.6–17.0 |
| Miscellaneous (back, foot/ankle, ribs) | 3.6–23.3 |
| **College** | |
| Knee | 18.6 |
| Face, eye, mouth, teeth | 17.6 |
| Shoulder, clavicle | 14.9 |
| Head, neck | 10.6 |
| Thigh, hamstring | 9.0 |
| Forearm, wrist, hand | 6.9 |
| Hip, groin, abdomen | 6.4 |
| Chest, back | 4.8 |
| Arm, elbow | 3.7 |
| Ankle | 3.2 |
| **Youth†‡** | |
| Head and neck | 10–23 |
| Upper body | 23 |
| Shoulder/arm | 19–55 |
| Trunk | 13–17 |
| Leg | 17–19 |

*Range, four studies.
†Range, two studies.
‡At Bantam level (ages 13–14 years), weight differences of 53 kg and height differences of 55 cm have been reported. Smaller players are more likely to be injured.

**TABLE 80.11 TYPES OF INJURY**

| Type | % |
|---|---|
| **Adult (Elite)*** | |
| Contusion | 25–47 |
| Laceration | 28–50 |
| Fracture† | 4–15 |
| Dislocation | 1–8 |
| Muscle, ligament | 3–12 |
| Other | 3–5 |
| **College** | |
| Sprains, dislocations | 22 |
| Contusions | 20 |
| Lacerations | 13 |
| Strains | 11 |
| Fractures | 10 |
| Concussions | 8 |
| General trauma | 6 |

*Range of three studies.
†Fractures are 12 times more common in leagues with checking.

- **Ice surface size and injury (Junior A)**
  - Injury rates: inversely proportional to ice surface size
  - Neurotrauma: no relationship to ice surface size
  - Aggressive penalties: no relationship to ice surface size
- **Incidence and severity of injuries is increasing.** Possible explanations: increased participation, increase in speed of game and size of players, longer seasons and more out-of-season participation at all levels, lack of appropriate training, and inconsistency in rule enforcement

## Acute Traumatic Injuries

Acute traumatic injuries account for 80% of all hockey injuries.

### Head

- Full spectrum of injury due to closed head trauma, including death
- **Concussions account for 8%–28% of all hockey injuries** with the highest incidence at the high school level. In males, data from the National High School Sports-Related Injury Surveillance System show the incidence of concussion in ice hockey ranks second behind football. Football has a game concussion rate of 22.9/10,000 athlete exposures (AEs), a practice concussion rate of 3.6/10,000 AEs accounting for 17% of all reported injuries. Hockey has a game concussion rate of 14.6/10,000 AEs, a practice rate of 1.1/10,000 AEs, representing 22% of all reported injuries. Lacrosse (10.4, 1.1), soccer (5.3, 0.4), and wrestling (4.8, 1.3) game and practice rates range third to fifth in concussion. Similar data are unavailable for concussion rates in ice hockey for girls.
- Age-group concussion incidence (meta-analysis): 5–14 years, 0.0–0.8 per 100 player hours; high school, 0.0–2.7 per 1000 player hours; college, 0.2–4.2 per 1000 player hours; and elite adult, 0.0–6.6 per 1000 player hours
- Frequency of closed head trauma has decreased because of mandatory use of helmets.
- Increased angular velocity of head and neck with helmet and facemask does not appear to increase head or neck injury risk.
- In an evaluation of sports-related head injuries reported from emergency departments from 1993 to 1999 for the sports of ice hockey, soccer, and football, the overall head injury rate for ice hockey was 8.1–13.7 per 10,000 participants, and 6.3–9.7 per 10,000 and 9.4–13.5 per 10,000 participants for soccer and football, respectively. The concussion rate was 2.0–3.5 per 10,000 for ice hockey, and 1.4–3.1 and 3.1–5.2 per 10,000 for soccer and football, respectively.
- Under-reporting of concussions in youth hockey; study results of same populations differ depending on the sources reporting.
  - Official injury reports—0.25–0.61 concussions per 1000 player game hours
  - Volunteer observer reports—4.44–7.94 concussions per 1000 player game hours
  - Player surveys (elite level)—6.65–8.32 concussions per 1000 player game hours
  - Player surveys (nonelite level)—9.72–24.30 concussions per 1000 player game hours
- See Chapter 45: Head Injuries, for information about diagnosis and treatment.

### Neck

- **0.4%–9.2% of all hockey injuries**
- If player has loss of consciousness, assume cervical spine injury.
- See Table 80.13 for emergency department data regarding neck injuries.
- **Since hockey helmets have been widely used, incidence of cervical spine trauma has increased; attributed to more aggressive play associated with better and more complete protective gear**

**TABLE 80.12 INJURY COMPARISON INTERNATIONAL WORLD HOCKEY CHAMPIONSHIPS (2007-2013)**

| | Men | | Women | |
|---|---|---|---|---|
| | IR/1000 | IR/1000 | IR/1000 | IR/1000 |
| Site | Player Games | Player Hours | Player Games | Player Hours |
| Face | 3.5 | 12.7 | 0.2 | 0.5 |
| Knee | 2.0 | 7.5 | 1.3 | 4.6 |
| Shoulder/clavicle | 1.6 | 5.8 | 0.5 | 1.6 |
| Head | 1.6 | 5.7 | 1.1 | 3.8 |
| Fingers/hand | 0.8 | 3.1 | 0.3 | 1.0 |
| Ankle/leg | 0.7 | 2.5 | 0.8 | 2.7 |
| Groin/hip/pelvis | 0.6 | 2.3 | 0.3 | 1.2 |
| Teeth | 0.5 | 2.0 | 0.1 | 0.3 |
| Thigh | 0.6 | 2.2 | 0.2 | 0.8 |
| Chest/throat | 0.5 | 1.9 | 0.2 | 0.7 |
| Foot/toes | 0.4 | 1.6 | 0.1 | 0.4 |
| Neck/back | 0.4 | 1.6 | 0.6 | 2.0 |
| Wrist | 0.4 | 1.3 | 0.3 | 0.9 |
| Arm/elbow | 0.3 | 1.2 | 0.4 | 1.3 |
| Abdomen/kidneys/genitalia | 0.1 | 0.6 | 0.2 | 0.6 |
| Eye | 0.1 | 0.3 | 0.0 | 0.0 |
| **Total** | **14.2** | **52.1** | **6.4** | **22.0** |

Data from Tuominen M, Stuart MJ, Aubry M, Kannus P, Tokola K, Parkkari J. Injuries in women's international ice hockey: an 8-year study of the World championship tournaments and Olympic Winter Games. *Br J Sports Med*. 2015;49:1-7 and Tuominen M, Stuart MJ, Aubry M, Kannus P, Parkkari J. Injuries in men's international ice hockey: a 7-year study of the International Ice Hockey Federation Adult World Championship Tournaments and Olympic Winter Games. *Br J Sports Med*. 2015;49(1):30-36.

**TABLE 80.13 EMERGENCY DEPARTMENTS 1993–1999**

| Type of Injury | Ice Hockey | Soccer | Football |
|---|---|---|---|
| **Sports-Related Neck Injuries** | | | |
| Total neck injuries | 5038 | 19,341 | 114,706 |
| Fracture/ dislocation | 105 | 214 | 1,588 |
| Contusions/ sprains/strains | 4964 | 17,927 | 104,483 |
| Lacerations | 199 | 0 | 621 |
| **Rates of Injury** | | | |
| Neck injuries | 1.68–4.26 | 1.34–2.60 | 4.56–7.18 |
| Fractures/ dislocation | 0.08–0.30 | 0.01–0.06 | 0.06–0.09 |
| Strains/sprains/ contusions | 1.68–3.87 | 1.14–2.31 | 4.25–6.38 |

Data from Delaney JS, Al-Kashmiri A. Neck injuries presenting to emergency departments in the United States from 1990 to 1999 for ice hockey, soccer, and American football. *Br J Sports Med.* 2005;39(4):21-25.

- **Increased incidence of severe cervical spine injury since the early 1980s**; before 1973, no spinal cord injuries caused by hockey were reported. First published report in Canadian literature occurred in **1984. Between 1981 and 1985, 15 major cervical spine injuries reported each year attributable to hockey.** From 2006 to 2011, 44 spinal injuries occurred (9.1% considered severe; 78.9%, cervical spine injuries)
- **Mechanism of serious injury:** axial load of cervical spine with head in neutral alignment; in most situations, the player is pushed or checked from behind and slides headfirst into boards.
- **Cervical spine injury data**
  - 96% men, 4% women
  - Of 117 cases, five died; 48% had injuries to vertebrae C4–C5, C5, C5–C6; with spinal cord affected in slightly over half of cases; 29 of 117 became permanently quadriplegic.
  - Causes: impact with boards, 65.0%; impact with player, 10.3%; impact with ice, 7.6%; impact with goalpost, 0.9%
  - 50% of spinal cord injuries occur in the 16–20-year age group.
- **Factors affecting high incidence of spinal cord injuries:** player taller and heavier, skating faster; increased aggressive behavior at all levels (imitating style of professionals); rules not consistently enforced; insufficient emphasis on conditioning; and equipment problems (lack of shock absorption boards) (see Chapter 46: Neck Injuries, for information about diagnosis and treatment).
- Cervical spine injury characteristics (1980–1996, Finland and Sweden): 16 cases with permanent disability; in 50% of cases, mechanism was checking from behind; 69% of vertebral injuries involved fractures and luxations between C5 and C7
- Recent Canadian ice hockey data reflect a trend toward lower rates of catastrophic cervical spine injuries, perhaps reflecting more severe penalties for injurious behavior.

### Eye and Face

- Significant injury reduction since mandatory face mask rule:
  - Unilateral injuries reduced from 478 to 42 in one season; blindness reduced from 37 to 12 in one season.

  - Hockey still accounts for 37% of eye injuries and 56% of blindness in sports.
  - **Face masks have been estimated to save in excess of $10 million/year in injury costs.**
- Injury types (before face mask rule): periorbital soft tissue trauma, 43%; hyphema, 19%; and iris damage, 13%; the stick responsible for more eye injuries than the puck

### Throat

- Goalies particularly at risk of throat injuries (blunt trauma to larynx) from high-speed pucks
- Padded collars and other deflectors worn by both skaters and goalies to reduce injury risk

### Shoulder

- **Acromioclavicular injuries**
  - Mechanism of injury: direct blows to shoulder and falls on outstretched hand
  - Acromioclavicular separation common
  - In Norfray's 1977 study of 77 hockey players, 45% had asymptomatic radiographic abnormalities, including osteolysis of acromioclavicular joint and callus from united and nonunited distal clavicle fractures.
  - In a study by Lorentzon and colleagues, only one of four players with acromioclavicular separations missed >1 week of practice or games.
- **Glenohumeral dislocation:** 8% incidence; greater morbidity than acromioclavicular separations; high rate of recurrence

### Elbow, Wrist, and Hand

- 20% of moderate-to-severe injuries are a result of wrist and hand problems
- **"Skier's" or "goalkeeper's" thumb:**
  - Associated with fall on outstretched hand while hockey stick is still in possession
  - Treatment same as that outlined for other sports
  - It is possible to fashion splint of thermoplastic, plaster, or fiberglass to fit in a hockey glove; may be prohibited by rules or game officials
  - Nonsurgical as opposed to surgical therapy recommendations are changing for early versus late repair of grade III ulnar collateral ligament sprain; check with hand specialist
- Scaphoid fractures: uncommon
- **Metacarpal fracture:** usually related to stick trauma ("slashing")
- **Lacerations** of forearm, wrist, and hand may occur after fall if another player skates over fallen athlete. Skate blades can cut through thinner leather over palm of gloved hand. Wear and tear of leather of glove's palm makes it paper-thin—affords little protection.
- During fights and melees, gloves are often dropped, allowing other traumatic hand injuries plus risk of human bites. Usual human bite precautions are necessary.

### Back

- Infrequent site of injury, 4.3%–7.0%
- Spondylolysis can occur.
- Most back pain is probably of muscular origin and may be treated nonsurgically.
- Severe spinal injuries: T1–T11, 9.2%; T11 to T12/L1–2, 6.0%; L2–S5, 5.1%

### Abdomen or Groin

- Common site of lower extremity injury because of forceful hip adductor contraction during skating stride; 10% of injuries in certain studies
- Inguinal hernias, osteitis pubis, hip impingement, and pelvic or hip stress fractures have been reported in hockey players.
- Rectus abdominis muscle injuries can also be debilitating and chronic in hockey players. When nonsurgical treatment fails, surgical reinforcement has been necessary to permit athletes to return to skating.
- NHL experience (1991–1992 to 1996–1997)
  - Injuries per 1000 player hours: 1991–1992, 12.99; 1996–1997, 19.87
  - Preseason injury rate 5 times greater than the regular season and 20 times greater than the postseason play
  - 23.5% are recurrent injuries; >90% are noncontact injuries
  - Mean time loss: abdominal injury, 10.6 sessions; groin injury, 6.6 sessions
  - Groin injury accounts for 77% of injuries (68% were adductor strains).
  - Abdominal injury: 23%
  - Position: forwards, 60.6%; defensemen, 28.9%; goalies, 5.5% (4.9% not recorded)
- **Femoroacetabular impingement (FAI)** (includes CAM and pincer deformities)
  - Identified with increasing frequency in ice hockey players, with a CAM deformity occurring more commonly
  - A recent review showed ice hockey had the highest occurrence of hip impingement, with football and soccer lagging slightly behind. Goaltenders tend to have a higher incidence of FAI compared with position players.
  - There are no data regarding the occurrence of FAI in women ice hockey players.
  - In general, women have a lower incidence of FAI when other similar sports are compared.
- **Sports hernia**
  - Common cause of groin pain that represents a hernia of the posterior wall of the inguinal canal with or without concomitant hip adductor muscle injury
  - Demand of certain sports such as ice hockey, soccer, and football seems to increase the rate of sports hernia diagnoses.
  - Male > female
  - Failing nonsurgical treatment with relative rest and physical therapy, surgical intervention may be necessary.

### Thigh Contusions

- Hockey players are at a high risk because of high incidence of collisions with players, goals, and boards.
- In general, players are protected, but protective padding may slide to one side.
- Treatment of quadriceps contusions is no different from other collision sports.

### Knee

- **Most common lower extremity injury**
- **Medial collateral ligament (MCL) sprain:** most common serious knee injury in most series; usually results from varus or valgus and rotational stresses
- Anterior cruciate ligament sprains: less frequent than MCL sprains
- Meniscal injuries usually occur in combination with ligamentous injuries.
- Hockey poses a lower risk of injury to the knee than soccer.

### Ankle

- Sprains are less common than in other sports because of protection offered by skate boots.
- Lacerations just above boot often involve tendons.
- Ensure that players tuck tongue of skate boot under shin guards to minimize exposure of anterior ankle to laceration.

### Foot

- Fractures of bones in the foot result from a direct blow, usually from the puck.
- Other foot injuries are uncommon.

## Overuse Injuries

- Limited data, other than incidence, about types of overuse injuries
- **Adductor tendinopathy and patellar tendinopathy are most common overuse injuries, specifically related to skating stride.**

## Special Medical Situations

- **Commotio cordis:** Manifestation of concussive injury to heart resulting in ventricular dysrhythmia and cardiac asystole
  - Despite protective equipment, rare case reports highlight a slight risk of such cardiac injury in hockey. Epidemiologic studies suggest that pediatric and adolescent athletes may be predisposed.
  - 70 deaths (34 organized sports, 36 recreational activity) have been reported; 40 attributed to baseball and seven to ice hockey; survival rate: 10%
- **Indoor air quality problems ("ice-hockey lung")**
  - Propane- or gasoline-propelled ice-resurfacing machines (known as *Zambonis*) have been implicated in causing illness when engines malfunction or ventilation within the arena is inadequate.
  - Players are at an increased risk of minimal exposures compared with spectators.
    - Nitrogen dioxide gas is heavier than air, found at higher concentrations at ice surface.
    - Thermic inversion occurs because of ice temperature.
    - Plexiglass shields along boards alter air circulation at the playing level.
    - During games and practice, players have minute ventilation up to 30 times higher than at rest
- **Nitrogen dioxide–induced respiratory illness**
  - Nitrogen dioxide is a byproduct of combustion
  - Recommended limit: <0.3 parts per million (ppm)
  - Common symptoms (acute onset after unknown indoor exposure after hockey practice or game): cough, hemoptysis, dyspnea, chest pain, headache, and weakness and pulmonary edema (rare)
  - Treatment: withdrawal from toxic environment, bronchodilators, and corticosteroids; untreated, most symptoms resolve within 2 weeks
  - Late complications of bronchiolitis obliterans may develop 2–6 weeks after initial symptoms.
- **Carbon monoxide poisoning**
  - Source: inappropriate combustion of fuel of Zamboni; inadequate ventilation system for arena
  - Recommended limit: <20 ppm
  - Symptoms: acute respiratory (hemoptysis, dyspnea, chest pain, and coughing spells) and central nervous system (headache, dizziness, sleepiness, nausea, and vomiting)
  - Treatment: withdrawal from source of toxin; rarely requires emergency treatment
- **Safeguards**
  - Thirteen states have tested the indoor air quality of ice arenas.
  - Only three states have mandatory air quality testing in ice arenas (Massachusetts, Minnesota, and Rhode Island). Both Pennsylvania and Connecticut have nonbinding guidance for arena indoor air quality.
  - Adequate ventilation of indoor arenas should be ensured.
  - Regular inspection and maintenance of ice-resurfacing machines should be implemented, and indoor air quality should be regularly monitored.

## Injury Prevention

- Continue to update protective equipment.
- Ensure use of mouth guards.
- Fabric designs with a high coefficient of friction are under evaluation to reduce the sliding speeds of fallen players.
- **Enforce rules**. One study suggests that 39% of all injuries were attributed to foul play.
- Effective training and conditioning may minimize the risk of injury. Adequate nutrition, training, and hydration may reduce third-period fatigue and reduce fatigue in situations when several games are played on consecutive days, as occurring in tournaments.

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Available at www.ExpertConsult.com.

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# ICE SKATING (FIGURE SKATING AND SPEED SKATING)

81

Carole S. Vetter • Emily B. Porter

## FIGURE SKATING

### Introduction

- Figure skating is a sport that focuses on a unique combination of athleticism, strength, endurance, gracefulness, and artistry on ice.
- Today, over 178,000 members and 690 clubs are registered with US Figure Skating.

### General Principles

#### History

- Began in the early 1800s; its name was derived from the complicated figures traced on the ice during its early years.
- Jumps and spins were introduced in the 1860s, with continuing development of more complex moves ever since.

#### Figure Skating Categories

- Singles skating: solo skater performing jumps, spins, and connecting steps
- Pairs skating: a male and female skater performing jumps, spins, and dance elements separately and in tandem along with overhead lifts, throws, and throw jumps
- Ice dance: a male and female skater performing intricate footwork and deep edges; partners have specific rules about lifts and the amount of time they can separately skate
- Synchronized skating: team comprising 8–24 skaters moving simultaneously as a group to form various moving patterns; team lifts and certain jumps are permitted

#### Discussion of Sports-Specific Skills and Other Considerations

- A majority of figure skaters are females who began skating when they were aged 5–8 years. Competitive careers peak in teens or the early 20s.

#### Biomechanical Principles

- At the senior level, men routinely perform at least one quadruple jump (4 revolutions), and several women have performed triple axels (3.5 revolutions).
- Jump landing impact forces can reach 5–8 times the skater's body weight.
- Skaters can perform upwards of 50 jumps/session, with 2–4 sessions/day.

#### Equipment and Safety Issues

- Figure skates are traditionally composed of leather boots and steel blades. Certain newer skates use synthetic boot materials to decrease weight.
- Boots
  - For cosmetic reasons, traditionally have an elevated heel; however, this results in increased landing impact forces.
  - Becoming increasingly stiff to accommodate the stress placed on them during triple and quadruple jumps, which may contribute to weaker ankles and poorer proprioception
  - Often custom-fit; may still fit suboptimally; boot shape makes the use of traditional orthotics difficult and may require special orthotics to be molded into the boot when needed.
- Figure skating blades have an inside and outside edge. They have a large front toe pick, which is used in jump landings and certain jump takeoffs.
- Boots and blades can cost $200–$2000 per pair and are usually replaced every 6–12 months due to breakdown.
- Several lower extremity overuse injuries arise from interactions between the skater's body and the skate. Factors contributing to injury include:
  - Boot fit
  - Tying technique
  - Boot condition
  - Blade mounting
  - Skating technique

#### Specific Training Issues

- Typical daily training comprises 2–4 hours of on-ice training and 1–3 hours of off-ice training, which includes strength and flexibility training, dance, conditioning, and choreography.
- Occurs ≥5 days/week throughout the year
- Can become all-encompassing to elite skaters and their families; it is not uncommon for young skaters to move to new cities or opt for home schooling because they make training their first priority.

#### Unique Environmental and Nutritional Issues

- Disordered eating is common in figure skaters. The weight and appearance may inappropriately be the primary focus of skaters and coaches over body composition and performance. Contributing factors include:
  - A major role that a skater's appearance plays in the judges' performance evaluation
  - Advantage of a lighter body weight when jumping
- Delayed menarche may result from restrictive eating patterns. This is often perceived as an advantage by several skaters because the onset of puberty is accompanied by the accumulation of additional body mass that must be lifted into the air with each jump as well as wider hips that contribute to decreased rotational speed.
- Osteoporosis has not been frequently found among skaters.
- Screening for nutritional deficiencies (vitamin D, ferritin, etc.) should be considered in this population.
- Exercise-induced bronchospasm (EIB) and asthma may be triggered because of exposure to the cold, dry air found in ice rinks as well as the chemicals used to maintain the ice surface or Zamboni fumes, which can contain air pollutants such as carbon monoxide and nitrogen dioxide.

### Common Injuries and Medical Problems

- Factors contributing to injuries are the boot, training regimen, nutritional practices, environmental factors, and the culture of the sport that rewards high-risk moves and slender body type.

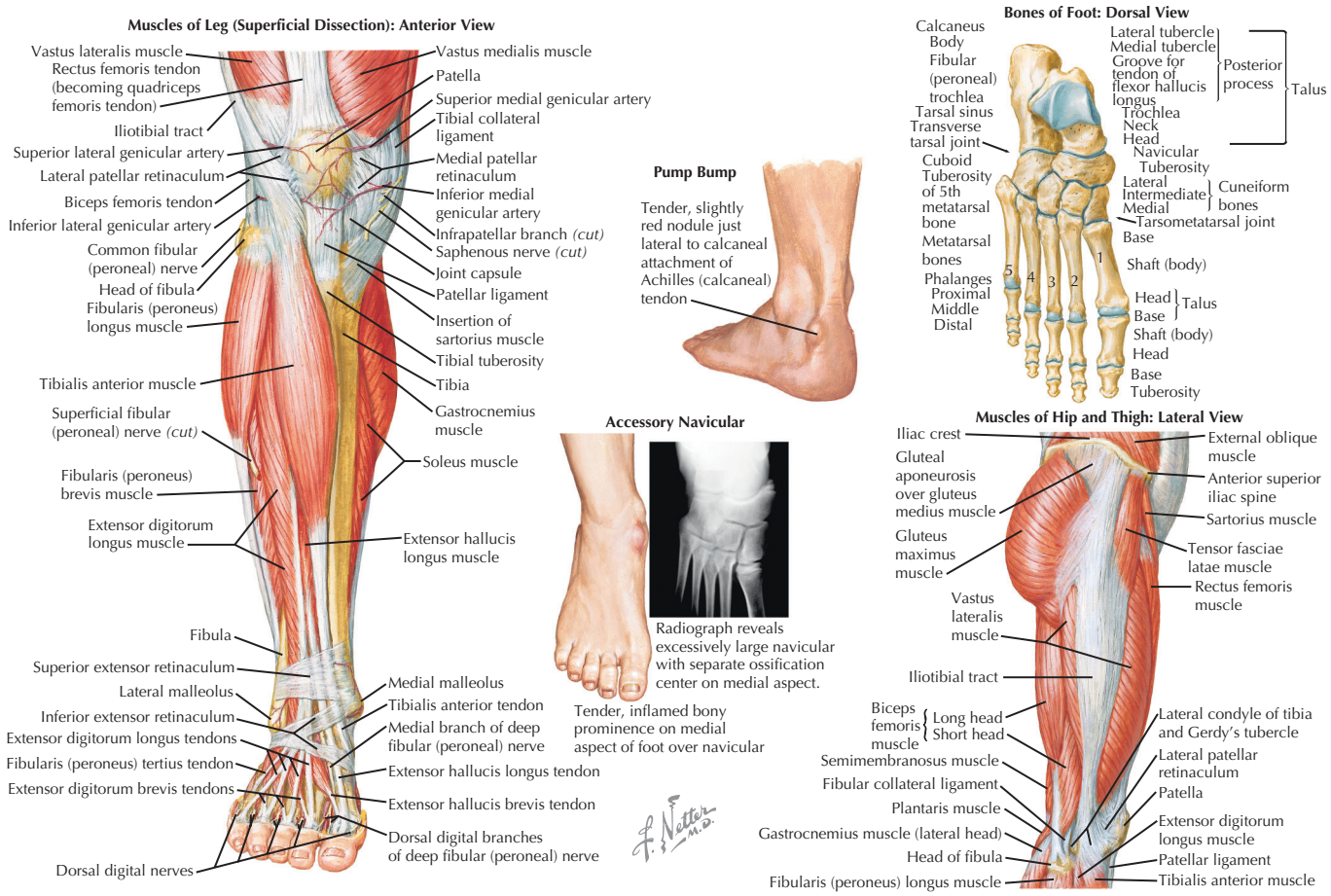


**Figure 81.1.** Figure skating injuries.

## Foot and Ankle Injuries

- "Lace bite" or tendonitis of the tibialis anterior and toe extensors can occur as a result of inappropriate placement of the tongue of the boot (Fig. 81.1). Impact from jump takeoffs and landings can also contribute.
- Ankle sprains are common; likely due to weaker peroneal muscles from wearing stiff skating boots
- "Pump bumps," or Haglund's deformity, and Achilles tendonitis can occur with skate boots that inappropriately fit posteriorly (see Fig. 81.1).
- Malleolar bursitis can occur from friction from the boot at the medial or lateral malleolus.
- Calcaneal apophysitis can be seen in skeletally immature skaters.
- Other common foot issues include inflammation of an accessory tarsal navicular, prominence at the base of the fifth metatarsal, corns, and hammertoes (see Fig. 81.1). Most of these problems are treated with modification of the skate boot by locally "punching out" the leather by stretching or use of moleskin or foam/felt donuts to reduce friction or pressure.

## Stress Fractures

- Lower extremity stress fractures were the most frequent overuse injury recorded in a 2003 study of elite junior women skaters.
- Common in the first and second metatarsals, particularly in the skater's takeoff leg for toe-pick jumps (see Fig. 81.1)
  - The fit of the boot should be checked to ensure that there is adequate room between the skater's toes and the boot.
- Also common in the tibia, fibula, and navicular

## Knee Injuries

- Patellofemoral syndrome is common as in most jumping sports. However, patellar tendonitis is less common in figure skating than in other jumping sports.
- Ligamentous and meniscal injuries are relatively uncommon. This may be due to the relative lack of fixation of the blade on the ice during landings, which decreases torque at the knee joint.

## Hip Injuries

- Muscle strains of the hip flexor, adductor complex, and oblique abdominal muscles are commonly seen in skaters performing triple and quadruple jumps.
- Iliac crest apophysitis can be seen in skeletally immature skaters because of the repetitive stress from jump takeoffs or missed landings (see Fig. 81.1).
- Acetabular labral tears may occur, particularly in elite skaters. Contributing factors include repetitive forces from jump landings and hyperflexibility moves that are unique to figure skating.

## Back Injuries

- Low back pain may be the result of acute trauma or overuse injury from repetitive falls or inappropriate equipment.

## Upper Extremity Injuries

- Wrist sprains and fractures may occur as a result of falling (more common at the recreational level).

**TABLE 81.1 "RETURN-TO-SKATE" PROGRESSION FOR FIGURE SKATERS FOLLOWING CONCUSSION**

| | |
|---|---|
| Phase 1 | Rest from physical and mental activity until asymptomatic from concussion symptoms |
| Phase 2 | Light aerobic exercise with light to moderate elevation of heart rate, off-ice stretching |
| Phase 3 | Heavier aerobic exercise and sports-specific stretches with limited head and body movements off-ice; return to ice with managed sessions, forward skating only |
| Phase 4 | Progressive managed time on the ice, forward and backward skating, changes of directions, choreography without spins and jumps; continue increased off-ice training |
| Phase 5 | Full program run-throughs on the ice without spins or jumps, reintroduction of jumps outside of the program |
| Phase 6 | Integration of jumps and spins into the program, with spins added last; full return to play |

- Rotator cuff injuries can occur in male pair skaters because of repetitive lifting.

### Concussions

- From head trauma that occurs during a fall or collision with another skater during practice or competition warm-ups.
- Figure skaters may have a different recovery/return to p lay course due to impact forces, rotational forces, and multidirectional movements unique to the sport. A return-to-play progression specific to figure skaters has been suggested (see Table 81.1).

## SPEED SKATING

### Introduction

- Speed skating is the sport of competitive racing on specially designed skates, typically around an oval track.

### Epidemiology

- During the 2010 Winter Olympics, 27.8% of male short track registered athletes sustained an injury, while <5% of all long track athletes sustained an injury.
- In-competition injuries reported in short track speed skating in a season-long study: shoulder dislocation/separation (9%), groin strain (6.1%), concussions (6.1%), and knee contusions (6.1%). The most common on-ice training injuries were groin strains (22.2%), knee contusions (14.8%), and ankle sprains (12.1%).
- Short track skaters at a higher risk during competition because of the collision nature of the sport.

### General Principles

#### History

- Early 13th century: Skating began as a mode of transportation across frozen lakes and rivers in Scandinavia using skates made of bone or wood.
- 1400: With growing popularity of skating in the Netherlands and Europe, metal skates appeared.
- 1572: All-iron skate was developed in Scotland.
- 1676: First known skating competition held in the Netherlands
- Speed skating has been an Olympic event for men since the first winter Olympics in 1924 and for women since 1960.
- Today, speed skating consists of two disciplines: *long* and *short track speed skating*; short track speed skating became an Olympic event in 1992.

#### Review of Different Types of Speed Skating

LONG TRACK SPEED SKATING

- A race against the clock
- Pairs compete in separate lanes on a 400-meter oval track.
- Each racer has only one opportunity to produce a winning time.
- All events are run in a counterclockwise direction.
- In order for the racers to skate the same distance,
  - Must change lanes during each lap
  - Begin from a staggered start
  - Crossover occurs on the backstretch of the oval with the racer in the outer lane having the right of way
- Contact is prohibited.
- A new team long track event, the Team Pursuit, was added to official competition during World Cups, World Championships, and the Olympic Games in 2003.
  - A team consists of three skaters and two different teams skate against each other at the same time, starting on opposite sides of the track.
  - The teams race without changing lanes. The last skater over the finish line gives the team their official finish time.

SHORT TRACK SPEED SKATING

- A race against other competitors and is an elimination event.
- Four to six racers compete per heat with the first- and second-place finishers advancing to the next heat.
- Skaters try to outskate and outwit the competition.
- Athletes skate on a 111-meter track within an internationally sized 30-by-60-meter Olympic-sized ice hockey rink.
- Lead skater has the right of way. Passing skaters must pass cleanly without body contact.
- Skaters often skim their fingers along the surface of the ice inside the track but must skate around the outside of the blocks.
- Short track also has a relay event. Four skaters skate in a relay team competing against three other teams at the same time. Skaters on the same team exchange by the lead skater being pushed forward by the exiting skater. Only one skater on each team can use the racing track, the others have to rest in the middle (inside of the blocks).

#### Discussion of Sports-Specific Skills and Other Considerations

- The average age of competitive long track speed skaters is 20–24 years, whereas that of short track speed skaters is 19–22 years.
- An average of 8 hours of training per day, including on-ice and dry-land training
- Competition season runs from September to April and during this time, the athletes travel an average of 2 weeks out of every month.
- Dry land training from May to July with August off
  - Consists of technique training, aerobic conditioning, balance, agility, and weight training
- Anthropometry studies have shown that elite speed skaters have normal body height and mass; however, shorter legs and a longer trunk with better-developed thigh musculature compared to other elite athletes.

#### Biomechanical Principles

- Elite skaters generate the highest mechanical power output and the least loss of power to frictional forces.

- Speed skaters skate in a "crouched position" for aerodynamic and biomechanical advantage.
- Optimal trunk position is horizontal (trunk angle, 15 degrees).
- Skaters generate power during push off (skate in contact with ice) by nearly completely extending their knee (knee angle, 171 degrees) in a short period of time.
- Smaller knee angle at the onset of push off results in better performance. Knee angle of elite skaters during the glide phase is typically 90–110 degrees.
- Skintight racing suits with hoods and arm position behind the back also contribute to improved aerodynamics.

## Equipment and Safety Issues

### LONG TRACK SPEED SKATING

- Skaters wear skin-tight racing suits (skin suits) with hoods to decrease air resistance (Fig. 81.2). The racing suit must conform to the natural contour of the racer's body and cannot be otherwise altered. There are certain restrictions for the materials used.
- Racers often wear glasses to protect their eyes from wind and ice chips and to reduce glare and improve visibility.
- The early 1990s brought a change in the skates for long track racing from a "fixed blade" skate to a "klap blade."
  - Unlike fixed blade skates, only the toe of the blade is affixed (with a hinge) to the boot (see Fig. 81.2).
  - At the end of each stride, as the skater picks up the skate, the blade briefly disconnects from the heel of the boot.
  - When the ankle has fully extended and the heel is already off the ice, the full length of the blade is still in contact with the ice pushing.
  - At the moment the blade leaves the ice, a spring mechanism mounted on the front of the boot snaps the blade back up to the boot, resulting in the clapping sound that gives the skate its name.
  - This mechanism keeps the blade on the ice longer, increasing the skater's pushing power and therefore speed.
- Pads to protect the athletes in case of falls surround the outer diameter of the track.

### SHORT TRACK SPEED SKATING

- Skaters wear similar skin suits for as long track skaters do but without a hood.
- Short track skin suits are required to have kneepads, shin guards, and neck guard to protect the skaters from the blades of other skaters. At international competitions, the skaters must wear cut-resistant underwear.
- Hard-plastic helmet to prevent potential head injuries from collisions with competitors, the ice, or padded rink side walls (see Fig. 81.2)
- Ceramic fingertip gloves to protect their hands from other skater's blades and the ice as they are going around a curve and skim their inside hand on the ice to help maintain balance
- Fixed blade skate with boots that lace up higher on the ankle than traditional skates
- Skates are constructed from customized foot molds to help stabilize the foot and ankle so that skaters can more tightly around corners.
- Blades are extremely sharp and are bent in at an arc that mirrors the direction of the turn to better grip the ice and placed off-center to the left to keep the boot from rubbing the ice when leaning into left turns at high speed.

## Specific Training and Physiologic Issues

- Skaters "crouched position" is a physiologic disadvantage to oxygenation of working muscles because of reduced blood flow (small knee and trunk angles).
- Deoxygenation of working muscles leads to faster fatigue compared to similar elite sports.

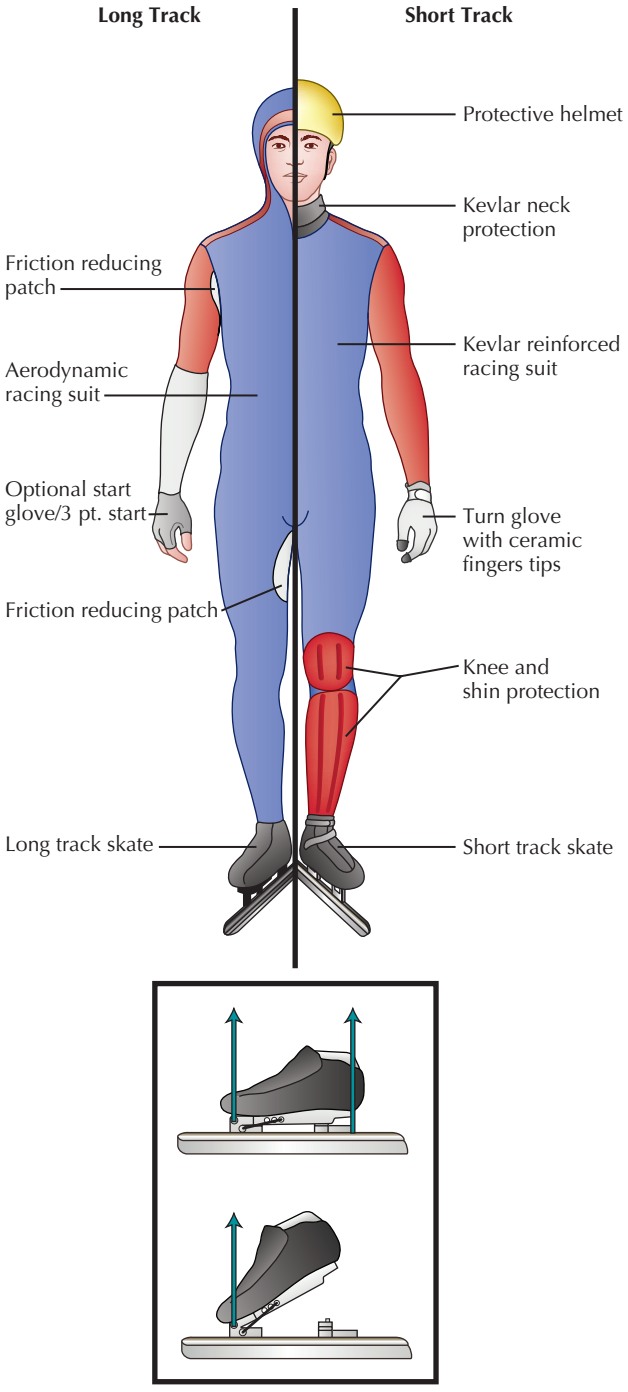


**Figure 81.2.** Speed skating equipment.

- Short track skaters experience asymmetry of quadriceps muscle oxygenation with the right leg having decreased blood flow. Skaters solely travel on their right leg during tight turns.
- Higher anaerobic demands for shorter race distances
- Aerobic demands increase with increased race distances.
- Reduced blood flow to working muscles results in higher blood lactate concentrations compared with athletes in other sports.

## Unique Environmental Issues

- Speed skaters are routinely exposed to cold dry ice rinks during training and competition and to the chemicals used to maintain the ice surface and Zamboni fumes, including carbon monoxide and nitrogen dioxide.

Lateral malleolus laceration from skate blade

Short track skater collision

Skaters bracing for fall

Skaters bracing for fall

**Figure 81.3.** Speed skating injuries.

## Common Injuries and Medical Problems

### Long Track Speed Skating

- Numerous overuse injuries are secondary to the position their bodies are in while skating and the constant repetitive motion of skating in the same direction.
- Mechanical low back pain and lumbar disc problems are common.
- Chronic hip flexor and iliotibial band (ITB) tightness as well as iliopsoas tightness cause painful hip symptoms.
- ITB tightness and chronic knee flexion leads to the common problem of patellofemoral pain syndromes, patellar tendonitis, and ITB syndrome.
- The skates, which are laced tight enough to cause numbness and often worn without socks, may also lead to certain common problems including callosities, corns, bunions, hammertoes, neuritis, and recurrent tinea pedis and can cause irritation to subcutaneous tendons, resulting in Achilles tendonitis/tenosynovitis and "lace bite," also known as *tibialis anterior*, *extensor hallucis longus*, and *extensor digitorum longus tendonitis*.
- Somewhat similar to sprinters in track, the start of a speed skating race involves an explosive sprint from a stationary position; however, instead of quadriceps and hamstring strains, speed skaters often sustain hip adductor and hip flexor strains.
- The most common injuries due to falls are lacerations from skate blades, contusions, fractures, and joint sprains, particularly of the upper extremity from the skaters bracing for the fall (Fig. 81.3).

### Short Track Speed Skating

- Similar to overuse injuries of long track athletes, includes mechanical low back pain, lumbar disc problems, hip pain syndromes, and patellofemoral pain
- Unique to the short track skaters is peroneal tendonitis caused by repetitive forceful dorsiflexion and eversion needed for the skate crossover on the short tight curves.
- Traumatic injuries that are most concerning for short track speed skaters include shoulder dislocations, ankle and lower extremity fractures, concussions, cervical-spine injuries, thoracic and rib contusions, and lacerations (most commonly to the lower extremity).

## INTERNATIONAL SKATING UNION (ISU)

For information on ISU Rule 139-Doping, Rule 140-Medical, and Rule 141-Safety, visit http://www.isu.org.

## ACKNOWLEDGMENT

Dr. Porter would like to thank Peter Zapalo, Director of Sports Science and Medicine for US Figure Skating, for his review and suggestions to the Figure Skating section of this chapter.

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Available at www.ExpertConsult.com.

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# 82 SWIMMING AND DIVING

Jennifer D. Stromberg • Nathaniel S. Jones

## SWIMMING

### Introduction

- Swimming is the most popular aquatic sports discipline and is an important part of triathlon and water polo.

### Epidemiology

- At the international level, swimming and the other aquatic sports disciplines are governed by the Fédération Internationale de Natation (FINA).
- USA Swimming registered 367,902 athletes and 3,096 clubs in 2014. Females made up 56.6% of year-round athletes, the majority of whose ages ranged from 10 to 14 years.
- US Masters Swimming consists of nearly 60,000 swimmers aged 18 years and older and provides support to more than 1,500 clubs and workout groups.
- US Synchronized Swimming oversees competitive events for synchronized swimmers who range in age from 12 to 20+ years.

### General Principles

#### History

- Swimming has been a part of the Summer Olympic Games since its inception in 1896.
- Synchronized swimming was first introduced to the Olympics in 1948, but solo and duet competitions did not become official events until 1984. Team synchronized swimming was added in 1996.
- Open-water swimming became a part of the Summer Olympic Games in 2008.

#### Types of Sports or Races

- Freestyle events do not require a particular stroke, but front crawl is the most popular and commonly referred to as freestyle (Video 82-1). Alternating arm strokes and an alternating or "flutter" kick propel the body forward in the prone position.
- Backstroke events are performed in the supine position with alternating arm strokes and "flutter" kick (Video 82-2).
- Breaststroke events are performed by advancing the arms forward along the midline; then pulling to the sides without leaving the water while executing a "whip" kick by flexing the knees and hips and abducting the legs; and then "whipping" them back together (Video 82-3).
- Butterfly events require the arms to be simultaneously advanced forward above the water and then pulled through underwater, while the legs kick up and down as a single unit similar to a tail in a "dolphin" kick (Video 82-4).
- Relay competitions typically consist of four swimmers. In a medley relay, each swimmer performs one of the four strokes.
- Synchronized swimming events can consist of solos, duets, teams of eight, and combinations of up to 10 swimmers.

#### Discussion of Sports-Specific Skills and Other Considerations

- Successful swimmers are often lean and tall with long limbs, wide shoulders, and large palms with a large muscle mass in the middle and upper body.
- Elite swimmers tend to be more flexible than their non-elite counterparts.
- Synchronized swimming requires exceptional breath control. Routines typically vary from 2 to 5 minutes in duration with 45%–50% of the time spent underwater.

#### Biomechanical Principles

- Speed is gained by decreasing drag/resistance while moving through the water.
  - Form drag is body position-dependent water resistance. This type of drag is reduced by using a "streamlined" horizontal position with good body roll and remaining underwater when possible.
  - Wave drag is caused by turbulence at the water surface. This type of drag is reduced by using deep pools with multiple lane lines, entering the water smoothly, and swimming in the wake of another swimmer ("drafting").
  - Frictional drag is caused by the contact of the body surface with the water and is reduced by shaving and using specially designed swimwear.
- Technique and efficiency play a greater role than power in performance.

#### Equipment

- Goggles protect the eyes from chlorine and aid in vision underwater.
- Caps decrease resistance and keep hair out of the face.
- Kickboards can be held in the arms while practicing kick techniques.
- Fins are used to increase kick propulsion in practice.
- Hand paddles add resistance to the pull during practice.
- Pull buoys are placed between the legs while practicing pull techniques.

#### Safety Issues

- Neck injuries are uncommon, but they can be catastrophic. Preventive measures include a minimum pool depth of 5 feet for dives and flags placed 5 meters from the pool wall to help avoid collisions during backstroke. Backboards should be available and accessible on the pool deck.
- Sudden death in triathlon is most likely to occur during the swim. There is no clear association with skill level or medical problems. No athlete should swim without adequate supervision.
- Synchronized swimmers practicing boosts and throws are at increased risk of traumatic injuries including concussions.

#### Specific Training and Physiology Issues

- Although races typically range from 50 to 1,500 meters, practices can range from 3,000 to over 10,000 meters, which can predispose swimmers to overuse injuries.
- Several important physiologic changes occur with submersion in water.
  - Gravitational blood pooling in the venous system is decreased, shifting fluids to the central circulation and pulmonary vascular system.
    - Athletes with poor ventricular relaxation or increased pulmonary venous pressure may be at risk of swimming-induced pulmonary edema (SIPE).
    - Overhydration prior to competition should be avoided.

- The parasympathetic nervous system is activated via the diving reflex.
  - Heart rate is reduced, and vasoconstriction of muscle vasculature preserves blood flow to the brain and heart.
  - These adaptive changes allow for increased time underwater without additional breaths.
- These changes can increase risk of arrhythmia and sudden death, particularly in athletes with long QT syndrome and/or paroxysmal atrial flutter or fibrillation.

- Prolonged breath-holding in synchronized swimmers can cause hypoxia and is often manifested by dizziness, disorientation, or even syncope.

### Unique Environmental and Nutritional Issues

- Exercise-induced bronchospasm is prevalent in swimmers. Asthmatic children may be drawn to the sport by the warm, humidified air, but exposure to airborne chlorine may lead to airway inflammation and bronchoconstriction.
- Water immersion can expose athletes to a variety of dermatologic issues.
  - Infectious conditions include:
    - Swimmer's itch (schistosome dermatitis): pruritic 3-5 mm erythematous papules in areas not covered by swimwear: lesions typically resolve spontaneously within 3–7 days
    - Swimming pool granuloma: verrucous nodules or plaques appear over bony prominences 6 weeks after inoculation with atypical mycobacteria; lesions may ulcerate and should be biopsied and treated with warm water soaks and antibiotics.
    - Diving suit dermatitis: diffuse erythematous papules in areas covered by swimsuits caused by *Pseudomonas*
    - Seabather's eruption: stinging sensation followed by pruritic vesiculopapular or urticarial rash on areas covered by a swimsuit within 24 hours of saltwater exposure; caused by larvae of thimble jellyfish trapped under swimwear; best treated with cold packs, antihistamines, and topical steroids
    - Bikini bottom: firm, deep nodules along the inferior gluteal crease caused by *Streptococcus* or *Staphylococcus aureus* due to prolonged use of tight-fitting swimwear; best treated with systemic antibiotics and frequent warm soaks
    - Swimmer's ear
    - Folliculitis
    - Molluscum contagiosum
  - Athlete's foot, warts, and pitted keratolysis may also result from skin contact with pool decks.
  - Contact dermatitis can occur from equipment or pool water. More severe forms include aquagenic urticaria, cold urticaria, and contact urticaria related to chlorine exposure.
  - Copper-chelating shampoos and hydrogen peroxide are used for green hair caused by copper ions in pool water.
  - Prolonged water exposure commonly leads to dry, itchy skin which is treated with application of petrolatum and avoidance of long, hot showers.
- Open-water swimmers are at increased risk of environmental injuries such as otitis, sunburn, jellyfish stings, dehydration, hypothermia, and hyperthermia.
- The necessity for revealing swimwear increases concern about body image and risk of disordered eating and relative energy deficiency, particularly in synchronized swimmers.

## Common Injuries and Medical Problems

### Swimmer's Shoulder

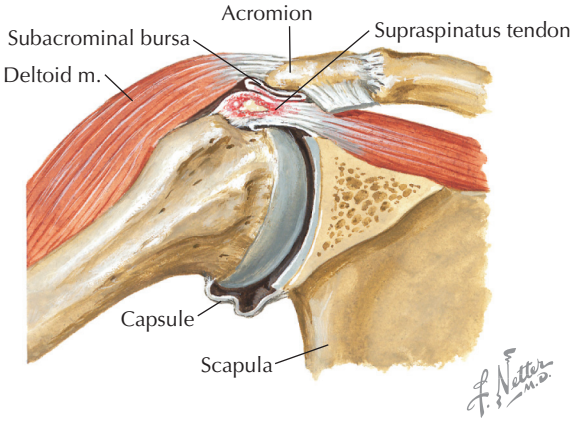
**Description:** The most common musculoskeletal complaint in competitive swimming; typically results from some combination of intra-articular or subacromial impingement (Fig. 82.1A), scapular dyskinesis, and overuse in the setting of joint laxity and rotator cuff muscle imbalance.

**History:** Pain is typically of gradual onset and often worse during the catch (Fig. 82.1B) and early to mid-pull (Fig. 82.1C) portion of the stroke. Poor body roll and unilateral breathing increase the propensity for impingement during freestyle.

**Treatment:** Includes avoiding the use of hand paddles

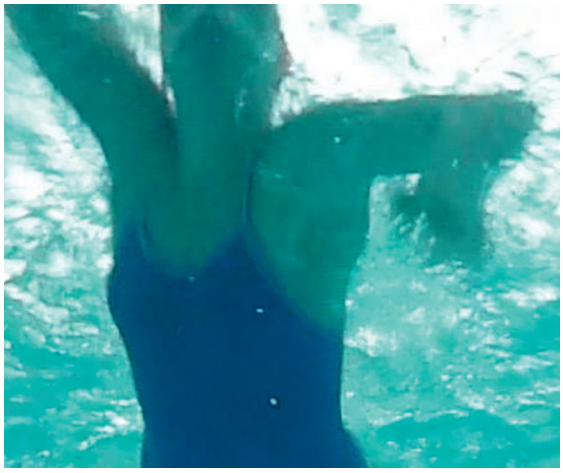
**Return to Play:** Emphasize lower extremity training, avoid strokes which exacerbate pain, and focus on proper technique with good body roll (45 degrees along the long axis), bilateral breathing, and less internal rotation on hand entry for freestyle (fingers first, not thumb).

**Prevention:** Dry land training with stretching and strengthening program for rotator cuff, scapular stabilizers, and core; training distance should be increased by no more than 10% per week




**A.** Abduction of arm causes repeated impingement of greater tubercle of humerus on acromion, leading to degeneration and inflammation of supraspinatus tendon, secondary inflammation of bursa, and pain on abduction of arm. Calcific deposits in the supraspinatus tendon may progress to acute calcific tendonitis and sudden onset of severe pain.

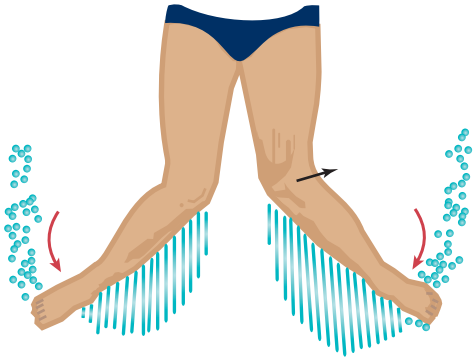
**B.** The catch occurs when the hand enters the water.



**C.** The hand reaches midpull when directly below the shoulder.

**Figure 82.1.** Swimmer's shoulder.

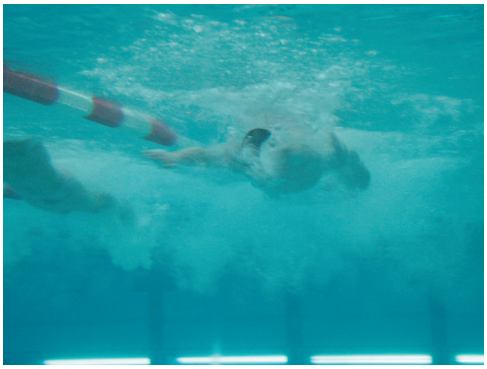
Correct alignment for breast stroke kick with hips held in closer adduction.



Lateral tracking of patella and/or medial knee stress with abnormal kick motion.

**Figure 82.2.** Knee pain associated with kick.

Extreme lumbar flexion seen in this swimmer on the block predisposes the athlete to lumbar injury when takeoff forces applied.



Center swimmer seen underwater with back flexed for butterfly stroke; swimmer on the left shows back extended with head and arms out of water for this stroke. Repetitive flexion and extension predisposed the athlete to lumbar back problems.

**Figure 82.3.** Low back pain.

### Breaststroker's Knee

**Description:** Medial and/or anterior knee pain; the second most common musculoskeletal complaint in competitive swimming
**History:** Results from repetitive valgus loading with breaststroke kick (Fig. 82.2) or the eggbeater kick used in synchronized swimming and water polo (Video 82-5)
**Physical examination:** MCL sprain, medial patellar facet tenderness, and/or inflamed medial synovial plica may be noted.
**Diagnostic considerations:** More common in older, more competitive swimmers with more years of experience
**Treatment:** Cessation of breaststroke kick; lower extremity and core strengthening
**Return to play:** Gradual reintroduction of breaststroke kick with proper technique—hip abduction between 37 and 42 degrees with good external rotation and dorsiflexion of the ankles
**Prevention:** Gradual increase in breaststroke distance; stroke diversity and stroke-specific warm-up; maintain quadriceps and hamstring flexibility

### Spine Pain

**Description:** Occurs in approximately 20%–25% of swimmers in a single season; facet pain results from lumbar hyperextension required for streamlined position; pain may also result from repetitive flexion of the thoracic spine in butterfly stroke and with diving from the starting block (Fig. 82.3).
**History:** Pain is often accentuated by the use of dolphin kick, as well as fins, kickboards, and pull buoys, and increases with training intensity, duration, and distance. Flexion-based pain and facet joint injury can also result from spine rotation and flexion during flip turns (Video 82-6).
**Physical examination:** Pain can be flexion-based or extension-based as detailed above.
**Differential:** Spondylolysis/spondylolisthesis and discogenic pain
**Return to play:** Gradual reintroduction of butterfly, dolphin kick, and flip turns
**Prevention:** Gradual increases in butterfly and dolphin kick; stroke diversity; core strengthening and stabilization

### Swimmer's Ear

See Chapter 48: Maxillofacial Injuries.
**Prevention:** Dry canals with hair dryer, and maintain acidic environment with ear drops; use molded ear plugs in cold water swimming.

### Thoracic Outlet Syndrome

**Description:** Impingement of the brachial plexus and/or vascular supply to the upper extremity occurring in the interscalene

triangle, the costoclavicular space, or the coracopectoral tunnel (Fig. 82.4)

**History:** Sensation of aching or coolness radiating down the arm associated with exertion and overhead activities

**Physical examination:** Findings include weakness or fatigue in the involved nerve root distribution, decrease in radial pulse in the affected arm noted while pulling down on the arm and having the patient inhale deeply and turn the head toward the affected side (Adson's maneuver), and reproduction of symptoms by placing the arm in hyperabduction and external rotation (Wright's test) or by manually compressing the clavicle.

**Diagnostic considerations:** Imaging may include apical lordotic view of the chest to look for a cervical rib, plain films of cervical spine and shoulder, and/or arteriogram. Nerve conduction studies may be useful in brachial plexus involvement. Axillary vein thrombosis should be considered in patients with arm swelling and venous distension.

**Treatment:** Strengthen scapular elevators, stretch cervical girdle, address poor posture, and avoid provocative positions; surgical treatments include rib resection and scalene release.

**Return to play:** Stroke modification to minimize impingement

## DIVING

### General Principles

Diving is an aquatic sport distinct from swimming and has its own set of sports-specific injuries.

#### Statistics

- Approximately 12,000 athletes register with USA Diving each year.
- Typical age of Olympic-caliber divers: 12 years to mid-20s

#### History

- Springboard diving became an Olympic sport in 1904, followed by platform diving at the 1908 London Olympics.

#### Terminology

- Save: divers, in order to avoid a non-vertical entry, must save a dive, a position in which the body is thrust into hyperflexed shoulders and a hyperextended spine
- Swimout: occurs during entry into the water, where arms are forcibly pulled forward to the side in an attempt pull in the rest of the body with minimal splash

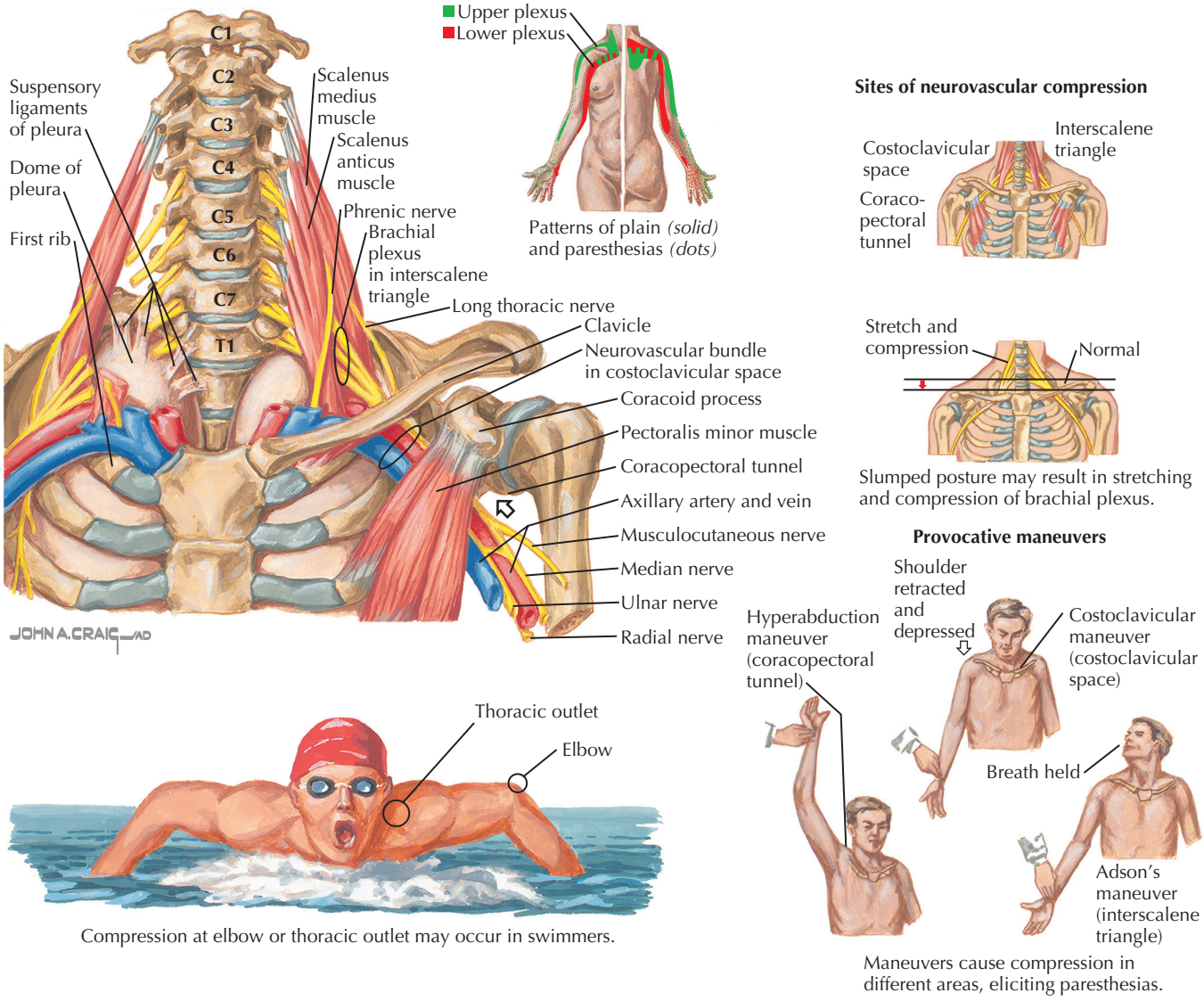



**Figure 82.4.** Thoracic outlet syndrome anatomy.

### Biomechanical Principles

- Energy is transmitted along the kinetic chain from the hands to the wrists to the elbows and the shoulders.
- The shoulder absorbs most of the water-entry axial loading.
  - Stabilizes the joint by elevating the shoulder girdle with increased scapular abduction, so the glenoid fossa is behind the humeral head and is better able to absorb axial load impacts.
  - Inadequate scapular abduction increases the demands on the soft tissue, causing injury, ligament laxity, and, in turn, shoulder instability.
- Upon water impact, velocity is decreased by more than 50% within 1 second.

### Competition Events

- Springboard: 1 meter and 3 meter. Platform: 10 meter

### Equipment

- Pool with adequate depth; having a dark-colored bottom pool or a water surface agitator (bubbles) can help the diver visualize the surface. The water surface agitator can also reduce the axial load energy from water entry.
- Dry-land equipment: trampolines with spotting rigs, landing pits, spotting harnesses, and poolside diving boards

### Injury Patterns During Different Diving Components

TAKEOFF

- **Phases:** approach, hurdle (jump onto end of board), and press (depression of board and upward acceleration of body). Timing is essential for maximal acceleration of body and efficient press.
- **Injuries during takeoff:** patellar tendonitis, quadriceps tendonitis, patellofemoral compression syndrome, Achilles and posterior tibialis tendonitis, and lumbar injuries due to compensatory hyperextension

FLIGHT OR MIDAIR MANEUVER

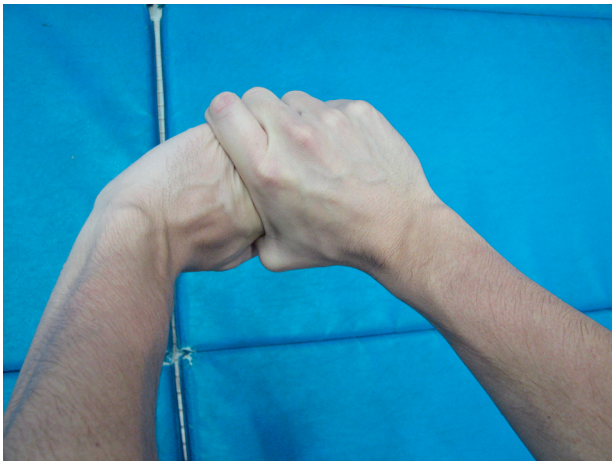
- Begins when a diver leaves the board or platform and ends with initial contact with water
- **Injuries during takeoff:**
  - Spine and long head of biceps injuries due to torsional overload during twisting
  - Can strike the board mid-air, particularly the head, causing concussions and/or lacerations
    - Only two reported fatal head injuries, and both occurred from the 10 meter platform during a reverse 3½ somersault tuck dive attempt.

ENTRY

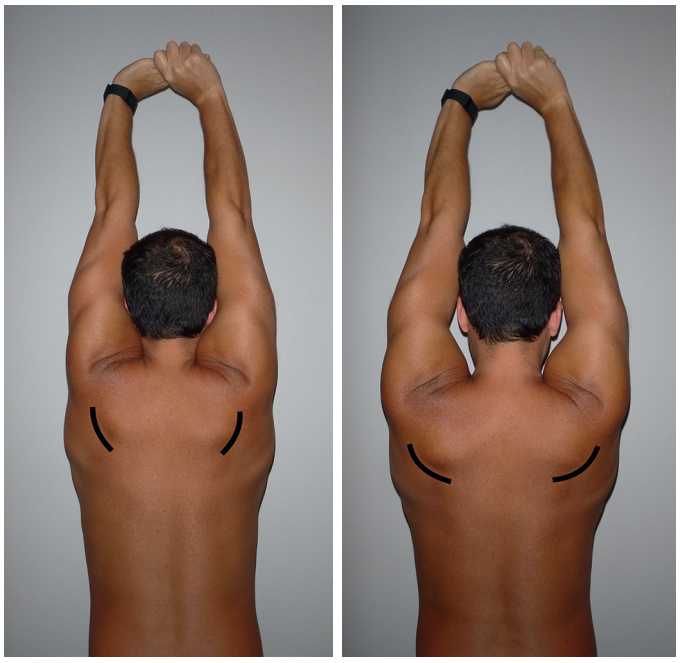
- Most injuries occur during water entry due to forces upon entry and during underwater maneuvers as diver attempts to have a splashless entry (Fig. 82.5).
- In order to protect the head and spine and dissipate energy, entry should be made with the arms extended and hands overlapping, maintaining a flexed wrist and extended elbows.
- **Injuries during entry:**
  - Can occur at any portion along the kinetic chain due to large forces, repetition, and overuse
  - Most injuries attributed to repetitive microtrauma: can include multidirectional shoulder instability, shoulder dislocations, shoulder impingement, elbow UCL strains/tears, ulnar neuritis from UCL laxity, scaphoid stress fracture, carpal instability, dorsal impaction syndrome, concussions, and lumbar spine injuries from attempting to save a dive

### Injury Incidence in Diving

- Higher in pool training and platform than in dry land and springboard, respectively, and is more frequent in practice than in competition.



Flat-hand water entry postion on a dive (demonstrated on dry land)



Flat-hand technique prior to entry  At moment of water surface

**Figure 82.5.** Proper hand technique for entry.

- Most injuries due to overuse; divers can perform more than 100 dives in practice.

## Common Injuries or Medical Problems

### Lower Extremity Injuries

- Osteochondritis dissecans lesions can occur in the knees; some suggestions to keep kids undergoing growth spurt away from a 10-meter platform
- Patellar tendonitis, quadriceps tendonitis, patellofemoral pain syndrome, Achilles and posterior tibialis tendonitis (most are secondary to overuse or incorrect technique with press on springboard)
- Ankle sprains and fifth metatarsal fractures with awkward landing on board
- Treatment: many of the standard treatments for knee and ankle injuries, such as braces or orthotics) are not viable options in diving. Therefore, taping of the knee and ankles can be

attempted in addition to training modifications to avoid repetitive jumping stresses.

### Shoulder Injuries

- Flexibility is important in diving but can also be a cause of problems, leading to glenohumeral joint instability and impingement.
- Multidirectional instability with associated microtrauma of repetitive diving can lead to shoulder instability.
- Mechanically at risk position for shoulder: 180 degrees abduction, 180 degrees flexion, and maximum internal rotation, with no inferior support.
- When attempting to save a dive, there is usually hyperextension of the back and hyperflexion of the shoulder, increasing risk of anterior glenohumeral subluxation.

### Multidirectional Instability

**History:** Many times, divers have noted increasing soreness after diving early on; then, as instability increases, pain is noted with diving. Pain at rest points to advanced instability.

### Elbow Injuries

- The elbow must be in extension upon water entry, taking on a large amount of stress.
- Instability is usually medial; can lead to ulnar neuropathy

### Triceps Strain

**Description:** Seen in response to repetitive water entry forces; appropriate strength and range of motion are vital for the elbow to remain in extension upon contact with water. Often, injury occurs at the distal musculotendinous junction.
**Return to play:** When pain has improved and ability to lock in extension

### Medial Collateral Ligament Injury

**History:** Pain at the medial elbow, particularly upon entry to water
**Return to play:** Pending ability to get elbow in full extension with appropriate strength to maintain extension during entry

### Wrist Injuries

- Can be secondary to overuse vs. trauma
- Can occur from hitting the board: fractures or ulnar collateral ligament sprains of the thumb
- Overuse: carpal instability, ganglion cyst, and flexor carpi ulnaris tendonitis

### Dorsal Impaction Syndrome

**History:** Pain at radiocarpal joint secondary to forced wrist dorsiflexion
**Prevention:** Taping of wrists can be helpful for prevention

### Cervical Spine Injuries

- Most commonly seen in recreational diving where there is lack of experience, shallow water, inadequate supervision, and alcohol ingestion

### Cervical Hyperflexion Injury

**Description:** Usually occurs secondary to poor technique
**History:** Pain, paresthesias, and radicular symptoms
**Physical examination:** Pain, restriction of cervical range of motion, specifically in extension; normal neck examination with neurological findings in the upper extremity, consider a possible brachial plexus injury
**Treatment:** Rest, rehabilitation, and focus on proper technique with the neck in the neutral position, sitting protected in between the arms
**Return to play:** Pain-free, normal range of motion with normal strength

### Lumbar Spine Injuries

- Flexion and extension with axial loading occur in most diving maneuvers along with shearing and torsional stresses at multiple spinal segments.
- Anterior segments (vertebral body, vertebral endplate, and intervertebral disc) vulnerable to increases in loads, particularly during takeoff and entry
- Posterior segment (facet joins, pars interarticularis) is the more common cause of back pain due to repetitive extension.
- A recent study showed low back pain to be the most frequently reported symptom in divers, with a rate up to 38.4% in 13-17-year-olds. Lack of flexibility at the shoulder joint translates into compensatory hyperextension of the lower back.
- Growing spine is highly vulnerable to trauma, particularly during the adolescent growth spurt. Most elite divers are in their teenage years.
- The reduction of speed upon water entry creates a tremendous amount of axial load.
- One study found 89% lifetime prevalence of back pain.
- Back pain is a common reason to retire from the sport.

### Spondylolysis/Spondylolisthesis

**Description:** Considered to be more of a posterior element injury affecting the facet joints and pars interarticularis. Extension of the spine can occur throughout the diving process, but it most commonly takes place in takeoff for a back dive, a front spinning dive when the diver over-rotates, or most commonly when trying to save a dive.
**History:** Diver presents with low back pain that worsens with activity, particularly extension. Many times, a decline in performance and technique due to pain is noted.

### Non-Orthopedic Injuries

#### TYMPANIC MEMBRANE PERFORATIONS

**Description:** From landing directly on the ear
**Treatment:** Usually closes within 4 weeks; if not, consider surgery.
**Return to play:** Use of ear plugs is recommended if continuing to dive.

#### VESTIBULAR ABNORMALITIES

**Description:** Thought to be related to changes in linear acceleration and rotation as well as water impact with rapid deceleration. In a forward somersault, there are 1260 degrees of rotation. Concussions can also occur. Therefore, anytime abnormal vestibular symptoms are reported, one should consider a concussion as the source. Visual orientation and cerebellar training occurs with time and experience.
**Treatment:** Rest and vestibular rehabilitation; spotting techniques are helpful and can be taught with land-based techniques and spotting gear.

#### SCALP LACERATION

**Description:** More common with reverse and inward dives

#### PULMONARY CONTUSION

**Description:** Rupture of pulmonary blood vessels and resultant hemoptysis secondary to landing flat, usually from a 10 meter platform dive.
**Treatment/return to play:** Resolution with rest, most return to play within 48 hours

#### ANXIETY AND PSYCHOLOGICAL STRESS

**Description:** Due to individualized performance pressure and complexity of dives; must be addressed prior to learning difficult dives

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:
eAppendix 82-1, Water Polo
Video 82-1, Front Crawl (Freestyle)
Video 82-2, Back Stroke
Video 82-3, Breaststroke
Video 82-4, Butterfly Stroke
Video 82-5, Eggbeater Kick
Video 82-6, Flip Turn

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

# eBOOK SUPPLEMENTS

## eAppendix 82-1, Water Polo

### Introduction

- Water polo is a team contact sport typically played in a 25-meter pool. The object of the game is to score goals by putting the ball into the opponent's net, similar to soccer.

### Epidemiology

- Water polo is governed by the FINA at the international level.
- In the United States (US), water polo is governed by USA Water Polo.
  - In 2013, USA Water Polo had 39,715 individual members (up from 26,873 in 2008).
  - Around 500 clubs are currently registered as of 2015.
  - With the inclusion of splashball, a youth water polo program for athletes aged 12 years and under, athletes now range in age from 0 to 50+ years.
  - The sport is particularly popular in the US west coast. The California Interscholastic Federation reported that water polo is the fastest growing men's high school varsity sport and second fastest growing women's sport in 2013.
- Water polo had the highest incidence of injuries at the 2013 FINA World Championships.

### General Principles

#### TERMINOLOGY

- "Hole set": primary offensive position centered in front of the goal at the 2-meter line
- When a minor foul is committed within the 2-meter marker, or if excessive force is used, a player may be ejected for 20 seconds, resulting in a "man-up" advantage for the opposing team.
- "Brutality": the most severe foul type due to excessive force; results in immediate ejection from the current game and possibly subsequent games as well
- "Nail check": refers to fingernail check prior to a competition, during which referees may ask players to trim any sharp fingernails or toenails deemed likely to cause injury

#### HISTORY

- Men's water polo was among the first team sports included in the Olympic Games, first appearing in 1900.
- Women's water polo became an Olympic sport in 2000.

#### DISCUSSION OF SPORTS-SPECIFIC SKILLS AND OTHER CONSIDERATIONS

- During the course of a game, players must swim approximately 1000 meters.
- They must also tread water typically using the eggbeater kick, in which the right leg is rotated counterclockwise, while the left leg is rotated clockwise.
- Players must also be able to wrestle with opponents and pass and shoot the ball.

#### BIOMECHANICAL PRINCIPLES

- Players often swim in a "head up" position to maintain visual contact with the field of play.
  - The cervical spine is subjected to sustained periods of protraction and extension.
  - Can lead to disc injury, muscular neck pain, and/or posterior shoulder impingement
- Throwing: force transmission to shoulder stabilizers is more increased in water polo than other throwing sports.
  - Water is the only support base.
  - Ideally, power comes from the legs, hips, and torso, but, in reality, this can be limited by the defense.
  - The ball is heavier and larger than that used in other throwing sports.
- Eggbeater kick
  - Accounts for 40%–55% of the game
  - Applies a compression force to the medial knee joint
  - Leads to degenerative changes over time

#### EQUIPMENT AND SAFETY ISSUES

- Caps are fastened under the chin and worn throughout the game. Opponents must wear caps of contrasting colors, with red caps reserved for goalkeepers. Caps are numbered and fitted with malleable ear protectors to prevent injury.
- Facemasks or prescription goggles without hard or sharp edges may be used to correct vision and prevent eye injury.
- Mouth guards are commonly used to prevent dental injuries.
- Sharp fingernails and toenails are considered likely to cause injury and must be trimmed. A nail check may take place prior to the start of competition.
- Multiple swimsuits may be worn so that the athlete can continue to play if the top layer is torn during the competition. Swimsuits are tightly fitted to prevent opponents from grasping the suit to pull or hold the athlete.
- Due to the contact nature of the sport, water polo players at risk of concussion and should be educated about this issue.

#### RULES OF GAME/BASICS OF PLAY: WATER POLO

**Field of play**

- 10–20 meters wide with 20–25 meters between goal lines for women and 20–30 meters for men; water depth should be a minimum of 1.8 meters
- Goal lines and the half court are indicated with white markers. Red markers are used for the 2-meter line, and yellow markers are used for the 5-meter line.
- Goal cages should be 0.9 meters high, 3 meters wide, and 0.3 meters deep.
- For pools less than 1.5 meters deep, goal cages should be 2.4 meters high as the goalkeeper is permitted to stand during play. Field players may also stand at times but may not push off the bottom to gain a competitive advantage.

**Gameplay**

- Seven players per side during game play, including the goalkeeper
- Unlimited substitutions are allowed.
- Only the goalkeeper may touch the ball with both hands simultaneously.
- Only players with ball possession may be attacked.
- The arm holding the ball may be treated as part of the ball and may be yanked or pulled by opponents.
- The ball cannot be put underwater in an attempt to maintain possession.

**Timing**

- Four quarters of 6–8 minutes each, depending on the level of play
- A 30-second shot clock limits the time of possession without an attempt on goal.

**Fouls**

- Minor fouls outside the 2-meter line result in a free pass.
- Major fouls (either inside the 2 meter line or those with excessive force) result in a 20 second ejection of the offending player giving the opposing team a "man-up" advantage. A 4-meter penalty shot may also be awarded. The most aggressive offenses will result in a "brutality," in which a player is immediately excluded for the remainder of the game and in all likelihood the next game in the competition.
- A player who has committed three major fouls is excluded for the remainder of the game.

**Unique environmental and nutritional issues**

- Without goggles to provide protection, eye irritation due to chlorine exposure and chronic conjunctivitis are common. Regular treatment with artificial tears should be considered.

- Greasy and oily substances covering the body are not permitted. Sunscreen is permitted, but the body cannot be greasy or oily after application.
- Water polo players are heavier and tend to have higher body fat levels than elite swimmers. This can help players maintain position and aid in buoyancy.

## Common Injuries and Medical Problems

### SHOULDER PAIN

**Description:** The combination of swimming and throwing places water polo players at very high risk of shoulder issues. Limited body rotation leads to anterior capsule tensile overload and posterosuperior labral and bony glenoid impingement. Body roll with swimming is limited by the "heads-up" position, and body rotation with throwing is often limited by opponents. Internal rotators are often disproportionally strengthened with both swimming and throwing, which can lead to an altered isokinetic torque ratio and increased risk of shoulder injuries, similar to what is seen in baseball.

**History:** Pain is often exacerbated with abduction, forward flexion, the cocking phase of the throw, and the start of the catch with freestyle swimming. Pain may be anterior or posterior depending on the site of impingement.

**Physical examination:** Positive impingement testing

**Diagnostic considerations:** Chronic changes, including thickening of the capsule and RC, as well as biceps long head tendinopathy and osteochondral fractures, have been demonstrated in elite level players, some of whom may be asymptomatic. Physical contact increases risk of traumatic shoulder injuries.

- Dislocation and subluxation
  - Players may already have significant shoulder laxity associated with a background in swimming.
  - Posterior dislocation can occur while blocking the arm of a shooting opponent.
- Acromioclavicular joint injury (shoulder separation)
  - Typically occurs during a block
  - The scapula and clavicle are driven posteriorly until the ligament(s) rupture.
- Frank tearing of the rotator cuff (rare)

**Treatment:** Activity modification: stroke change during workouts, change in training schedule, substitution of a dry land program. Rotator cuff stretching and strengthening; surgical stabilization for traumatic shoulder dislocations, particularly if recurrent

### ELBOW PAIN

**Description:** Typically associated with stressing the ulnar collateral ligament (UCL) during throwing

**History:** Medial pain with overhead throwing

**Physical examination:** Medial pain or instability with valgus stress test, milking maneuver; olecranon impingement

**Diagnostic consideration:** Jones view may demonstrate compression damage to the lateral elbow joint. CT arthrogram to assess for acute ligament rupture

**Treatment/return to play/prevention:** See UCL Injury.

### HAND INJURIES

- Traumatic injuries, including lacerations, dislocations and fractures, are common.
- May occur during possession of the ball, blocking, follow-through, or while grasping an opponent to establish position

**Dislocation of the proximal interphalangeal joint**

- Dorsal dislocations
  - Occur with hyperextension while catching the ball or blocking a shot
  - Follow-up radiographs should be obtained to assess for fracture of the middle phalanx.
- Collateral ligament rupture
  - Occurs with twisting of the finger by an opponent in an attempt to escape the athlete's grasp
  - Treated by buddy taping the fingers for 3 weeks

**UCL injury (Gamekeeper's thumb)**

- Maximum abduction of the thumb is used to control the ball due to the large diameter.
- Impact to the ball or a misjudged pass may result in ligament rupture.

**Dislocation of the metacarpal-phalangeal joint**

- Rare
- "Buttonhole" type injury requires surgical reduction.
  - Metacarpal head inserts between the flexor tendon and the lumbrical.
  - The volar plate is trapped in the joint.

**Fractures**

- Result from direct blows during passing or shooting
- Waterproof splints allow for early return to swimming.

### HEAD AND FACE INJURIES

- Common due to the physical nature of sport
- Players may need assistance to be removed from the pool in case of closed head injury involving disorientation or loss of consciousness; an emergency action plan should be in place.
- A slap to the side of the face can sometimes result in rupture of the tympanic membrane despite use of protective caps; if the player cannot be kept out of water, a molded ear plug and bathing cap may be used to protect the area.
- Eye injuries include corneal abrasions, hyphema, and orbital blow-out fractures.

### LACERATIONS

- Web space tears
  - The most common laceration in water polo
  - Result from forcible abduction of two adjacent fingers
  - Simple lacerations may be irrigated and closed.
  - Can also cause ligament rupture involving the metacarpal heads or metacarpal–phalangeal joints
    - Treated with debridement, antibiotics, and reconstruction
    - Subsequent scarring may increase risk of recurrence due to limited abduction
  - Supraorbital lacerations are also common.
- Plastic-based waterproof antiseptic sprays can be used to seal and protect wounds closed by steri-strips, allowing return to play.

### NERVE INJURY

**Description:** Suprascapular nerve injury or entrapment; traction neuropraxia

**History:**

- Suprascapular nerve injury
  - Vague, diffuse, deep posterior shoulder pain
  - Onset may be gradual or sudden if associated with glenohumeral dislocation or injury to the cervical spine.
- Traction neuropraxia
  - Acute onset of symptoms
  - Associated with injury to the neck or shoulder girdle

**Physical examination:** Weakness of the arm and shoulder; atrophy of supraspinatus or infraspinatus in chronic nerve entrapment

**Diagnostic considerations:** Stryker notch view may demonstrate a calcified superior transverse scapular ligament or narrow suprascapular notch. MRI may detect soft tissue changes or ganglion cyst. Nerve conduction studies/EMG can help with confirmation and lesion localization.

**Treatment:** Rest, strengthen rotator cuff and scapular stabilizers, operative decompression in severe or refractory cases

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# SCUBA DIVING

# 83

George A. Morris

## GENERAL PRINCIPLES

### Overview

- Diving includes multiple activities performed in an aquatic environment.
- Technology allows people to enjoy the underwater experience.
- An estimated 1.2–1.5 million United States (US) divers participate each year.
  - People dive for various reasons, including new experience, unique environment and surroundings, new challenges, jobs, military duties, sport, and environmental awareness.
- There are few competitions including diving and underwater activities.

### Types of Diving

- Breath-hold diving (apnea, free, or skin diving)
  - Snorkel is the main piece of equipment used; may use fins for propulsion
  - Often performed in conjunction with spear fishing, shellfish gathering, and competitions
- Recreational scuba (self-contained underwater breathing apparatus)
  - Enjoyment of unique environments
  - Performed in several locations worldwide
- Commercial, technical, and advanced recreational diving
  - A wide range of gases used for respiration depending on the characteristics of the event
  - Specialized training for wreck, cave, night, and exploratory diving
  - Various gases are used, and decompression techniques may be required.

### Environments for Diving

- Multiple different underwater environments, each with different risks and techniques involved
- Open ocean water is the most common location for recreational diving.
- Fresh-water lakes and rivers
- Ice diving
- Cave and wreck diving
- Pools for competition and training

### Physiology of Diving

- Pressure at sea level on the surface is 1 atmosphere absolute (ATA).
  - Pressure in Denver, Colorado (elevation of 5280 feet) is 0.8 ATA, but at 6 feet under sea level, it will be 1.2 ATA.
  - Every 33 feet of seawater (FSW) below the surface increases the pressure by 1 ATA.
- Saltwater is approximately 775 times denser than air.
  - Increased density provides buoyancy to the diver.
  - Creates a sense of weightlessness, which can lead to disorientation or allow a freedom of movement for trained divers with neuromuscular disabilities
- Increased viscosity of water increases resistance to movement.
  - Requires approximately 13 metabolic equivalents (METS) to swim 1.3 knots (1 knot = 101 feet/minute), i.e., approximately 1.5 mph, compared to 3.3 METS to walk 3 mph on level ground
  - Contributes to the need for increased physical fitness of the diver and importance of fitness for diving evaluation
- Increased heat capacity of water
  - Conductive heat loss in water is 25 times faster than that in air, which leads to an increased risk of cold illness and need for thermal protection while diving.
- Human body's response to the aquatic environment
  - Air spaces subjected to increased pressure and rapid pressure changes with depth changes
  - Thermoregulation can be a challenge.
  - Shivering may hasten cooling in the water.
  - Diving reflex can occur as a response to facial immersion and leads to bradycardia, peripheral vasoconstriction, lower temperature, and shunting of blood to the core.

### Physics of Diving

#### Boyle's Law

- For a fixed amount of gas at a uniform temperature, volume and pressure are inversely related.
- $V_1/P_1 = V_2/P_2$
- As the diver descends and the surrounding environmental pressure increases, the volume of air-filled flexible wall structures (e.g., lung or gastrointestinal [GI] tract) will decrease; the volume of these structures will subsequently increase on ascent.
- Liquid and liquid/solid organs (e.g., blood, bone, muscle, and organs) will equally transmit pressures in all directions according to Pascal's principle, which states that pressure is distributed equally over surfaces and transmitted equally through compartments containing gas or liquids.
- Air-filled rigid-walled structures (sinuses, middle ear, and face mask air space) will remain at surface pressure at a depth owing to their inability to change volume.
- The pressure gradient is responsible for barotrauma injuries.
- The gradient can be resolved by equalizing spaces during depth changes.
- Gradient change is the greatest near the surface.

#### Dalton's Law

- The total pressure exerted by a mixture of gases is equal to the sum of the pressures that would be exerted by each of the gases if it alone was present and occupied the total volume.
- Air pressure at sea level (1 ATA) is composed of nitrogen (0.79 ATA) and oxygen (0.21 ATA), with trace amounts of carbon dioxide ($CO_2$), water vapor, and other gases.
- At greater depths, the air will be breathed in at the ambient pressure.
  - 33 FSW has a pressure of 2 ATA, which will be composed of nitrogen (1.58 ATA) and oxygen (0.42 ATA).
- Increases the partial pressures of the inspired gases and the body is sensitive to changes

### Oxygen

- Our bodies tolerate a wide range of oxygen pressure (0.158–2 ATA) and extract enough to meet the metabolic requirements without developing toxicity.
  - Below 0.158 ATA, the body will experience hypoxia, and the diver will develop air hunger, fatigue, confusion, loss of consciousness, and death of tissues.
- Brain cells are most sensitive and can die if deprived of oxygen for only 4 minutes.
- Oxygen toxicity can occur if the partial pressure is too high for too long.
  - Effects can include nausea, disorientation, visual changes, and seizures; life threatening underwater
  - Enriched air mixtures have varying concentrations of oxygen and different depth limits owing to the increased partial pressures of oxygen.

### Henry's Law

- The amount of gas that will dissolve in a liquid is directly related to the pressure (P) of the gas. Thus, $G_{(PD)} :: P$, where $G_{(PD)}$ is the gas physically dissolved in a liquid phase and :: stands for *proportional*.
- As divers descend to greater depths and pressures, the number of molecules of the gas dissolved in their body tissues increases.
- Factors that can affect how fast the dissolution occurs include temperature, solubility coefficient, and metabolism of the gas by our cells.
- Different tissues absorb and release gases at different rates.
- Tissue saturation eventually occurs if the diver stays at depth long enough.
- Upon ascent, partial pressure decreases and gas will leave the solution phase. If ascent is too rapid and/or decompression stops are not utilized, bubbles may form in the local tissue or the bloodstream.

### Nitrogen

- Usually considered an inert gas because it does not unite chemically with other substances in the body
- No effects at normal sea level atmospheric pressures (0.79 ATA)
- Nitrogen can interact with cells at increased pressures and lead to nitrogen narcosis.
- Effects of nitrogen at depth are comparable to taking one alcoholic drink for every 50 FSW descended, and at 750 FSW, it has anesthesia-like effects. This effect is also called "rapture of the deep" and can cause confusion and strange behaviors.

## Portions of the Dive

**Surface:** Swimming or wading at the surface before going underwater; can use energy or cause exposure to cold temperatures if prolonged

**Descent:** Involves changes in pressures of 1 ATA per 33 FSW; need to have equilibration of pressure in closed air spaces

**Bottom time:** Traditionally, the amount of time spent at lowest depth; however, with the advent of dive computers, it has evolved to the amount of time spent underwater. Activity at depth can change respiratory needs and risks of complications. Good buoyancy management can decrease the oxygen consumption and conserve the air supply.

**Ascent:** Risk of injuries because of changes in pressure and the release of absorbed gas back into local tissues and the bloodstream

**Surface interval:** Allows the body to resume its usual physiologic status and normalize tissue gas concentration before the next dive; complications or injuries encountered during the dive may not present until the diver is at the surface.

## Incidence of Dive-Related Injuries

- Difficult to determine because exact number of divers and dives performed each year is unknown
- Decompression sickness (DCS)
  - Approximately 1 DCS event per 5,000 dives
- 11–18 diving-related deaths per 100,000 members per year reported by Divers Alert Network (DAN) in 2007
- Increased age and decreased physical conditioning were the risk factors.
- Dehydration, exercise level during the dive, hypothermia, and hyperthermia may also play crucial roles.

## Safe Dive Profiles

- Follow standard recommendations on dive tables or dive computer algorithms for duration of dives at various depths, with decompression stops as needed.
  - US Navy Air Dive tables
    - Based on rectangular or square profiles that assume that the diver directly descends to the deepest depth and stays at that depth until returning to the surface.
    - Designed for safety to minimize risks of DCS, oxygen toxicity, and nitrogen narcosis
- Dive computers are now very common and easy to use.
  - Allow flexibility in dive profiles
  - Potential for less calculation errors compared to tables
  - Provides information on planning a dive profiles, safety stops, return to surface alarms, decompression times, or dive intervals
- Recommended that most sport scuba divers perform only no-decompression dives to minimize risks
- No dive is 100% safe because of individual variation in conditions, health, physiology, and equipment.
  - Diving within skill limits, equipment, and certification makes diving safer.
  - Keep ascent rate below 30 feet/minute.
- Always dive with a buddy.

## Importance of Basic Fitness for the Diver

- MET level needed for typical diving activities
  - Average scuba and skin-diving activities use 7 METS.
  - Remaining stationary against a 1-knot current can take up to 13 METS, similar to the level required to run 7–7.5 mph.
  - Getting in and out of the water can require significant strength and coordination.

## Guidelines for Diving

- Conditions that may disqualify a diver should generate a discussion regarding the risks versus benefits of recreational diving.
- Clearance for commercial, military, and technical divers is much more restrictive, and current US Occupational Safety and Health Administration (OSHA) and military guidelines should be reviewed.
- DAN is available for support if there are any questions about specific medical conditions and diving. A physician trained in dive medicine or appropriate specialist with knowledge of diving can be a local resource.

## Medical Events During Diving

- Cardiac causes are implicated in approximately 40% of diving-related deaths.
- Important to evaluate underlying fitness for activities in a predive physical examination.

- Predive physical examination may only occur once in a person's lifetime; hence, it is important to educate the diver about ongoing monitoring of his or her health.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Pressure-Related Diving Problems

#### Barotrauma

**Ear squeeze:** Most common pressure-related injury; occurs when the pressure inside the middle ear does not equilibrate with the ambient pressure and the tympanic membrane (TM) starts to displace inward, causing pain.

**Tympanic membrane (TM) rupture:** Pain of the ear squeeze is relieved, followed by a rush of cold water and dizziness; infection and chronic perforation can occur.

**Inner ear barotraumas:** Poor equalization can lead to rupture of the round or oval window. Perilymph may leak out. Tinnitus, dizziness, and hearing loss may occur. Chronic vestibular dysfunction or hearing loss may require surgical repair.

**Return-to-dive recommendations following barotrauma:** Divers with middle-ear symptoms may return to diving once the TM is healed or protected, hearing has improved, and the diver can equalize. Divers with chronic perforation or round or oval window rupture should not dive further because of the risks of permanent impairment.

**Sinus squeeze:** Failure to equalize pressure in the sinuses can lead to pain and epistaxis.

**Dental squeeze:** May occur if there is a small amount of air trapped under a filling or presence of a dental abscess

#### Pneumothorax (PTX)

**Description:** Can become a tension pneumothorax (PTX) on ascent due to expansion of trapped air in the pleural cavity (Fig. 83.1)

**Return to dive:** Consider no further diving if spontaneous; other causes, 3–6 months with documented healing, normal appearance on imaging, and normal function

#### Decompression Illness (DCI)

**Description:** A major concern for divers because it is a potentially avoidable event with possible serious consequences. Decompression illness (DCI) encompasses events that include DCS and arterial gas embolus (AGE) as well as other events that may be related.

**Etiology:**

- Henry's Law is the primary factor playing a role in DCI.
  - On-gassing
    - Gas molecules dissolve into tissues as a diver descends.
    - Different tissues have different rates of gas permeability.
    - Fast—lung
    - Medium—blood and organs
    - Slow—joint capsules/ligaments
  - Off-gassing
    - Dissolved gas molecules diffuse back into the bloodstream and transported to the lung for exchange/removal
    - Equilibration takes different amounts of time in different tissues.
- Small venous bubbles are common during the ascent portion of a dive, and they are generally filtered or exchanged without problems in the lung.
  - Large bubbles can cause a problem if they enter the arterial side of the circulation, such as in a diver with a patent foramen ovale (PFO).
  - Local effects of nitrogen bubbles in the tissue can affect skin, peripheral nerves, joints, connective tissue, and muscles. This is the most common type of DCS.

**DCS types:** Can be divided into categories with overlap possible

- **Type 1 DCS, mild**: Characterized by one or more symptoms including:
  - Arthralgia and myalgias that resolve within minutes
  - Pruritus and paresthesias
  - Rash, may be violaceous, mottled, and papular
  - Repeated or deep DCS exposure can lead to osteonecrosis.
- **Type 2 DCS, serious**:
  - Pulmonary symptoms can include burning substernal pleuritic pain, nonproductive cough, and severe respiratory distress.
  - Neurologic signs and symptoms can include focal spinal cord lesions with paresis, paresthesias, paralysis, loss of sphincter control, and impotence.
  - Symptoms do not always follow radicular or dermatomal patterns; may change over time

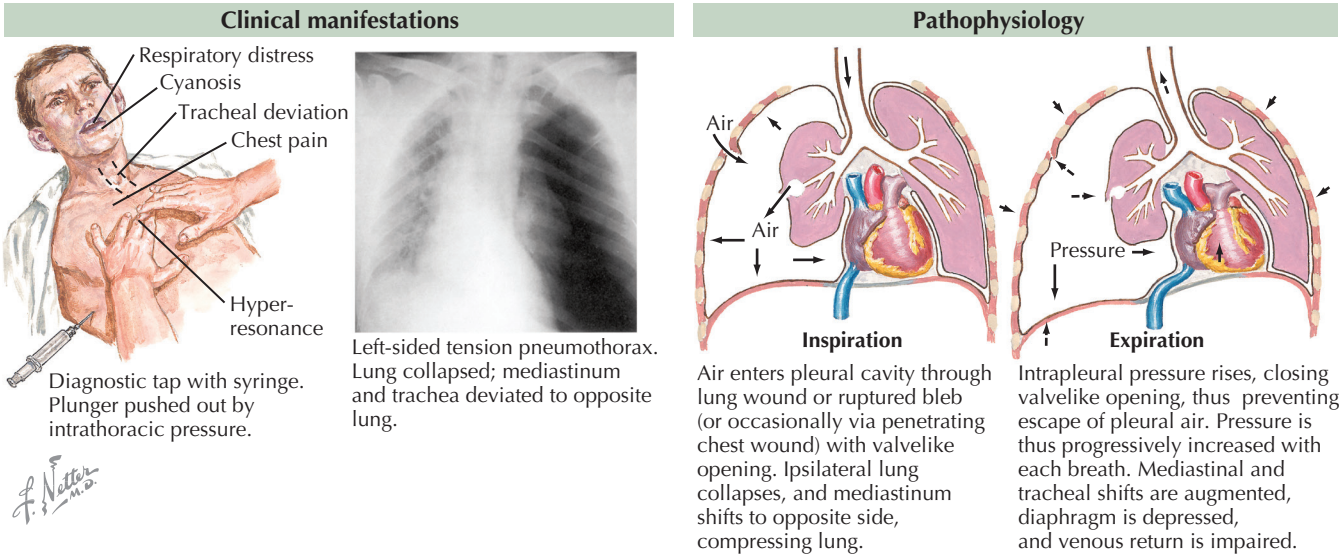


**Figure 83.1.** Tension pneumothorax.

- Cerebral type 2 DCS may present with headaches, visual changes, dizziness/vertigo, nausea, vomiting, tinnitus, hearing loss, focal neurologic deficits, and changes in mentation.
- Hypovolemic shock, circulatory collapse, and thrombus formation may occur.

**Presentation:** DCS injuries have been reported up to 36 hours after dive. AGE is a catastrophic event caused by rupture of alveoli with leakage of air into the pulmonary venous system, resulting in bubbles occluding the arterial circulation of the brain, heart, and other organs. Symptoms have an acute onset within 10–120 minutes of surfacing. Symptoms can begin with dizziness, anxiety, and headache and progress rapidly to loss of consciousness, seizures, shock, and death; often difficult to differentiate AGE from central nervous system (CNS) type 2 DCS. Treatment is similar: recompression and oxygenation. AGE can occur with any dive. DCS injuries often involve longer, deeper dives wherein tissue saturation occurs.

**Prevention:**

- Dive within tables/computer/conditions
- DCI has occurred in all types of dives, including free-diving
- Slow ascent with stops as required

**Risks of DCI:**

- Dive profile that exceeds no-decompression limits for time and depth
- Rapid ascent, dehydration, and exertion at depth
- Age, fitness, obesity, lung disease, and cardiac defects
- Prior DCI

**Treatment:**

- Recompression via hyperbaric oxygen treatment (HBOT)
- Dissolves gases back into the tissues and gradually returns the patient to atmospheric pressures and air source
- 100% oxygen during transport can displace nitrogen from the tissues and decrease symptoms and complications.
- Correct hypovolemia with intravenous fluids
- Aspirin may be considered for antiplatelet effects
- Transportation and initial resuscitation should begin while HBOT facility is being arranged.
- Rapid resolution of symptoms with HBOT is the hallmark of DCI.

**Return to dive recommendations following DCI:** Should be evaluated for predisposing factors, including equipment, dive profile, and medical causes; medical causes should be evaluated and either corrected or decisions made regarding the advisability of further diving.

- **Type 1 DCS**—usually may dive in 2 days
  - Consider 2–4 weeks if symptoms are prolonged or if oxygen or recompression required
  - Joint involvement: 2–4 weeks
  - Paresthesias: 4 weeks
- **Type 2 DCS** with:
  - CNS involvement cleared with recompression: minimum of 4 weeks
  - Persistent or residual deficits: no further diving
- **Arterial gas embolus (AGE):** minimum of 3 months if all symptoms cleared and no medical causes identified

## Loss of Consciousness Underwater (LOCU)

**Description:** Can occur for different reasons and is life-threatening owing to an individual's inability to protect his or her airway and return to the surface

- Seizures
- Hypoglycemia
- Syncope
- Cardiac event, ischemia, or arrhythmias
- Nitrogen narcosis
- Medication, alcohol, or drug use before diving
- Hypoxemia
  - Hyperventilation before breath-hold diving is common and results in decreased $CO_2$ levels; this leads to cerebral vasoconstriction and can reduce cerebral oxygen delivery; may lead to "shallow water blackout"
  - Carbon monoxide (CO) contamination can lead to hypoxemia despite adequate oxygen.

## Breathing/Air-Related Problems

- Air is the most common gas used and is composed of approximately 21% oxygen and 79% nitrogen.
- Oxygen toxicity can occur if pure oxygen is used or on prolonged deep dives with air.
- Nitrogen narcosis may occur on deep, prolonged dives.
- Nitrox is a gas with an increased percentage of oxygen (usually 32% or 36%) may be used to prolong bottom time and decrease risks of nitrogen narcosis.
  - Different risks, including oxygen toxicity, and should be limited to a depth based on oxygen percentage.
  - Nitrox tanks should be specially labeled and not filled with air or vice versa.
- Heliox is a mix using helium as the inert gas, which can alleviate the risks of nitrogen.
  - It requires specialized support staff and equipment; hence, use is generally limited to commercial and technical divers.
  - Longer decompression time is required.
  - Increased risk of tremors, seizures, loss of consciousness, and even death, if diving on heliox below 660 FSW.
- Trimix is a blend with oxygen, nitrogen, and helium, which also requires specialized equipment and training.
- Dirty air: If the compressor is incorrectly set up or malfunctioning, air may be contaminated with CO, which binds to the hemoglobin molecule approximately 250-times stronger than oxygen.
  - Symptoms may first start during bottom time with headache, confusion, LOCU, and can lead to death.
- Bad tanks: Interior of tanks may expose the diver to contaminants via their air supply. Tanks can become rusted if the interior is exposed to moisture, leading to a decreased partial pressure of oxygen inside the tank. Tanks should be annually inspected and pressure tested at least every 5 years. Tanks should be stored on their sides with approximately 500 PSI of gas in these to prevent exposure to moist air.

## Marine Animal Exposures

- Different types of marine creatures are encountered underwater. These need to be treated with respect and caution to minimize environmental degradation and risk to the diver.
- Underwater animals can move very quickly.
- Bites from animals such as eels, rays, sharks, groupers, and barracudas can occur.
  - Risks may be increased if spear fishing or in a feeding zone.
  - Treatment should start with standard treatment for traumatic injuries. Remove the diver from the exposure while not risking your life. Control bleeding and obtain treatment at the nearest trauma center. Clean the wound as able, and use broad-spectrum antibiotics that include coverage of *Escherichia coli*, *Pseudomonas*, *Mycobacterium marinum*, *Staphylococcus aureus*, *Streptococcus*, *Clostridium*, and *Vibrio*.
- Jellyfish stings usually only cause discomfort, but certain types can be fatal. For most jellyfish:
  - Nematocysts on the tentacles are responsible for the envenomation.
  - Local inflammatory response with pain and erythema

- Neurologic and respiratory depression along with nausea, vomiting, and abdominal pain from certain species such as the Portuguese man-o-war.
- Initial treatment is to remove the diver from the exposure and gently remove the tentacles to prevent further nematocyst firing.
- Ice, flushing with saltwater or vinegar, meat tenderizer paste, or topical anesthetics are helpful. Rinsing with alcohol may worsen the symptoms.
- Patients at a risk of anaphylaxis should be monitored further.

- Box jellyfish, also called *sea wasp*
  - Multiple species; the most venomous are in the Indo-Pacific region with related species in the Gulf of Mexico
  - Stings from multiple tentacles occur in linear whip-like arrangements on exposed skin.
  - Immediate and severe pain
  - Hypotension, tachycardia, and respiratory distress may alternate with hypertension, bradycardia, and apnea.
  - Cardiogenic shock can progress quickly, and death can occur within 10 minutes.
  - Treatment must be initiated early. Remove the tentacles and get out of the water, administer vinegar to the stings, oxygen, CPR, and treatment for anaphylaxis.
  - Parenteral pain medications and steroids may provide benefit along with intubation, sedation, administration of antivenom, if available, and intensive support in the hospital.
  - Delayed problems can include paralysis, pulmonary edema, abdominal pain, irritability, and localized skin necrosis.
- Sea bather's eruption is a reaction to thimble jellyfish larvae that can occur without the diver being aware of their presence because of their small size.
  - Larvae become trapped under wetsuit or bathing suit and discharge their nematocyst into the skin when they are removed from the water and are exposed to air or freshwater.
  - Effects include erythema and pruritus
  - Treatment can be initiated at the first sign of irritation and includes rinsing with saltwater, vinegar, 5% acetic acid solution, or using a meat tenderizer paste.
  - Topical corticosteroids generally help.
- Envenomations from fish, rays, and sea urchins on the ocean floor
  - Shuffling your feet and wearing thick protective footwear may minimize the risks.
  - Initial treatment: elevation and warm water or hot packs
  - Barbs and spines can be considered for removal.
  - Antivenom for stone fish is available.

## Dive Entry and Exit Injuries

- Low back pain from use of heavy and awkward equipment
- Exit and entering the boat with equipment often requires the maximum exertion for a diver. Swells and waves may increase the risk.
- Shore dives have risks related to slipping on rocks or coral or being thrown into them by waves, stepping on objects, wave trauma, and rip tides.

## Cardiovascular

**Coronary artery disease (CAD):** May allow if diver has adequate left ventricular ejection fraction (LVEF), no abnormalities with max stress testing to at least 13 METs, no recent ischemic events, and no history of exercise-associated events; should wait at least 6 months following percutaneous treatment or coronary artery bypass graft (CABG) and complete cardiac rehabilitation

**Congestive heart failure (CHF):** New York Heart Association (NYHA) classification ≥II should be advised against diving or if LVEF is <50% and divers require medications. Fluid shifts may occur with diving with possible immersion pulmonary edema.

**Hypertension:** May allow if well controlled, no trouble with exertion, and medications well tolerated with few side effects

**Murmurs, structural, and valvular abnormalities:**

- ASD and VSD should be a contraindication to diving if right to left shunting; may consider diving if there is a small membranous VSD without shunting
- PFO is often undiagnosed and asymptomatic.
  - Can increase the risk of DCI because of right-to-left bubble shunting during Valsalva maneuvers or ascent
  - Should be discouraged from diving if DCI during a safe dive profile; commercial divers may be considered for repair
- **Aortic stenosis (AS):** contraindicated if severe or if the diver has ECG abnormalities, poor blood pressure response to exercise, or is symptomatic
- **Mitral stenosis (MS):** contraindicated if severe, symptomatic, or with an elevated pulmonary artery pressure
- **Mitral valve prolapse (MVP), mitral regurgitation (MR):** not contraindications unless associated with arrhythmias, dilated left ventricle, pulmonary hypertension, or syncope
- **Artificial prosthetic valve patients:** generally disqualified because of anticoagulation
- **Hypertrophic obstructive cardiomyopathy (HOCM):** disqualification
- **Marfan syndrome**: disqualification
- **Congenital heart disease with cyanosis or decreased exercise tolerance:** disqualification

**Arrhythmias:** Long QT syndrome: contraindication to diving; patients with symptoms related to brady- or tachyarrhythmias should be disqualified from diving. Ventricular arrhythmias should disqualify a diver particularly if associated with structural abnormalities or reduced cardiac function. Pacemakers may be allowed limited diving following an appropriate postoperative period, exercise testing, discussion with the cardiologist, and remaining below the depth limits to which the pacemaker has been tested. Diving with an implantable cardiac defibrillator (ICD) is not recommended.

**Syncope:** Contraindication to diving if triggers unknown or if triggers may be encountered during dive-related activities.

**Peripheral artery disease (PAD):** Claudication can be worsened during a dive because of vasoconstriction from the dive reflex, cold temperature, and strenuous exercise, and symptoms may be mistaken for DCI.

## Pulmonary

**Asthma:** Not an absolute exclusion from diving; mild, stable asthma may be allowed to dive if the divers have adequate lung function, no current wheezing or symptoms, are able to achieve appropriate levels on exercise testing. Cold triggers are potentially dangerous for divers. The risks associated with asthma and diving are related to air trapping with the risk of alveolar rupture and AGE.

**Chronic obstructive pulmonary disease (COPD):** In general, because of anatomic changes and alterations in function, COPD patients are unable to tolerate the exercise capacity needed to dive; generally contraindicated in patients with decreased exercise tolerance and significant airflow obstruction

**PTX:** Higher incidence of spontaneous PTX in tall, thin, young male smokers, and up to 40% experience recurrence; this is a contraindication to further diving. Traumatic PTX patients should not dive for a minimum of 3–6 months, with absolute contraindication to diving if pleurodesis is performed. If dive-related PTX, no diving for 3–6 months if cause can be determined and healing and normal lung function ensured

**Pulmonary embolism:** No diving while on anticoagulants; no diving for 6–12 months until normal lung function and anatomy can be determined; underlying hypercoaguable state may represent a contraindication

## Central Nervous System (CNS)

**Seizure disorder:** Risk of death is high if a seizure occurs underwater; seizure medications may have undesired side effects and increase the risk of nitrogen narcosis; may dive if stable and seizure free and off medications for 5 years. Febrile seizures as a child are not a contraindication.
**Craniotomy:** Generally, a contraindication to diving
**Concussion:** Avoid diving while symptomatic.
**Cerebrovascular accident, transient ischemic attack (CVA/TIA):** Need to have appropriate recovery and rehabilitation time; functional limits should be evaluated in light of specific needs of diving and whether equipment can be modified; if patient is cleared to dive, may need to consider having two dive buddies; presence of carotid stenosis >70% may disqualify a diver.
**Motor/nerve disorder** (such as amyotrophic lateral sclerosis [ALS] or MS): May consider allowing if equipment needs can be met or modified; patients may have increased metabolic needs. Difficulty compensating for changes associated with diving, such as inability for the vascular system to adapt to the dive reflex; pulmonary/chest/diaphragm muscle involvement may reduce respiratory capacity during the dive. Thermoregulation may be affected; extra dive buddy recommended

## Gastrointestinal (GI)

**Gastroesophageal reflux disease (GERD):** Stomach squeeze following Nissen procedure may occur; reflux can increase because of frequent head-down position
**Abdominal hernias:** May have an increased risk of rupture or incarceration as the trapped air within the herniated segment expands upon ascent
**Bariatric surgery:** An uncomplicated surgery and successful postoperative course should not preclude diving; may wait for 1 year after the procedure
**Ostomies:** Not a disqualification

## Genitourinary (GU)

**Menses:** No increase in the risk of shark attack during menstruation
**Gas-filled penile implants:** Contraindicated because of the risk of rupture
**Pregnancy:** Diving is contraindicated during all trimesters.

## Ear, Nose, and Throat (ENT)

**Chronic sinus disease, allergies, or eustachian tube dysfunction:** Can predispose the diver to problems
**Sinus squeeze:** Can cause headache, pressure, and even bleeding
**Middle ear squeeze:** May cause pain and lead to TM perforation or round window or oval window rupture; equalization is best performed within the initial few feet of a dive and intermittently at the first sign of pressure
**TM perforation:** No diving with an unprotected perforation because of exposure of the middle ear to water and contaminants
**Motion sickness:** Primarily affects people on the surface portions of their dive; scopolamine is compatible with diving. However, several other antiemetics can increase the risk of nitrogen narcosis.

## Ophthalmology

**LASIK procedure:** Recommended to wait at least 2 weeks with final clearance per ophthalmologist
**Retinal tear and detachment:** Diving should be avoided for at least 2 months.
**Glaucoma:** May be disqualifying if surgically treated
**Gas-filled prostheses:** Are a contraindication to diving
**Corneal implants/rings or radial keratotomy (RK):** Wait at least 3 months before returning to diving, with final clearance per ophthalmologist.

## Psychiatry

**Anxiety:** Phobias and poorly controlled anxiety may lead to panic underwater, and place the diver and buddy at risk. Medications often used to treat anxiety can affect diving performance and increase the risk of nitrogen narcosis.
**Depression:** If controlled, is not a contraindication to diving; most antidepressants do not have significant adverse effects on diving
**Narcolepsy:** Advisable to be episode free for 1 year

## Endocrinology

**Diabetes mellitus (DM):** Insulin use is not an absolute contraindication to recreational diving. Restrict diving if problems occur with unrecognized hypoglycemia or hypoglycemia requiring treatment by others within the past 12 months. Restrict diving if poorly controlled or poor understanding of the disease and relationship to exercise; if allowed, the diver should monitor blood glucose levels before a dive. The dive should be canceled if ketones are present.
- Blood glucose goal ranges in mg/dL:
  - 1 hour before dive: 80–250
  - Immediately before dive: 150–250
- If too low or dropping faster than 20 mg/dL/hour, diver should eat a snack, delay the dive, and recheck blood glucose
  - If too high and no ketones, may treat with insulin and/or delay the dive
  - Sugar and glucagon should be available.
  - Dive buddy should be aware of the disease and know emergency treatments.
  - Consider an extra dive buddy.

**Thyroid disease:** Thermoregulation may be an issue.

## Musculoskeletal

- Should not dive with recent fractures or significant injuries because of an increased risk of DCI
- Diving with an acute disc herniation or radicular symptoms is not advisable.
- Total joint replacement with solid components is not a contraindication to diving.

## Hematologic

- Sickle cell disease: contraindication
- Sickle trait: may be allowed to dive within certain depth and duration limits
- Anemia: relative contraindication
- Coagulopathy, therapeutic anticoagulation: contraindication

## Rheumatology

- Raynaud's phenomenon: May lead to pain and inability to use hands when diving, increased risk of injuries, or inability to perform safe diving
- Lupus: Depends on severity and extent of the disease

## Medications and Diving

- Need to consider how the pressure changes, oxygen, nitrogen, potential for bleeding, and the underlying disease may interact
- Medications generally considered safe for divers include:
  - Antidepressants, aspirin, cholesterol-lowering agents, nonsedating antihistamines, and nonsteroidal anti-inflammatory drugs (NSAIDs)
- Medications that are contraindicated in divers:
  - Anticoagulants, anxiolytics, and opioids
- Other medication considerations:
  - Decongestants: should be used with caution and consider not diving if symptoms are severe enough
  - Antihypertensives: ACE inhibitors and ARBs are preferred.
  - Antibiotics appear safe, but ensure the infection is controlled.
  - PDE5 inhibitors have not been associated with any adverse effects although it may be prudent to avoid use of these medications within 2 hours of diving owing to risks of hypotension.
  - Antioxidants have not shown any benefit in decreasing dive-related injuries.

## Age-Related Concerns

- Professional Association of Dive Instructors (PADI) recommends that individuals must be at least 10 years old to participate in their Junior Divers program.
  - Divers aged 10–11 years must dive with a certified parent, guardian, or PADI professional, with a limited dive depth of 40 feet.
  - Divers aged 12–14 years must dive with a trained adult to 60 feet.
  - A junior diver who has reached the age of 15 years may transition to a regular certification without any additional testing.
- No upper age limit to diving; base decisions on overall health and fitness.

## Disabled Divers

- 15% of the US population is considered handicapped or disabled.
- Handicapped Scuba Association (HSA) International is a group of divers and trainers who have developed training programs and different levels of certifications to allow challenged/disabled individuals to enjoy opportunities to dive with safe and appropriate support.

## Postdive Care

- Divers may present with new symptoms a long distance from water owing to speed of travel and delay in symptom onset.
- DCI can present up to 36 hours after exiting the water.
- Safe interval before flying for no-decompression divers who have not experienced DCI symptoms and will be flying in airplanes pressurized between 2,000 and 8,000 feet.
  - A minimum 12-hour interval following a single dive
  - 18 hours minimum if multiple dives have been performed on the same day or during multiple days of diving
  - Longer intervals advisable for other situations; the longer the interval, the lower the chance of DCI

## SUMMARY

- Dive training agency certification cards (C-cards) are required by dive shops in order to fill tanks.
- Do not dive when ill or with recent changes in health status.
  - Acute upper respiratory infection may lead to difficulty equalizing.
  - Bronchitis or pneumonia may lead to pulmonary air trapping due to bronchial edema and mucous.
  - Acute gastroenteritis may lead to dehydration, increasing the risk of DCS or aspiration by vomiting into the regulator.
- Diving is a very dynamic activity; diving conditions in the same area can be completely different on different days.
  - Unknown risks, dangers, or emergencies can be encountered underwater.
  - Dive buddy is depending on you; your fitness may affect your buddy's safety as well as your own.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 84 SAILING

Joanne B. “Anne” Allen

## INTRODUCTION

- Understanding the sport of sailing and the wide variations in sailboat classes, events, equipment, crew positions, and physical demands is essential to caring for a sailing team.
- The sport has evolved from early yacht racing in England in the 1600s, to Olympic-level racing and now, worldwide extreme sailing.
- The recent advent of high-tech sailboats capable of increasing speeds is now causing the sports medicine world to improve their efforts toward safety and injury prevention.
- The specific needs of a team competing in the Volvo Ocean Race, where sailors race offshore for weeks at a time but are able to communicate to a “home-base” hospital via satellite and require an on-board medic to be a member of the crew, will be different than the needs of an Olympic sailing team racing on small dinghies and keelboats in day races.
- All events require high-performance considerations, as well as emergency management preparations, particularly with the increase in extreme sailing. However, individual events and crew demands will differ significantly.

## GENERAL PRINCIPLES

### History

- Sailing has been a vital mode of transportation and trade since the dawn of history, when boats were built by the Phoenicians, Egyptians, Greeks, and Romans.
- Different types of sailboats have been developed through the centuries, ranging from the Polynesian outrigger proa and the Chinese lugsail or “junk rig” to the Arabic triangular sail dhow.
- The first Dutch “yacht” arrived in England in 1660 as a gift to King Charles I.
- In 1661, two more yachts were built: *Catherine*, a second yacht to King Charles, and *Anne*, for the King’s brother. A competition between the King and his brother ensued, and the vessels raced along the Thames in the first pleasure sailing race in history.

### Competitive Sailing

- The first yacht club was founded in Ireland around 1720, and it was initially named the Water Club of Cork. It was later refounded as the Cork Yacht Club in 1828.
- The first club in England was founded in 1820 and was named the Royal Yacht Club. The first yacht club in the United States was founded in 1844: the New York Yacht Club.
- The golden age of yacht clubs and sailboat racing regattas started with the launching of the Royal Yacht Squadron’s Cowes Week regatta, held annually since 1826 in Cowes, England.
- Competitive sailboat racing continued to grow in popularity with the America’s Cup Race, which started in 1851 off the Isle of Wight, when the yacht “America” was victorious over the British competitors and claimed the “Hundred Guinea Cup,” which is now the oldest continuously contested trophy in the world and known as the “America’s Cup” (Fig. 84.1).
- As the sport of sailing grew, many other regattas and competitive events have developed, including offshore ocean racing, global solo-circumnavigation, and in-shore course racing.
- Well-known events such as the Fastnet Race, Volvo Ocean Race, and Around Alone Race are aided by modern day satellite navigation and other technologic advances.
- Sailing regattas have also been a part of the Olympic Games since 1900 and the Paralympic Games since 1992.
- Although professional sailors now dominate the sport in America’s Cup, Grand Prix, and Volvo Ocean Race events, a vast number of amateur sailors continue to participate in and enjoy the sport through international and national class championships, college and high school events, regional race weeks, and local club regattas.

### The Players

- The “players” in the sport of sailing are the sailors racing their boats in events governed by the international rules of the sport.
- Sailing is a sport played by all age groups, from junior sailors to masters and grand masters.
- Crews are often coed and open, but there are sometimes men’s and women’s divisions depending on the event and the boat class.
- Weight may be a limiting factor, but there are no “weight classes”; combined crew weight limitation suggested in order to equalize the boat’s performance.

### The Playing Field

- The sailboat racing “playing field” is a body of water—whether a river, a lake, or the open ocean.
- A distance race is held in or a “course” is placed on a constantly changing environment.
- The primary factors affecting the playing field include the wind, waves, current, temperature, and all types of weather systems.
- The “race course” itself varies depending on the type of event. It may be around a fixed buoy course or on a course of longer distances, perhaps from one seaport to another, or even from one part of the globe to another.

### Rules

- The Racing Rules of Sailing (RRS) are governed by the International Sailing Federation, and there are slight variations to the basic RRS depending on the type of race or the event. For example, match racing rules are slightly different from fleet racing rules because of the difference in format.
- Although a jury is often present to interpret the rules in case of protests, sailing is considered a Corinthian sport, in which participants are expected to abide by the rules and take their penalties when appropriate.
- Many sailboat races are similar to race car driving: the boat that completes the “course” quickest and crosses the finish line first, having followed the rules without infringement, wins.
- Some larger boat races have a “handicapping” system, requiring a rating system such as the Performance Handicap Racing Fleet rating system to be in place. In these races, finish times are calculated based on the rating and distance of the race.

**Figure 84.1.** America's Cup hydrofoiling yacht.

**Figure 84.2.** Rounding the mark.

- Basic rules of the "road" or "waterway" that apply to sailing vessels whether competing or not:
  - Right-of-way rules apply in situations when boats meet, such as starboard/port and windward/leeward convergences.
  - Knowledge of basic sailing techniques, including the points of sail, steering the boat through a tack or a jibe, and maneuvering the boat safely, is essential for anyone who is learning to race sailboats.

## Sport Classes

### Types of Races

- The most common types of races are fleet races, match races, team races, and distance races.
  - Fleet races involve multiple boats starting together and racing around a course (Fig. 84.2).
  - Match races are done in "flights," pitting two boats against each other in a round-robin format.
  - Team races involve teams of boats working together around the course to win a regatta.
  - Distance races are often from point to point and may even involve global circumnavigation.
- Events vary in duration. Some are single-day events; others are weekend regattas, race weeks, or even part of a longer series that occurs over a few months.
  - America's Cup races are match races that involve both a series of races and an elimination ladder.
  - The Volvo Ocean race is an around-the-world race with many individual legs and cumulative times.
  - Olympic and Paralympic races are a series of individual races with cumulative point totals.
- Races may involve many different types and sizes of boats competing against one another, with each having a "rating" or handicap assigned to it to even out the competition. Other races are "one-design," where all boats are identical.

### Boat Classes and Crew

- There are many different types of boat classes—from the America's Cup class, which is currently a fixed wing hydrofoiling catamaran reaching speeds of 46 knots, to the Volvo 70s, which require 13 crew members working together as a high functioning team, to the Optimist dinghy class that junior sailors learn to race single-handed.
- The number of crew or team members varies according to the type of boat and the specific requirements of each individual class.
- The 2016 Olympic Games sailing program will have 10 different disciplines. The boat class may change from one Games quadrennial to the next. In 2016, the boat classes will be Laser (men's one person dinghy), Laser Radial (women's one person dinghy), Finn (heavyweight dinghy), 420 (men's and women's two person dinghy), windsurfing RS:X (men's and women's board), 49er and 49erFx (men's and women's high performance), and NACRA 17 (multihull mixed class).
- Currently, the Paralympics has three boat disciplines—single-person (2.4 mR class), two-person, (SKUD 18 class), and three-person (Sonar class).
- Although the Laser is a single-person Olympic class boat, it is sailed very competitively all over the world by all age groups—from juniors to great grandmasters.
- Other boat classes that are very well known and participated in widely include J-24s, Etchells, Lightnings, Sunfish, 29er, Snipes, Melges 24s, Farr 40s, and TP-52s.

## Equipment and Skills

- The basic equipment for sailing includes a boat with a mast, sails, a keel or centerboard, a method to trim the sails to the wind, and a steering mechanism or helm.
- While some boats may be very complicated with extremely advanced technology, satellite navigational systems, and carbon fiber rigging, other classes of sailboats control and limit the allowed equipment in some fashion, usually permitting only one common design.
- Many major one-design regattas require sail measurements and boat weigh-ins, and some also require crew to weigh-in on a daily basis.
- Variations in sailing equipment exist depending on the type of boat sailed—whether it is a two-person dinghy that requires hiking straps, a multihull with trapeze capabilities, or a 10-person keelboat with large "coffee grinder" winch handles for trimming sails. Knowledge of the specific classes is essential for the proper care of the sailors.
- The skill sets required to competitively race sailboats at an advanced level are also variable but include helming (whether on a tiller or a wheel), trimming the sails (either on block

systems or large winches), hoisting and dousing different types of sails, spinnaker pole/system management, rigging the boat and making fine-tune adjustments, hiking or trapeze work, maneuvering the boat with speed and agility, and navigation of the vessel.

### Protective Gear

- Sailor athletes often require protective equipment based on the environmental conditions in which they are sailing. For example, cold and wet weather requires proper foul weather gear, and offshore distance racing requires safety harnesses, lifejackets, etc.
- Extreme sailing, for example, through the intimidating Southern Ocean around Cape Horn in the Volvo Ocean Race, may require dry suits and head protection in addition to harnesses and other safety equipment due to high winds, volatile weather systems, and floating icebergs. On the other hand, a youth sailor racing a 420 dinghy in a summer regatta may only need a bathing suit, hiking shorts, sailing gloves, boat shoes, a hat, some sunscreen, and a lifejacket.
- There is constant debate over the "requirement" of wearing lifejackets/personal flotation devices (PFDs) in regattas.
  - Usually the decision to wear PFDs is primarily the responsibility of the sailor, although the race committee or regatta organizer may make it a mandatory requirement based on factors such as temperature, weather, and sea conditions.
  - Use of PFDs is encouraged because there are many documented instances of capsizes, even in favorable conditions, where a lifejacket saves a life (Fig. 84.3).
  - With the increasing awareness of concussion in sports, debate about the possibility of mandating helmet use for young sailors and in high-speed sailing has been escalating.
  - Sun exposure is also a concern, and proper sunscreen in addition to UVA and UVB protective eyewear is recommended as ocular melanoma is on the rise in water activities.

### Safety

- Safety is a major issue because the marine environment can be a hazardous playing ground.
- Capsizes, collisions, and man overboard situations are the riskiest.

**Figure 84.3.** Life jacket and foul weather gear in Opti sailor.

- An understanding of the properties of the body of water/geographic area where a competition occurs is essential.
- Along with emergency management plans, appropriate seamanship skills and water rescue skills are needed. Safety at Sea training assistance courses are readily available.
- The proper prevention of injury and illness are also required elements of safety in this sport.
- Understanding of right-of-way rules and avoiding collisions, knowledge of navigation signals, proper training, practice of man overboard rescue drills, and overall seamanship are important for safe participation in sailing.

## SAILING-SPECIFIC MEDICINE

### Epidemiology

- Efforts have been made to advance evidence-based knowledge of sailing and sports medicine over the last 20 years with an increase in research.
- Research by groups such as Olympic and America's Cup teams has produced many beneficial changes that have trickled down to high school and junior sailing.
- The overall injury rates have been estimated to be between 0.29 and 4.61/1000 days of athlete exposure. The incidence of injury in an America's Cup team was reported by Neville to be between 2.2 and 8.6 per 1000 hours in sailboat racing and training, with the most common injury being sprains and strains. Nathanson reported fatality rates of 1.19 deaths per million sailing person-days, which is comparable to those in US football. The USCG reported 23 deaths/year related to sailing.
- Since there are hundreds of boat classes, each with their own specific crew positions and demands, there are distinct injury profiles, optimal training regimens, and individualized physiological stresses and demands that have been described.

### Physical Demands

- Physical demands on sailors vary with boat class and crew position; injuries may differ on the basis of specific job stressors.
- Some injuries appear to be more related to overuse dynamics, causing sprains and strains usually in the upper extremities, yet others may be more acute due to accidents, with contusions and lacerations being the most common traumatic injuries. Falls on the deck or companionway and being hit by objects (including the boom) are common mechanisms of getting injured.
- Many actions in sailing are sudden and sporadic, which may place the sailor at high risk while performing explosive, powerful moves, particularly during a tacking or jibing maneuver. This is when concussions and man overboard issues that can lead to drowning can occur.
- Constant isometric contractions occur during hiking on a long upwind leg and result in significant strength and endurance requirements for sailors.
- Whether using the technique of straight leg hiking or droop-seat hiking, sailors may be susceptible to back and knee problems (Fig. 84.4).
- Repetitive grinding on winches while trimming can result in shoulder and arm injuries.
- Inherent postures in many crew positions also play a role in musculoskeletal issues because some actions on a sailboat require twisting and hyperextension of joints and, in particular, may predispose a sailor to back injuries.
- Lifting sails and hoisting sails pose a particular risk, with difficulty in maintaining proper form on a moving vessel or using proper technique even on the docks.

### Injury Studies

See Table 84.1 and Fig. 84.6.

Figure 84.4. Medical safety team.

Figure 84.5. Cooling vest.

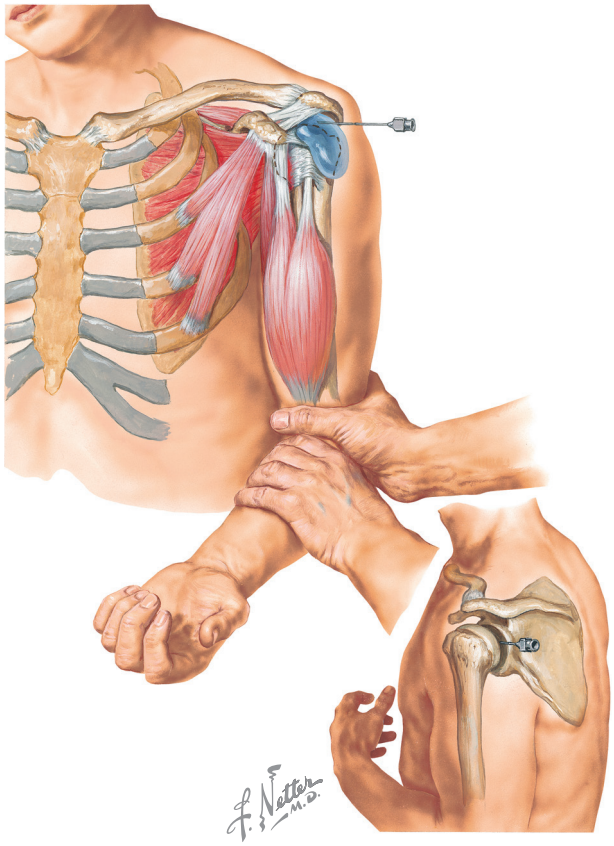


Figure 84.6. Injuries: Grinder's shoulder may require injections.

**TABLE 84.1 COMMON AREAS OF INJURY/PAIN FOR SAILORS**

| | |
|---|---|
| Smaller boats<br>Olympic classes | Lower back (52.9%)<br>Other back areas (41.2%)<br>Knees (25%–32%)<br>Thigh/leg (26.5%)<br>Neck (23.5%)<br>Shoulder (23.5%)<br>Forearm or elbow (20.6%) |
| Larger boats<br>America's Cup class | Lumbar spine (16%)<br>Shoulder (16%)<br>Knee (10%)<br>Cervical spine (8%)<br>Hand (7%) |
| Offshore endurance races<br>(Volvo Ocean boats) | Low back<br>Shoulder<br>Neck<br>Skin lesions |
| Disabled sailors<br>(Paralympic classes)<br>Boardsailing<br>(Windsurfers) | Upper extremity (60%)<br>Spine (20%)<br>Lower extremities (44.6%)<br>Upper extremity (18.5%)<br>Head and neck (17.8%)<br>Trunk (16.0%) |

## Environment and Illnesses

- Environmental issues concerning sailing include potential problems with polluted waters, such as those in Brazil at the Rio 2016 Olympic and Paralympic Games. Preventable illnesses have been prevalent among sailing teams working closely on a day-to-day basis, and proper hygiene always must be considered—including not sharing water bottles.
- Since many elite sailors often travel internationally to events, consideration of water and food contamination is also essential.
- Environmental issues such as hypothermia, heat illness, dehydration, seasickness, poor nutrition, and sun-related problems are also prevalent within the sport of sailing. Nathanson reported that in the Newport to Bermuda race, cases of seasickness were more prevalent when the weather was stormier. In a recent Chicago to Mackinaw Island race, severe thunderstorms and wind gusts up to 60 mph caused a racing yacht to capsize, and two of the tethered crew could not free themselves and drowned.
- Other obvious risks at sea include water immersion, near-drowning, drowning, and contact with aquatic marine life such as jellyfish.
- A unique property of this sport is the field of play environment. Special consideration should be taken with regard to the amount of time spent on the water, and necessary preparations should be made regarding thermoregulation with cooling/heating units, hydration kits on and off the water, and sun protection (Fig. 84.5).

- Since most events require sailors to be on the water for extended periods of time without access to land-based facilities, it is necessary to also consider gastrointestinal issues, bladder problems in some disabled sailors, diabetic/blood sugar management, adequate nutrition, and the ability to readily manage minor emergencies on the water.
- Illness and environmental influences may affect health care within the sport of sailing. As such, appropriate preventative measures, including proper clothing, adequate nutrition, and safety regulations, are recommended.

## Physiology of Sailing

- Research addressing the biomechanics and physiology in the sport of sailing has been recently increasing as more scientists are analyzing the performance of the sailboat racer.
- In general, studies suggest that the most physiologically demanding positions of the sport are the dinghy classes, which require more intense dynamic hiking maneuvers, and the big boat crew positions, which require the most agility or repetitive motion.
- One study of the 2002 Danish Olympic team showed that Laser sailors had the highest $\dot{V}O_2$ max at 58.3±4.2 mL/kg/minute, whereas the Finn and Star sailors had the lowest, with an average value of 47.6±3.5 mL/kg/ minute. Helmsmen and crew on trapeze boats have had results of 55.3±4.0 and 57.3±3.7 mL/kg/minute, respectively.

## Biomechanics of Hiking

- The physical stress of hiking, which is using body weight to counterbalance the tilting forces of the boat, has been documented and is believed to play an important factor in performance.
- Other recent studies have recognized hiking as a dynamic activity, measuring physiologic responses and force demands within an active sailing environment, whether on a simulator or on the water.
- One study found that the elite status and national rankings of Laser sailors were strongly correlated to quadriceps maximal voluntary contraction, isometric endurance, and tolerance of muscular fatigue (Fig. 84.7).
- Improper technique is commonly a factor in hiking-induced knee pain, resulting in unbalanced muscular forces around the knee joint.
- With fatigue, most sailors tend to isolate the vastus lateralis, leading to patellofemoral pain; turning out both feet with the legs extended increases the workload of the vastus medialis.
- Use of tight toe straps and plantar flexing the foot may help straighten the knees, centralize the force of gravity, and reduce the effort required by the quadriceps.
- The biomechanics of hiking in Paralympic sailing may change the dynamics, due to different force couples when using prosthetic legs for example, and should be considered when working with athletes with disabilities (see Fig. 84.7).

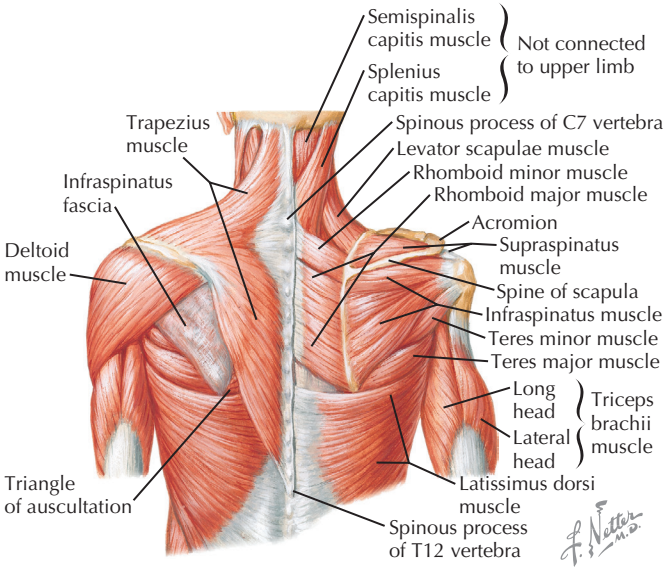
Physical demands of hiking out on a Laser.

Paralympic amputee hiking on a Sonar in Sydney Games.

**Figure 84.7.** Biomechanics of hiking.

## Training for the Sport

- With physical fitness joining boat speed and tactical intelligence as the main determinants of sailing performance, the issue of fitness training for sailors is accompanied by many opinion articles, but very little scientific research.
- The existence of various boat classes, crew positions, racing conditions, and baseline fitness levels makes it unfeasible to provide a uniform recommendation for fitness requirements.
- Strength, power, muscle endurance, cardiovascular fitness, weight management, flexibility, and agility all play varying roles in sailors' training regimens.
- Agility exercises may improve hand-eye coordination and the efficiency of movement about a sailboat, especially in high performance boats.
- Weightlifting routines should involve commonly used muscle groups, plus their antagonists, to maintain proper balance of strength, and should include a core workout.
- Aerobic training and fitness has been directly correlated to sailors' reaction speed to wind shifts, as well as enhanced endurance, decision making, and concentration, particularly in the latter stages of longer races.
- Physical and mental recovery between races and regattas also may improve with cardiovascular fitness.
- Position specific fitness training:
  - Hiking training should include muscular strength and endurance of the core and lower extremities, maintaining balanced force and flexibility about each joint.
  - Sailors who hike rely on muscle groups in the thighs, abdominals, hips, and arms, whereas sailors on a trapeze may focus more on upper body strength/endurance, aerobic endurance, and agility.
  - Grinders and many other big boat sailors should address aerobic endurance, as well as muscular strength, power, and endurance, particularly in the upper body.



Trimmer demonstrates Thera-Band shoulder exercises on the docks.

**Figure 84.8.** Training for the sport.

- Board sailors require sustained isometric action of the pectoralis major, deltoid, and scapular stabilizers, and therefore training should involve the shoulder girdle (Fig. 84.8).
- Sail trimmers (any boat size) should focus on training the arms, shoulders, and upper back (see Fig. 84.8).

- The proper timing of fitness training is important, whether that entails maintaining fitness in the off-season or reducing heavy training loads prior to regattas.

## Nutrition and Weight

- Weight management issues for sailors commonly involve reaching a perceived ideal mass for racing a small boat, or meeting the weight limits of a one-design class.
- Any weight loss should be gradual, focusing on reduced caloric intake while increasing aerobic exercise.
- The awareness of problems associated with the female athlete triad in women sailors is also critical, because they are often required to "make weight," which can lead to eating disorders.
- Whether sailing in a 1-day regatta or a long-distance offshore race, adequate nutrition to sustain proper blood glucose levels throughout the event is important for maintaining concentration and coordination.
- America's Cup sailors have been found to average 56 kcal per kg of body weight in daily energy expenditure.
- Dehydration can hamper performance, including increasing cognitive impairment and increasing risk of injuries.
- General guidelines for hydration in sports apply to sailors.

## Sports Psychology

- In recent years, there has been a growth in understanding of the importance of mental skills training, especially in high-level competition.
- Relaxation techniques, pre-race routines, and mental rehearsal may be beneficial to sailors at any level.
- Sports psychology can facilitate teamwork, focus, and organization.
- Including sports psychology professionals as part of a performance enhancement team is essential to the health and wellness of the sailors.

## Injury Prevention

- Understanding and accounting for different skill sets, an attention to proper mechanics, technique, posture, and positioning will assist the clinician in injury prevention.
- Injury prevention is best addressed through appropriate fitness training and properly caring for prior injuries.
- There is limited research to support injury prevention programs in sailing. However, it may be effective to include flexibility, hip flexor mobility, and core stability programs to reduce injury risk.
- Acute injuries are often related to accidents involving boat equipment and are difficult to prevent (e.g., lacerations from hitting sharp metal edges on the rig, abrasions from mishandling sheets, finger fractures from problems with winch overrides, contusions from free flying winch handles, concussions from contact with an accidentally jibing boom, or slips and falls on a wet foredeck). Fisher reported that although head injuries only make up 10% of sailing related injuries, many of these are severe, with >50% of them being fatal, usually caused by boom related injuries. It is uncertain if the use of helmets would prevent these.
- Properly addressing ergonomic developments, particularly in big boat design, also holds potential for injury prevention.
- Certainly, while the avoidance of accidents is preferable, overuse injuries may be more easily addressed with certain considerations.
- Returning to play is dependent on the nature of injury and the physical demands for which the athlete is returning to sail. Clinical decision making is appropriate.

## Crew Positions and Specific Injury Examples

**Helm:** Carpal tunnel syndrome (CTS) can be seen in both big boat sailing and dinghy sailing. Symptoms of CTS may be preventable by adjusting the grip width of the wheel at the helm of a larger yacht; avoiding a prolonged tight sustained grip position on the tiller may also decrease stress at the wrist and forearm in a dinghy (Fig. 84.9). In distance racing, platforms for the helmsman have recently been used on the large yacht in the Volvo Ocean Race to properly position the sailor's posture on long tacks, because standing in one place on a heeled boat for a prolonged time period can lead to overuse problems for these drivers.

**Grinders:** Attention to the grip size and angle of rotation on winch handles, and to the height and angle of pedestal winches, may also help prevent other overuse injuries such as lateral epicondylitis, often referred to as "grinder's elbow" (see Fig. 84.9). Lumbar spine injuries also frequently affect the grinder

Helm on a Maxi-Yacht.

Grinding on pedestal winches aboard Stars and Stripes.

The Bowman lives dangerously up the rig.

Trapeze work on the NACRA.

Hiking straps and foot position may affect knee injuries.

Protective helmet for Sonar Helmman.

World Champion Yngling Team hiking out.

**Figure 84.9.** Crew positions and specific injury examples.

position, and the biomechanical forces required for this skill set should be considered. These athletes must be assessed on an individual basis because the height of the sailor, for instance, may directly affect both potential injury prevention and performance of peak power output on a pedestal winch if the height of the pedestal is not adjusted appropriately.

**Mast:** The mast man on a larger yacht is also susceptible to injuries. Repetitive halyard hoisting at the mast with proper hand-over-hand technique, using the legs and trunk for strength and stability, may reduce overhead injuries to the shoulder.

**Trimmer:** "Trimmer's neck" has been described as neck pain related to the angle of cervical rotation and extension required to visualize the sail while constantly monitoring the tale-tells. Spinnaker trimmers may be especially susceptible to such problems because of the constant attention aloft that is required to fly this specialized sail.

**Bowman:** One of the most frequently injured crew members on a sailboat is the bowman, primarily because of the multiple physical tasks required by this position (see Fig. 84.9). Safety is essential for the bowman, who may be changing headsails in heavy weather and sea conditions, or jibing the spinnaker pole when running downwind. Concussions have been reported in this position from contact with the spinnaker pole, and many bowmen must be clipped on with a safety harness in order to avoid falling overboard in heavy sea conditions.

**Hiking crew:** Hiking is often required in heavy air conditions no matter what size boat is being sailed, but there is a difference in hiking out while sitting on a rail with a lifeline on a J-24 and hiking on a Laser. Dinghy sailing often requires a dynamic effort in heavy air conditions and will vary with the sailor and boat class. The hiking strap placement and foot position on the straps have been shown to be factors in forces generated at the knee and should be observed in any sailor with knee pain (see Fig. 84.9). The different hiking styles and torques placed on the knee when tacking the boat from a hiked out position may affect development of meniscal injuries or patellofemoral injuries in these athletes. Hiking crew members are also susceptible to lumbar spine disc problems and core stability is essential to preventing injury. In boats that use a trapeze, the harness fit may play a role in preventing low back pain, suggesting that custom-fit harnesses should be considered (see Fig. 84.9). Finally, hiking crew may also be susceptible to "rail rider's rump," or cutaneous rash, which can be worsened with unclean wetsuits or hiking shorts.

**Shore crew:** Unlike other sports, most sailors also have shore-based responsibilities for the boats on which they race, and proper caution when lifting boats onto trailers or moving large heavy sails/equipment on and off the larger vessels is essential to prevent lumbar strains and other injuries. However, as in other sports, some injuries occur during fitness training that can impact performance on the water, and should be monitored adequately.

## Boardsailing

- Since boardsailing is slightly different in its rules and techniques, the prevailing injuries may differ from other classes in the sport.
- The constant pumping action of the sail by the board sailor often causes increases in forearm compartment pressures, which can sometimes be relieved with daily massage and flushing.
- The newer segment of kite-boarding will have its own unique injuries, with problems including traumatic injuries from lofting and wind-shear dropping.

## SUMMARY

- While a team doctor's handbag for sailing will not differ from many other sports, his or her knowledge about the sport is critical for the successful caring for sailors.

- Injuries and illnesses will occur and will vary with the type of boat, the event, and the crew position.
- Finally, as with most teams, caring for the athlete on and off the field of play is essential to a healthy team.
- Prevention, whether through proper nutrition, adequate hydration, strength and endurance training, cardiovascular fitness, injury assessment, mental preparedness, environmentally appropriate clothing, or safety awareness, is the cornerstone to a healthy sailing team and the enhancement of the team's performance.

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Available at www.ExpertConsult.com.

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# 85 ROWING

Jane S. Thornton • Constance M. Lebrun

## INTRODUCTION

- As a competitive sport, rowing dates back several hundred years and was an original sport in the modern Olympic Games.
- First intercollegiate sport in the United States; initial race held in 1852 (Harvard vs. Yale)
- Since the introduction of Title IX regulations requiring equal proportions of male and female athletes in collegiate sports, the participation of women in collegiate rowing has surged, from roughly 1000 in 1981–1982 to approximately 7500 today.

## GENERAL PRINCIPLES

### Physiology

#### Training

**On-water:** Usually high volume (1–3 times daily, 1–2 hours in length), with higher intensity pieces and intervals during the summer racing season

**Indoor:** On rowing ergometer; simulates water training and monitors fitness

**Cross-training:** Weights, running, cycling, and cross-country skiing; used to supplement water training or during winter. Several injuries result from inappropriate transition to cross-training from on-water practices (and *vice versa*).

#### Racing

- Anaerobic contribution 10%–30%; the aerobic system supplies the remainder
- Ranks among the most strenuous of sports with high cardiovascular strain and lactate measurements of ≥15–20 mmol; $\dot{V}O_2$ max values can exceed 70 mL/kg/min in elite rowers

#### Race Distances

**2000 meters:** Olympic distance and standard for collegiate and club spring/summer racing; boats line up side-by-side at starting gates in up to six lanes. Races typically last for ≥5.5–8 minutes, depending on the event, weather, and rowers' ability. The World Best Time in the Olympic men's eight event is under 5 minutes 20 seconds.

**1000 meters**: Paralympic distance and standard for Adaptive (para-rowing) and Masters competitions

**Head racing:** Predominantly in the fall season; distance usually is ≥3 miles against the clock from a moving start and involves steering on rivers that bend and turn.

### Athlete Classification

- Rowers are classified by sex, age, weight, and ability (para-rowing).
- Age categories are Junior (age ≤18 years), Senior B or U23 (<23 years), Senior (open), and Masters (≥27 years).
- Weight categories are lightweight and heavyweight/open.
  - Both types of rowers have similar build, although lightweights typically have lower body fat.
  - Being tall and lean allows maximum stroke length while minimizing drag on the boat.
- Weight restrictions: Lightweight rowers typically weigh-in 2 hours before racing at or below the maximal weight. Weight restrictions are as follows:
  - **Men:** 70-kg crew average, 72.5-kg individual maximum
  - **Women:** 57-kg crew average, 59-kg individual maximum for an international competition, and domestically, 130 pounds with no average
- Coxswains: Steer the boat using a rope attached to the rudder; make technical and motivational calls and decisions about strategy; minimum weight:120 pounds (men's crews) and 100 pounds (women's crews); if underweight, must carry weight for racing
- Para-rowing comprises three classifications based on the nature of disability:
  - AS: Arms and Shoulders
  - TA: Trunk and Arms
  - LTA: Legs, Trunk, and Arms; further divided into PD (physical disability), VI (visually impaired), and ID (intellectually disabled)

### Equipment and Safety Issues

#### Boat Types

**Sweep:** Rower uses one oar, placed on starboard or port (left and right, respectively, from rower's perspective); available boats: pair, four, and eight; the eight is the only boat to always use a coxswain (Fig. 85.1A)

**Sculling:** Rower uses two oars; available boats: single, double, and quadruple sculls (Fig. 85.1B)

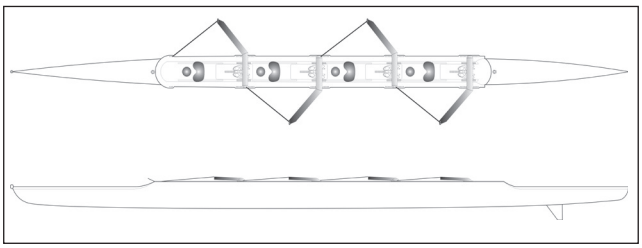
**Rowing shell:** Classically wooden, now constructed of synthetic materials such as carbon fiber; seats with wheels roll along fixed tracks. Athlete faces backward and pushes away from fixed shoes. The oar is held in an oarlock attached to a rigger that extends out from the shell. Equipment can be adjusted within a range (e.g., changing placement of feet or oar lengths to aid in loading), called "rigging" (Fig. 85.1C).

**Oars:** Classically wooden, now primarily carbon fiber; consists of a handle with varying grip sizes and material, a collar and sleeve that fits into oarlock, and a blade that enters water and provides resistance to move boat. Oar design has changed over time to generate more force per stroke, typically through shorter overall length and greater surface area, which may account for an increase in injury rate.

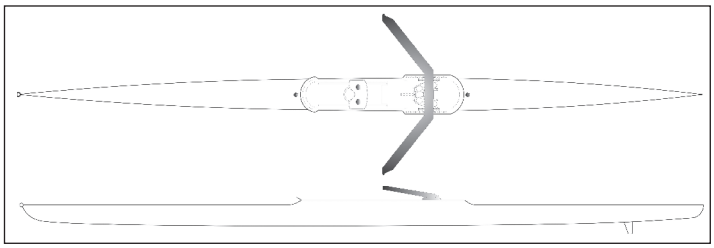
**Indoor rowing ergometer (colloquially "erg"):** Used for fitness testing, training, and racing, primarily during winter, although erg testing is often used selectively throughout the year. Most common models consist of a sliding seat on a single rail, a fixed footplate, and a handle attached to a chain. The chain spins a flywheel that creates resistance, and small monitor displays power output, split times, and stroke rate. In addition to stationary erg, dynamic erg systems are available where flywheel and footplate also move, resulting in lower load per stroke and, theoretically, fewer injuries.

### Phases of Rowing Stroke

**Catch:** Legs and back fully flexed, arms fully extended; rower's seat is at front of slides and the blade enters water in a "squared" position (blade perpendicular to water) (Fig. 85.2A).



**A.** Example of sweep boat: the straight or coxless four.



**B.** Example of sculling boat: the single.

**C.** Inside of rowing shell.

**Figure 85.1.** Types of rowing boats. (Photographs © Dr. Volker Nolte.)

**Drive:** Legs extend and back begins to extend slightly, while arms and shoulders remain relatively fixed; once back has extended to neutral position, arms begin to flex and continue acceleration of blade through water (Fig. 85.2B).

**Finish or release:** Legs flat and fully extended, shoulders behind hips but back still slightly flexed, arms flexed; blade is removed from water by simultaneously putting weight on handle (tapping down) and feathering (turning the wrist so that the blade is parallel to water) (Fig. 85.2C).

**Recovery:** Reverse of drive sequence; arms extend to move the oar handle forward, back becomes more flexed, and knees are flexed to bring rower into position for next catch.

## APPROACH TO INJURY EVALUATION

- A vast majority of injuries to competitive rowers of all ages/abilities are overuse injuries.
- Inappropriate stroke mechanics and asymmetries can predispose a rower to injury.
- Other factors include poor weather conditions that affect stroke mechanics, fit of equipment and rigging, and inappropriate transition from indoor to on-water training.
- If possible, observe rowing technique on water or by using an ergometer. Watch for compensatory behavior related to poor flexibility, muscle or strength deficiencies, abnormal asymmetrical movements, or undue force placed on the injury site in question.
- Most common injury sites for males: lumbar spine, forearm/wrist, and knee; for females: chest, lumbar spine, and forearm/wrist
- Rib stress fractures are also typical and generally unique to rowing.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Low Back

**Description:** Most frequently injured region, accounts for up to 25% of all reported rowing injuries

**Mechanism of injury:** Large loads are placed on lower back; lower back muscles are relatively relaxed as rower approaches catch position. At catch, spinal extensors are quickly loaded with resulting compressive forces at the spine, building and reaching peak compressive forces at approximately mid-drive (estimated at >4 times a rower's body mass). Sweep rowing introduces increased rotation through spine because rower reaches at catch to maximize stroke length. Fatigue of spinal extensor muscles, training intensity, and skill level may result in decreased ability of lumbar spine to resist forces during drive on passive spinal structures (e.g., ligaments and discs). Onset of low back pain is generally associated with a prior history of injury and ergometer training sessions of >30 minutes.

**Types of injury:**

- **Muscle strain:** Most common injury; low back pain, involving erector spinae muscles, quadratus lumborum, and/or sacroiliac joints
- **Sacroiliac joint dysfunction:** May result in pain over buttock, lateral thigh, pelvis, and groin; contributing factors may include leg length discrepancies, underlying hypermobility, or constant unforeseen balance changes. Greatest demands on sacroiliac joints occur as forces generated by legs are transferred to trunk during early to mid-drive.
- **Lumbar disc herniation:** Also very common; compressive loads applied to the lumbar spine in flexion may contribute to disc bulge or herniation. This may be associated with or progress to spinal nerve impingement, causing radicular symptoms (pain or numbness radiating into legs) (Fig. 85.3AB). However, it is important to remember that initial symptoms may be limited to centralized back pain.
- **Spondylolysis:** Generally observed in young rowers, likely caused by repetitive axial loading of pars interarticularis; weight training rather than rowing induced all cases of spondylolysis seen in a group of rowers. If spondylolysis exists bilaterally at a particular level of the spine, spondylolisthesis (forward slippage of one vertebra on the one below) can result (Fig. 85.3C).

**History:** Usually insidious onset during periods of high-volume or high-intensity training; pain may be localized to one region and radicular symptoms may be present. If pain worsens with flexion and extending from the flexed position, suspect muscle origin and disc pathology; pain worsens with extension may indicate spondylolysis and spondylolisthesis

**Physical examination:** Standard; observe range of motion, reflexes, distal motor and sensory nerve function. Special addition: standing single-leg back extension for spondylolysis (worse

**A.** The catch.

**B.** The drive.

**C.** The release.

**Figure 85.2.** Phases of rowing stroke. (Photographs © Dr. Volker Nolte.)

back pain on side of standing leg); straight leg raise, sitting root, and neural tension tests for discogenic and herniation problems

**Diagnostics:** If indicated, begin with plain radiographs with oblique views to search for pars interarticularis defect or proceed to bone scan or computed tomography (CT), including single photon emission CT (SPECT). If disc herniation suspected, proceed to magnetic resonance imaging (MRI)

**Treatment:** Include therapeutic modalities such as ultrasound and interferential current as indicated and/or mobilization/manipulation; incorporate core stabilization exercises, relative rest, and cross-training, with gradual return to sport. Disc herniation infrequently requires surgical intervention although outcomes are similar. Spondylolysis usually requires extended periods of time off from training (e.g., a full season). Spondylolisthesis infrequently requires surgical fixation (Fig. 85.3D).

**Technical changes:** Analyze trunk movement, particularly lumbopelvic motion. Lumbar flexion and extension should be accompanied by appropriate pelvic tilt; maintaining a more neutral spine during stroke may help. Although equipment adjustments can lessen load per stroke, this may be unrealistic during racing season. Rigging changes may affect entire boat, impacting speed. However, lowering load setting on an ergometer during indoor season is recommended and simple to perform. Address imbalances: low hamstring-to-quadriceps strength ratio, strength asymmetries between right and left erector spinae, and hip muscle imbalances in females. Emphasize appropriate breathing patterns: expiring through the drive phase may offset high levels of shear force and compression.

## Thorax

**Mechanism of injury:** Remains controversial; factors include weakness of surrounding musculature, specific rib architecture (angulation, diameter, etc.), joint stiffness, and repetitive strain. Serratus anterior and external obliques historically implicated; more recently thought to be repetitive stress from external obliques and rectus abdominis (Fig. 85.4)

**Types of injury:**

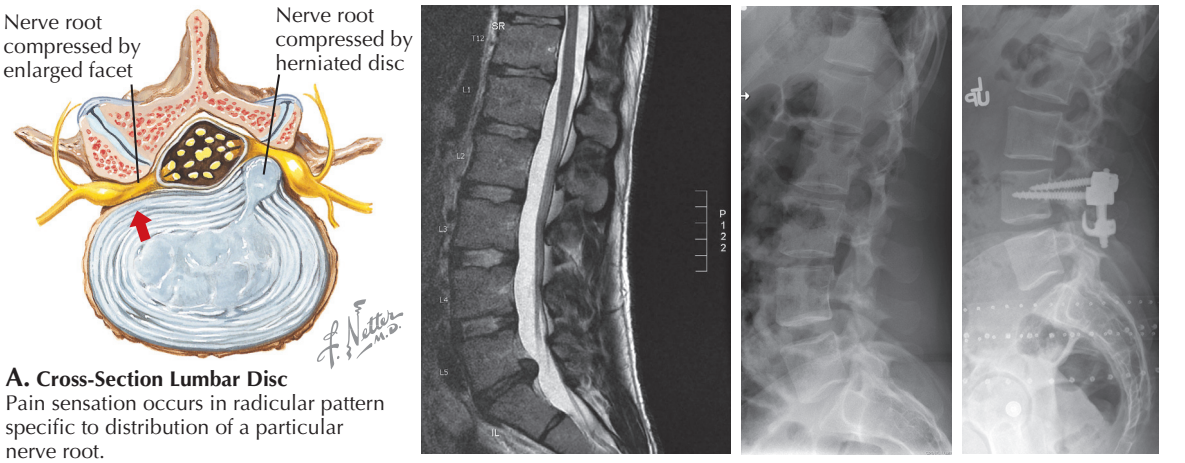
- **Rib stress fracture:** Increased incidence after more efficient blade design in 1992; rates of occurrence range 6%–24%; higher incidence in elite, heavyweight, and female rowers; most common in anterolateral/lateral aspects of 4th–8th ribs; equal incidence between sweepers and scullers, although location differs. AS and TA para-rowing athletes may be at an increased risk due to compression of ribs from chest strapping that is used to secure athlete to boat.
- **Costochondritis:** Poorly understood condition, although most likely to occur during sweep rowing because of excessive rotation or altered thoracic spine and rib mobility
- **Intercostal, rhomboid, and serratus anterior muscle strain:** Often confused with rib stress fracture at initial presentation; case study of complete avulsion injury of serratus anterior

**History:** Usually insidious onset with generalized chest wall pain; worse with rowing, reaching, rolling over in bed. Over time, pain associated with stress fracture becomes more localized to a specific rib. Costochondritis will exhibit pain and tenderness on costochondral or costosternal joints without swelling. Muscle strain exhibits nonspecific tenderness on palpation.

**Physical examination:** Stress fracture: pain localized over rib; most commonly anterolateral fourth through eight ribs but can be posterior; worse with rib compression. Athletes may have palpable rib callus. Costochondritis: adduction of arm on affected side and rotation of head toward the affected side will reproduce pain. Examine thoracic rib cage to ensure appropriate segmental rotation.

**Diagnostics:** Important to exclude stress fracture; plain radiographs may detect callus formation, but bone scan remains the gold standard for timely diagnosis (see Fig. 85.4).

**Treatment:** For stress fractures: analgesics, icing, electrotherapy, and thoracic spine mobilization; relative rest (4–6 weeks), with gradual return to sport; rowers should be evaluated for low calcium, vitamin D, disordered eating, and hormonal disturbances such as oligomenorrhea/amenorrhea or low testosterone that can lead to decreased bone density and higher rate of fracture



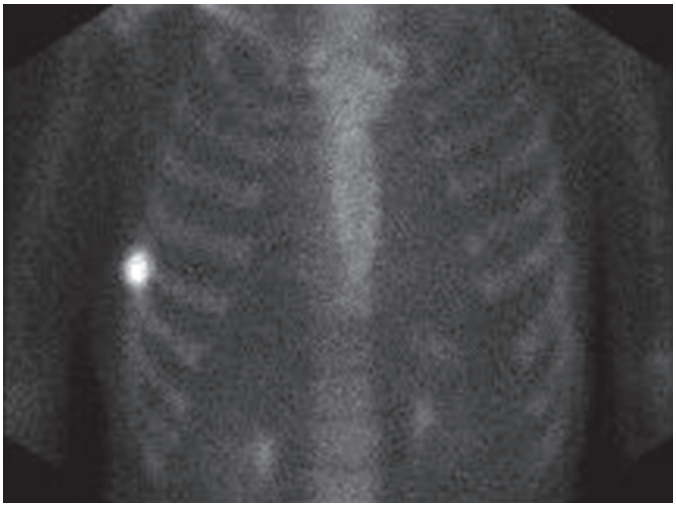

**A. Cross-Section Lumbar Disc**
Pain sensation occurs in radicular pattern specific to distribution of a particular nerve root.

**B.** Disc herniation (Courtesy of Grant James.)

**C.** L4 bilateral spondylolysis (without spondylolisthesis) (Courtesy of C. Lebrun.)

**D.** Spine fixation (Courtesy of C. Lebrun.)

**Figure 85.3.** Low back injuries.



RSF bone scan (Courtesy of C. Lebrun.)

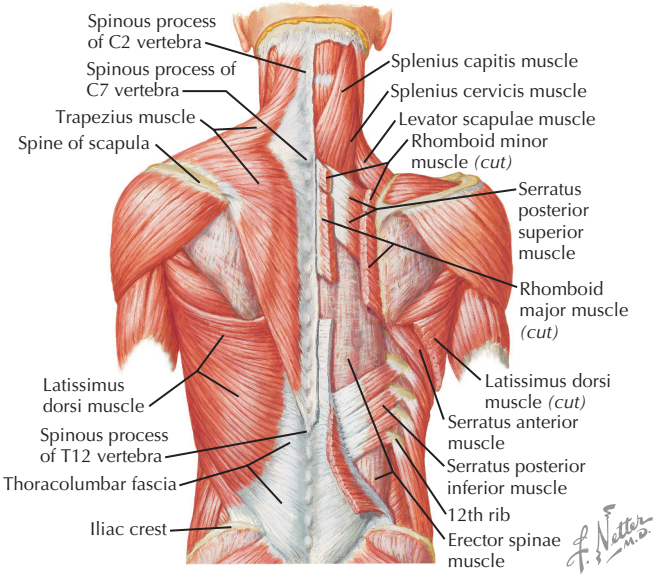



**Figure 85.4.** Thorax injuries.

Costochondritis: thought to be self-limiting, continue athletic participation as symptoms allow

Muscle strain: often rower is able to return to sport earlier, even with lingering pain (not indicated for stress fractures)

**Technical changes:** Address muscular imbalances and/or hypermobility in specific regions. Rowing on dynamic ergometers may reduce the risk of overuse injury without compromising training efficiency and rowing performance. For para-rowers, adjustment of strapping may help, although this may be limited by rules and regulations.

## Shoulder

**Mechanism of injury:** At catch, the scapula is retracted and the humerus is elevated to transfer forces from the oar to legs and back. Most common pathology is a combination of anteriorly placed glenohumeral head, tight posterior capsule, tight latissimus dorsi, and weak rotator cuff muscles. Common technical error is to activate upper fibers of the trapezius at catch instead of engaging mid-to-lower trapezius and latissimus dorsi, particularly in scullers; usually exaggerated in outside (furthest from blade) shoulder in sweep rowers

**Types of injury:**

- **Rotator cuff tendinosis:** Overuse injury related to technical error and/or muscle strength imbalance
- **Capsular instability:** May be exaggerated by reaching too far forward at catch or not extending fully at finish through the upper back
- **Clavicular stress fracture:** Case report of lightweight rower with sudden resumption of training; uncommon but highlights the need to consider surrounding structures of shoulder girdle in differential diagnosis

**Physical examination and treatment:** On physical examination, there will be localized tenderness over affected structures or muscle insertion (most commonly supraspinatus and/or biceps). Physical therapy should focus on stretching the tight posterior

capsule and correcting posture and muscle imbalance, with special attention to scapular stability. Surgical correction may be indicated in severe cases.

**Technical changes:** Limit over-reaching at catch and ensure linear motion of shoulder girdle through the entire drive

## Knee and Hip

**Mechanism of injury:** Inappropriate foot and knee alignment during each phase of the stroke cycle can lead to overuse injury and pain in the knee and hip regions. Vastus medialis muscle, usually underdeveloped in rowers, may cause patellar tracking problems.

**Types of injury:**

- **Patellofemoral pain:** Two primary factors to consider: overcompression at catch straining surrounding structures of flexed knee and tendency of the knees to slightly buckle or pop up at finish. If athletes cannot fully extend at finish, normal function of vastus medialis cannot occur; three remaining quadriceps will cause lateral tracking of the patella. Certain females may be predisposed to patellofemoral pain because of wider pelvis (Fig. 85.5).
- **Iliotibial band (ITB) friction syndrome:** Compression at catch combined with varus knee alignment may cause friction and ensuing pain as iliotibial tract slides over lateral condylar prominence of the knee. Rowers who run for cross-training may encounter similar problems.
- **Labral tears:** May be caused by repetitive hip hyperflexion (+/– internal rotation) or with athletes with underlying femoroacetabular impingement (FAI)—seen in young rowers—may be career-ending

**History:** Patellofemoral: dull generalized pain in retropatellar area, worse going up and down stairs, or prolonged bent knee position (positive "theater sign"); ITB: pain over lateral aspect of the knee, remaining knee examination typically normal

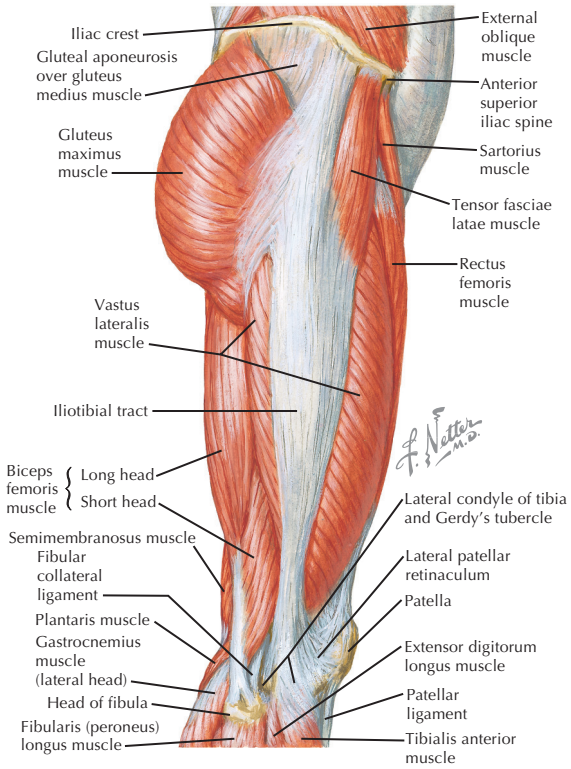



**Figure 85.5.** Knee and hip injuries.

**Physical examination:** Usual knee and ITB examination; check for lateral tracking with bent knee, malalignment such as genu valgum or genu recurvatum, excessive pronation, or internal tibial and/or femoral torsion. ITB symptoms should prompt examination of possible leg-length discrepancy or pelvic malalignment.

**Diagnostics:** None indicated, but large knee effusion or significant locking or catching may suggest meniscal or other knee pathologies

**Treatment:** Taping the patella to prevent maltracking useful in the short term. Strengthening of vastus medialis and gluteus medius muscles; bracing not advised due to range-of-motion limitation

**Technical changes:** Modify the shoe position to alleviate symptoms (angle, distance between toes and heels, and height); wide foot placement may compound problems; modify position of tracks to ensure full knee extension at finish of the stroke

## Wrist and Forearm

**Mechanism of injury:** Relatively common, usually from excessive wrist motion during feathering/squaring actions, as well as excessively tight grip of the handle in rough water conditions or with inexperienced rowers; other factors: wrongly sized/slippery grips, poor rigging, and fatigue

**Types of injury:**

- **Exertional compartment syndrome:** Often volar compartment, overuse injury that may occur because of repetitive and inappropriate initiation of pull-through with elbow instead of shoulder girdle at catch, or inability to relax forearm grip at release
- **Lateral epicondylitis:** May occur because of same factors; characterized by pain over lateral aspect of elbow and with resisted wrist extension
- **De Quervain and intersection syndrome:** Tenosynovitis of first and second dorsal compartments, respectively; intersection syndrome, often misdiagnosed as de Quervain, is also referred to as "oarsmen's wrist" (Fig. 85.6).
- **Tenosynovitis of wrist extensors or "sculler's thumb":** Swelling over dorsal aspect of forearm, caused by hypertrophy of abductor pollicis longus and extensor pollicis brevis muscle bellies; may be due to inappropriate use of thumb to feather oar at finish or by allowing palm to slide down handle while keeping thumb rigid against handle end
- **Carpal tunnel syndrome:** Pain over wrist and distal median nerve paresthesias; positive Tinel's and Phalen's sign; evidence of wasting of the thenar eminence in severe cases

**History:** Pain in wrist or forearm region, particularly when rowing at high stroke rates, or in cold or bad weather conditions; often, pain will lead to inability to appropriately feather or square and feeling of burning or swelling.

**Physical examination and treatment:** Pain and tenderness over affected muscles, with additional discomfort on resisted muscle testing; diagnosis of exertional compartment syndrome may require examination for pain and compartment tightness (and measurement of compartment pressures) immediately after exercise because there may be few physical findings at rest. If feathering action causes pain, advise the athlete to row "on the square" or on an ergometer. Cortisone injection only when conservative treatment fails; steroid injection (0.5–1 mL) into tendon sheath (for tenosynovitis) or into insertion of wrist extensors onto lateral epicondyle (for lateral epicondylitis), with return to rowing in 1–3 days; surgical intervention required only in more severe cases (i.e., fasciotomy for exertional compartment syndrome)

**Technical changes:** Encourage relaxed grip without excessive wrist motion. Ensure appropriate grip size/material. Advise the use of pogies (fleece or similar material coverings for hands and oar handle) in cold weather.

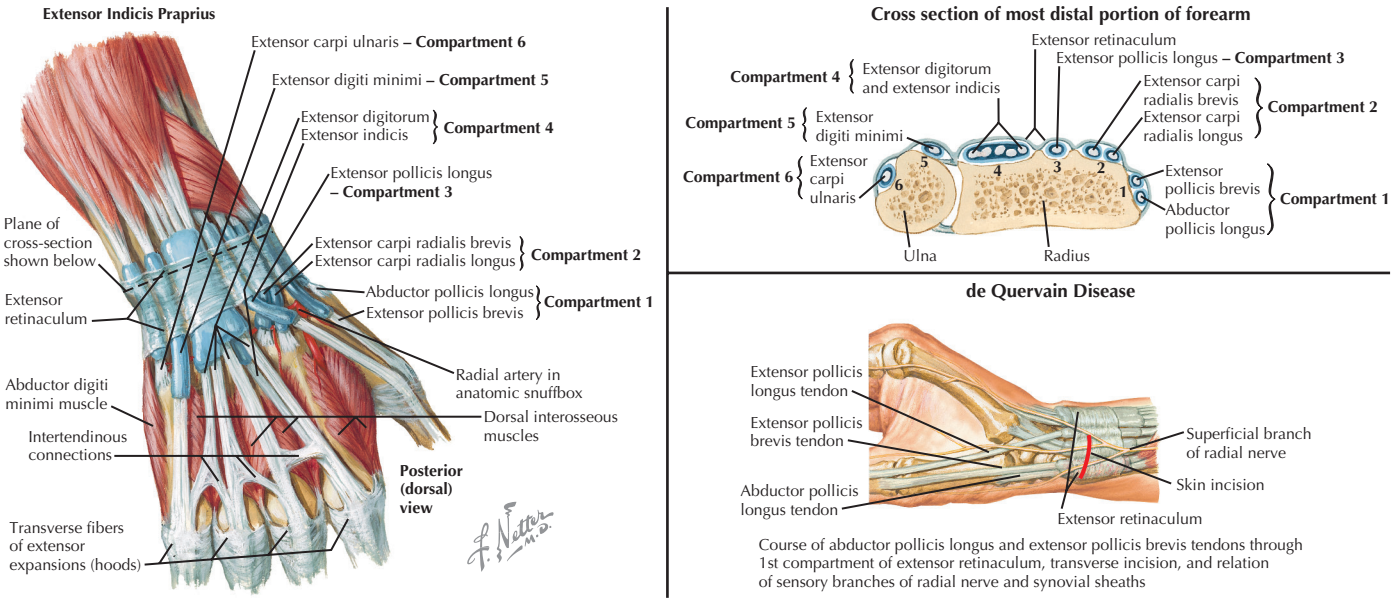



**Figure 85.6.** Wrist and forearm injuries.

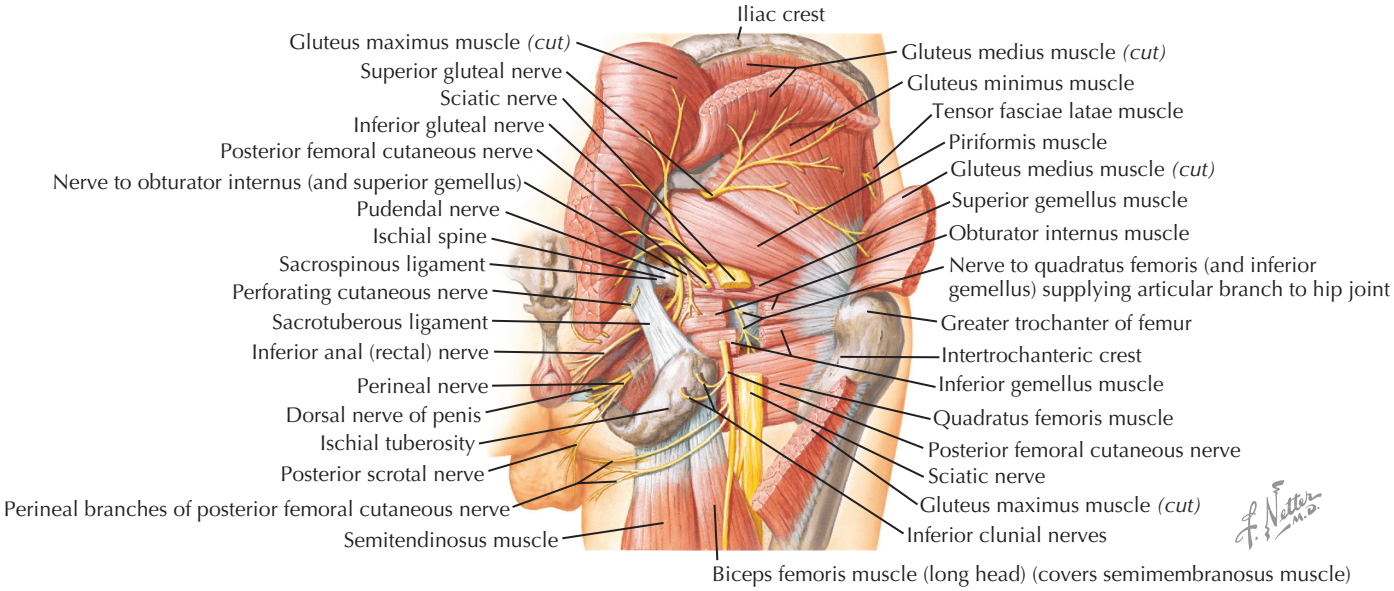



**Figure 85.7.** Sciatic nerve compression.

## Sciatic Nerve Compression

**Mechanism of injury:** In general, rowers' seats are made of wood or carbon fiber, specifically molded to accommodate ischial tuberosities; however, mold is usually generic and may not fit a particular rower's pelvic width, leading to constant compression of sciatic nerve. Different seat sizes are now available.

**History:** Numbness radiating down legs, only when rowing, without back pain or leg weakness

**Physical examination:** As standard, rule out radiculopathy, disc disease, and piriformis syndrome (compression of sciatic nerve by tight piriformis muscle) (Fig. 85.7)

**Technical change:** Change seats or use seat pads. Elevate or lower shoes in boat.

## Hand Blisters and Skin Abrasions

**Mechanism of injury:** Most severe upon resumption of on-water training; blisters and abrasions can occur at any of the three points of contact: hands/oar, buttocks/seat, and feet/shoes; imperative to prevent secondary infection or scarring

**Types of injury:**

- **Hand blisters:** Found on the anterior aspect of fingers and palm; caused by repetitive friction between skin and oar handle; usually only painful when rower resumes on-water training, but can be affected by heat, humidity, and change in grip size or material; may lead to infection (Fig. 85.8A)
- **Sculler's knuckles:** Superficial abrasions on dorsal aspect of right hand, caused by crossover of port and starboard handles

**A.** Blisters.

**B.** Crossover in sculling. (Courtesy of C. Lebrun.)

**C.** Track bites. (Courtesy of C. Lebrun.)

**Figure 85.8.** Hand blisters and skin abrasions.

mid-drive or mid-recovery; can cause significant bleeding and pain while rowing (Fig. 85.8B)
- **Track bites:** Superficial abrasions on posterior aspect of lower leg, caused by tracks for sliding seat digging into calf muscles; can lead to scarring (Fig. 85.8C)
- **Rower's rump:** Term coined for rowing-associated lichen simplex chronicus; uncommon, but several rowers have superficial abrasions on buttocks, ranging from slight indentations to skin ulcerations, secondary to repetitive chafing caused by inappropriately fitted seats

**History:** Recent resumption of on-water training, increase in training volume, or sudden shift in weather or equipment (sweep to sculling or grip change)

**Physical examination and treatment:** Standard; monitor closely for signs of secondary infection (erythema, fever, and red streaking along lymphatic drainage route)

**Technical changes:**
- **Hand blisters:** Change grip material and/or size of handles; several manufacturers offer a variety of grip material (wood, hard plastic, foam, etc.); in most cases, some blistering is unavoidable. Ensure scrubbing of oar handles after each use if shared among crew. Case study of spread of hand warts caused by oar sharing.
- **Track bites:** Use circumferential tape; consider moving tracks wider or toward bow of boat if possible.
- **Sculler's knuckles:** Oarlock height can be modified to increase gap between port and starboard oar handles. Circumferential tape may help in the short term; extended use of gloves not advised because they may compromise proprioceptive feedback
- **Rower's rump:** Regular application of corticosteroids in severe cases; in most cases, change seats or use seat pads

## Energy Availability, Body Composition Issues, and Disordered Eating

- Rowers, particularly lightweights and coxswains, may restrict calorie intake to the point of disordered eating; may be inadvertent
- In females, resultant inadequate energy intake for energy needs can lead to menstrual dysfunction and altered bone mineral density (female athlete triad).
- Underlying cause is overall energy imbalance—Relative Energy Deficiency in Sport (RED-S): this condition can also affect males and their reproductive hormones.
- This can result in lowered bone density and other musculoskeletal, cardiovascular, and electrolyte abnormalities, affecting performance.
- Prevent through education, awareness, nutritional counseling, and appropriate selection of athlete's natural weight
- Need for health care professionals to be aware of energy imbalance/deficiency, disordered eating behaviors, menstrual dysfunction, and other hormonal abnormalities and potential for altered bone mineral density

## ENVIRONMENTAL EXPOSURE AND SAFETY CONSIDERATIONS

**Personnel:** Coaches should always accompany or be in sight of rowing shells, with appropriate numbers of personal flotation devices (PFDs) and communication devices to call for outside help, if required. Collisions may occur due to rowers facing backward and not checking the course enough. Rowers should be advised never to attempt to swim away from a swamped shell (even if shore is close by). If rescue is not imminent, rowers should attempt to get as much of their bodies on top of an overturned shell to reduce heat loss.

**Storms and wind conditions:** High winds and choppy water can lead to swamping of boats because rowing shells sit very low in water. No boating of any kind should be attempted during a thunderstorm.

**Sun exposure:** Athletes and coaches should wear water-/sweat-proof sunscreen, sunglasses, and hats during prolonged sun exposure.

**Cold exposure:** Rowing in cold temperatures places rower at a risk of excessive heat loss. Rowers should dress in layers of synthetic clothing, avoid splashing with oars, and wear fleece pogies and hats.

**Water conditions:** Whitecaps signify high winds and should be avoided. For rowers who train on rivers, currents and eddies may be dangerous, particularly during flood conditions.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# MARTIAL ARTS

# 86

Robert Virgil Masocol • Irfan Asif • W. Franklin Sease Jr. • Bryant Walrod • Steven Erickson

## INTRODUCTION

- Martial arts are bodies of codified practices or traditions of training for unarmed and armed combat, usually without the use of guns and other modern weapons.
- People study martial arts for various reasons, including improved fitness, self-realization (meditation), mental/character development, and self-defense.

## EPIDEMIOLOGY

- There are ≥140 major and minor martial arts styles practiced worldwide.
- Participants
  - Approximately 2–8 million participants in the United States
  - The male-to-female ratio is 5:1.
- Injuries
  - Over 35,000 visits to hospital emergency rooms for injuries related to marital arts in 2011
  - The actual incidence of injuries remains unknown because of a lack of reporting of injuries and lack of studies regarding the sport. However, it is thought to be low compared with other sports since most of the instructions and training is noncompetitive and noncontact.
- Fatalities
  - A previous study reported six deaths during an 18-year surveillance of multiple disciplines of martial arts injuries: four occurred after trauma to the head (one strike, one kick, and two falls), one after trauma to the neck, and one after trauma to the chest.

## FACTORS AFFECTING INCIDENCE AND PREVALENCE OF INJURY

### Form of Participation in the Martial Arts

- Many participants train several times per week, year round, without a natural break in training for rehabilitation/recovery.
- Emphasis of training is often on personal development, both physical and mental, and not on competition.
- Muscle mass and strength are typically not as important as speed, strategy, technique, mental discipline, and flexibility.
- Individuals of different levels of training may "spar" (to engage in practice competition with another individual to simulate the actual competition or bout) in practice, exploiting the mismatch in skill levels between participants.
- Tae kwon do
  - Three times the injury rate and a four-fold risk of multiple injuries compared with Shotokan karate
  - More severe injuries and a greater incidence of multiple injuries than other disciplines
- Incidence of injury is directly related to the amount of time spent in full-contact sparring, limited-contact sparring, or competition.
- A competition setting may also influence the injury rate and severity.
- Types of martial arts training include:
  - Kata—detailed choreographed patterns of movements/techniques used by practitioners to practice on their own
  - Basic hand strikes
  - Basic kicking
  - Strength training
  - Conditioning
  - Stretching
  - Flexibility
  - Breaking—using striking techniques to break boards or bricks
  - One-step sparring—noncontact sparring during which students practice techniques to be used during free sparring or a competition
  - Grappling
  - Ground fighting
  - Joint locks—grappling technique involving the manipulation of an opponent's joint in such a way that the joint reaches a maximal degree of motion in order to induce submission or to injure an opponent
  - Chokes—employed via various mechanisms using the upper or lower extremities to temporarily disrupt the vascular supply to the brain or compress the trachea to prevent breathing
  - Free sparring—to engage in a practice competition with another individual to simulate the actual competition or bout
  - Point-scoring competition
  - Full-contact competition

### Protective Equipment

**Mouth guard:** Risk of orofacial injury is 1.6–1.8-times greater when mouth guards are not worn. Use does not reduce the risk of concussion.

**Headgear:** May reduce peak acceleration forces to the head; different brands have varying safety profiles (Fig. 86.1)

**Hand and foot protection:** Hand protection (as used in tae kwon do) and foot-padding gear have not been shown to reduce peak acceleration; combination of headgear and hand/foot protection superior to either used alone (see Fig. 86.1). Padding may lead to decreased inhibition and poorer control of striking, which may lead to a greater number of blows with a larger amount of force. Hand and foot padding are thought to decrease the amount of superficial injuries, such as lacerations and abrasions, to both the attacker and the defender. Equipment varies with different martial arts.

**Padded flooring:** Padded flooring may reduce the intensity of a blow by absorbing some of the impact of falls and throws (see Fig. 86.1). The surface must be closely monitored for risk factors for fall, such as moisture (water, blood, or perspiration) or gaps between the padding. Mat pads are common reservoirs for fungi and bacteria and therefore must be cared for in a similar manner to wrestling mats in order to prevent the spread of infections (e.g., community-acquired methicillin-resistant *Staphylococcus aureus* or tinea corporis).

### Age

- Age is not a reliable predictor of the likelihood of injury, although one study reported decreased incidence of injury in adolescent karate practitioners during elite competitions.

Head gear.

16 oz boxing gloves.

MMA style gloves.

Padded flooring.

**Figure 86.1.** Protective equipment.

## Experience

- Experience and number of hours of participation appear to be associated with higher rates and severity of injury.
- A previous study reported a greater incidence of head injuries and fractures in professional Muay Thai kickboxers when comparing amateurs to beginners.
- Individuals with at least 3 years of experience were at twice the risk of injury than less-experienced individuals.
- The number of tournaments and months of practice are directly and significantly associated with the likelihood of injury.

## Setting

- Tournament and competitive situations are associated with a lower absolute number of injuries but higher rate of severity compared with noncompetitive situations, possibly secondary to increased aggression in tournament settings.
- Informal training sessions are associated with a higher risk and severity of injuries compared with more formal supervised instructions.

## Sex

- Males have higher rates and severity of injuries compared with females, probably related to increased aggressiveness, except in karate, where females have a higher injury rate than do males.

## Weight Class

- Muay Thai kickboxing participants who compete at a heavier weight are at a higher risk of injury.
- In competition, governing bodies of martial arts have established weight classes. Physicians must be aware of their athletes that are attempting to make different weight classes and provide appropriate counseling for weight maintenance.

## Prevention

- The American Academy of Orthopedic Surgeons (AAOS) offers these tips for martial arts participants to safely train and compete:
  - Consult with a physician before beginning your conditioning to establish your readiness.
  - Train under the direction of a martial arts instructor who focuses on form and technique rather than competitive strategy.
  - Wear appropriate protective gear for your type of activity: e.g., tae kwon do, as a full contact sport, requires a head guard, a body protector, forearm and shin guards, and a groin guard.
  - Exercises that strengthen rotator cuff muscles and hip adductors/abductors are critical to martial arts for injury prevention, balance, and improved striking ability.
  - Maintain appropriate breathing techniques when practicing martial arts to avoid injury—breathing out during the contraction portion of any stretching movement, and breathing in during the extension portion of any stretching movement.

## Coverage of Martial Arts Events

- General
  - Attending physician must be present when the rules of the competition are reviewed.
  - It is important to identify yourself and briefly review certain medical considerations.
- Prefight examinations
  - Perform in a quiet, well-lit environment.
  - Enquire about previous and/or recent concussions and "knockouts."
- Match stoppage
  - Typically done by the referee; however, the referee may consult with the physician for medical guidance regarding whether the match should continue.
  - Typically, intervention will be required for lacerations and bleeding.
- Cervical spine injuries
  - Often teammates or coaches will want to attend to their fighter, and they may inappropriately move a fighter with an unstable cervical neck injury.
  - Review the rationale and need for appropriate cervical spine immobilization with the entire group of participants and their team.
- Postfight examinations
  - After a loss or a knockout, the participant may be confused, belligerent, or emotional.
  - It is important to maintain control of the situation to complete an appropriate examination.
- Medical kit suggestions: disposable gloves, gauze, silver nitrate, band aids, suture kits, sling, athletic tape, Coban, nonsteroidal anti-inflammatory drugs (NSAIDs), cold/flu/allergy medication, antidiarrheal medication, shears, petroleum jelly, nasal plugs, bandages, otoscope/ophthalmoscope, tooth saver solution, syringes, needles, lidocaine with/without epinephrine, alcohol swabs, betadine, contact and eye wash solution, splinting material, betadine, oral airway, cervical collar, Epipen, and albuterol

## Martial Arts–Specific Injury Considerations

- It is critically important to understand the unique aspects of each martial art discipline in order to best predict potential injuries.
- Certain martial arts disciplines focus more on contact and sparring, whereas others focus more on technique.
- When covering a martial arts competition or evaluating a martial artist, familiarize yourself with the techniques, emphasis, equipment, scoring, and target areas of each discipline in order to anticipate various possible injuries.

# COMMON TECHNIQUES IN MARTIAL ARTS

## Hand Strikes

**Punching:** Striking an opponent with a closed fist (Fig. 86.2). Appropriate punching technique places the wrist in slight volar flexion, with the second and third metacarpals aligned with the long bones of the forearm. Contact is made with the second

**Figure 86.2.** Common techniques in martial arts.

and third metacarpal heads. A "boxer's fracture" of the fifth metacarpal typically results from poor punching technique, wherein an individual makes contact with the fifth metacarpal head instead of the second and third metacarpal heads. Common injuries from poor punching technique include phalanx fractures, fourth or fifth metacarpal fractures, wrist sprains, extensor tendon injuries, and first metacarpal phalangeal ulnar collateral sprains.

**Knife hand chop ("karate chop"):** A strike with an open hand during which contact is made with the ulnar aspect of the fifth metacarpal head

**Ridge hand (reverse knife hand chop):** A strike during which the thumb is tucked into the palm and contact is made with the radial aspect of the second metacarpal head

**Spear thrust:** Open hand technique during which contact is made with the fingertips of the second, third, and fourth fingers, most commonly targeting the eyes and throat

**Hammer fist:** Closed-hand strike with the ulnar aspect of the fist

**Spinning back fist:** The attacker swivels 360 degrees and strikes the opponent with dorsum of the hand and second and third metacarpophalangeal (MCP) joints, employing great power and momentum.

## Foot Strikes

**Front snap kick:** From a standing position, the hip is flexed up to bring the femur parallel to the floor (see Fig. 86.2). The leg is then extended, resulting in the ball of the foot making contact with the defender's abdomen. Note: If the target is the groin, the foot is plantar flexed and the point of contact is the dorsal aspect of the proximal first metatarsal. If the target is the face, the ankle is dorsiflexed and contact is made with the plantar aspect of the heel.

**Side kick:** Delivered sideways relative to the position of the person executing the kick; contact is made with the heel targeting the abdomen or face (see Fig. 86.2).

**Back kick ("donkey kick," "mule kick," or "spinning back kick"):** Kick is delivered backward, keeping the kicking leg close to the standing leg and striking with the heel. Most often, it is delivered with a spinning motion generating great power.

**Roundhouse kick:** From the position of the hip flexed up to 90 degrees, the attacker swings his or her lower leg up in a circular motion, striking with the dorsal aspect of the proximal first metatarsal (see Fig. 86.2).

**Miscellaneous:** Certain disciplines of martial arts encourage kicking with the shin opposed to the ball of the foot or instep to reduce the likelihood of injury.

## Chokeholds

**Rear naked choke:** Attacker approaches opponent from behind, wrapping his or her arm around the opponent's neck and then grasping the biceps of his or her other arm. Then, using his or her free hand, the attacker forces the head of the opponent into flexion, resulting in compression of the carotid arteries, temporarily rendering the opponent unconscious (see Fig. 86.2).

**Triangle choke:** Attacker is on his or her back (guard position) and wraps one leg around the neck and shoulder of the opponent with their knee next to the opponent's neck, the other leg crosses the ankle of the first leg, using the foot of the first leg to lock the second leg into position at the knee (see Fig. 86.2).

**Guillotine choke:** Applied from a standing position or the guard; the attacker faces the opponent and wraps an arm around the opponent's neck so that his or her humerus is on the dorsal aspect of the neck and their forearm wraps around anteriorly to apply pressure to disrupt the vascular supply to the brain, temporarily rendering the opponent unconscious, or to compress the trachea in order to restrict breathing (see Fig. 86.2)

### Joint Locks/Manipulation

**Arm bar:** A joint lock that hyperextends the elbow joint by placing the opponent's extended arm over a fulcrum such as an arm, leg, or hip (see Fig. 86.2). The opponent is controlled in this position, and if he or she does not tap out, continued force will result in dislocation of the elbow.

**Leg lock:** A joint lock that is directed at the joints of the leg such as the knee or ankle. A kneebar is similar to an armbar, which aims to forcefully hyperextend the knee of the opponent (see Fig. 86.2).

**Ankle lock:** A submission applied to the opponent wherein the attacker uses his or her arm to secure the opponent's ankle in his or her armpit. Once the ankle is secured, the attacker leverages his or her hip forward, which forcefully plantar flexes the ankle. The forearm serves as a fulcrum in leveraging and may cause severe pressure on the Achilles tendon.

**Miscellaneous:** Small joint manipulation is typically prohibited.

## TYPES OF INJURIES

**Contusions:** Overall, the most common injury; orbital contusions can obstruct vision. Cold steel or ice can be employed between rounds to reduce edema.

**Sprains/strains:** Overall, the second most common injury; most commonly sprained joints include ankles and knees; hamstring and groin strains common from inappropriate stretching. Aikido has a higher incidence of sprains and strains than Shotokan karate.

**Abrasions/lacerations:** Laceration management: direct pressure, petroleum jelly, clotting agents such as silver nitrate or a vasoconstrictive agent; a physician may only examine a laceration during a fight to determine whether the participant may continue. The physician may not tend to or treat the laceration until after the round. Do not suture the laceration until after the competition. A competition should be stopped if the laceration bleeds to the extent that it will obstruct the vision of either competitor.

**Epistaxis:** Exceedingly rare to stop a competition for epistaxis. Direct pressure and petroleum jelly work well. Monitor for bleeding into either competitor's eyes such that it may obstruct vision. Careful examination to exclude septal hematoma or nasal fracture

**Fractures:** More common in experienced martial artists; finger and toe fractures common due to inappropriate kicking and striking techniques; rib fractures common from being kicked

**Dislocations:** Various digit dislocations can be immediately reduced with rapid return to competition. Postreduction radiography is recommended.

**Mild traumatic brain injury (MTBI):** Of severe injuries, concussion is the most common one. Increased rate of concussion in younger participants, in those who demonstrate poor blocking skills, and in association with receiving a kick to the head; decreased opportunities for head trauma in martial arts that emphasize grappling and joint locks over striking; individuals who regularly participate in full contact sparring should undergo baseline neurocognitive testing for appropriate management of mild traumatic brain injury.

## LOCATION OF INJURIES

**Lower extremity:** Instep of the foot is the most common lower extremity location of injury in tae kwon do. Common foot and ankle injuries are bruising, lacerations, ankle sprains, and digital fractures. Receiving and delivering a kick are the most common mechanisms associated with a lower extremity injury in tae kwon do. A roundhouse kick is the most common type of kick to produce a lower extremity injury. Hamstring and adductor strains can occur secondary to overstretching or inappropriate stretching.

**Upper extremity:** A unique injury with submission techniques is an arm bar, wherein the anterior capsule of the elbow is stretched and may result in complete rupture of the capsule and ligaments if continued force is applied and the participant does not tap out. An arm bar hyperextends an already completely extended elbow, stretching the surrounding capsule. If the elbow is rapidly extended, the capsule may tear and the elbow may dislocate. Moreover, an arm bar may result in a fracture of the humerus, potentially causing damage to the radial nerve. Treatment of a simple arm bar with stretching of the capsule and anterior elbow pain consists of ice, NSAIDs, and a sling for comfort (but be sure not to immobilize the joint to prevent stiffness). Complex arm bar injuries consisting of fractures and dislocations almost always require orthopedic evaluation for cartilaginous, ligamentous, or nerve injury. Mixed martial arts (MMA) size gloves (4–6 ounces) are typically worn to prevent hand injuries to the individual wearing the gloves. Injuries that occur from striking or kicking rather than from being struck are typically caused by poor technique.

**Head/face/neck:** These injuries are the most severe. There is an increased frequency of head injuries in martial arts disciplines that emphasize striking and head contact; clear similarities in the force, kinematics, and biomechanics required to produce cervical neck injuries in motor-vehicle accidents and common MMA maneuvers

## MARTIAL ARTS IN THE OLYMPIC GAMES

### Judo ("Gentle Way")

- Founder: Jigoro Kano
- Country of origin: Japan
- Emphasis
  - Flexible or efficient use of balance, leverage, and movement in the performance of throws or techniques
  - Skill, timing, and techniques are emphasized over brute strength.
  - Soft method in which there is an indirect application of force applied to defeat an opponent
- Techniques
  - Throwing
    - Most throws are performed in a standing position.
    - Forceful throws are used in an attempt to render an opponent unconscious.
    - Increased points are given depending on the severity of the throw.
  - Strikes (kicks and punches) are practiced, but they are not permitted in competition.
  - Pinning
    - Attempt to hold an opponent to the mat
    - Pins of 25 seconds will win the match.
    - Pins of 10–25 seconds will score points.
    - Significant pressure is applied to the opponent with pain-generating techniques.

    - Certain body parts, particularly the ribs, are vulnerable for injury during these techniques.
  - Joint manipulation
    - Only elbow joint locks are permitted.
    - Leg locks, wrist locks, and spinal locks are not permitted.
  - Chokes (permitted depending on the age of the participants)
- Equipment
  - Judogi, "gi," meeting certain specifications and appropriately worn
  - A gi is a Japanese name for a traditional uniform used in judo for practice and competition.
    - Consists of two parts: a heavy fabric uwagi (jacket) and a lighter fabric zubon (pants)
    - Garments are typically loose fitting and long sleeved.
- Medical coverage
  - Medical team not allowed on mat unless granted by referee; only one medical person on the mat
  - Bleeding: Same bleeding injury may be treated on two occasions. Third time of the same bleeding injury will be a medical disqualification

### Tae Kwon Do ("The Way of the Feet and Fist")

- Founder: Unknown
- Country of origin: Korea
- Emphasis
  - Kicking techniques are emphasized because the leg is the longest and strongest limb in the body and thus has the potential to deliver the most forceful blow, rendering one's opponent unable to retaliate.
  - Union of physical and mental strength in such techniques as breaking of boards
- Techniques
  - Hand techniques
    - Legal target areas: Front of the torso; this is restricted to the front of the body, starting at the hipline and going up to the base of the throat from one side seam of the uniform to the other side seam
    - Legal hand techniques include typical punches and strikes.
    - Spinning back fists and finger strikes and other "blind techniques" are considered illegal.
  - Feet techniques
    - Legal target areas, front of torso as discussed above; the sides and back of the neck are legal as are all areas of the head including the face, mask, the sides, and the top.
    - One may only strike from below the ankle, no shin or knee striking.
    - Hand and standing-foot techniques to the legal torso area will score one point.
    - Standing kicks to the head, face, and neck will score one point, whereas a jumping kick to the body will also score two points.
    - A jumping kick to the head will score three points.
  - Breaking
    - Typically use hand or foot but may also use fingertip, toe, head, elbow, knuckle, or knee
    - It is postulated that the skeletal system, after being exposed to stress, will be stronger after it heals. Thus, many martial artists who practice breaking will continually stress their skeletal system and allow it to heal, which subsequently becomes stronger.
    - Many train to strengthen their fists by practicing bare knuckle push-ups, initially on a soft surface such as a carpet and then progressing to a harder surface such as concrete.
- Equipment
  - White American Tae Kwon Do (ATA) uniform
  - Mouthpiece
  - Protective cup
  - Headgear
  - Hand pads that cover the entire length of the fingers
  - Foot pads that cover the entire length of the toes
  - Chest protector
- Medical coverage
  - Medical director is appointed for international competitions
  - Team physician needs to get accreditation by attending the meeting before competition. Please see World Taekwondo Federation (WTF) medical code for further details.

## MARTIAL ART DISCIPLINES THAT FOCUS ON TECHNIQUE AND FORM

### Aikido ("The Way of Harmonious Spirit")

- Founder: Morihei Ueshiba
- Country of origin: Japan
- Emphasis
  - Joining with an attacker and redirecting the attacker's energy
  - Moving together rather than clashing
- Techniques
  - Body throws
  - Joint locks
  - Strong strikes or immobilizing grabs are employed but not emphasized.

### Jujitsu ("Art of Gentleness")

- Founder: Unknown
- Country of origin: Japan
- Emphasis
  - The art of breaking balance
  - Yield force provided in an opponent's attack in order to apply a counter technique
- Techniques
  - Strikes and kicks
  - Throws—perfect throws score more points than a throw that is strong but not perfect as deemed by the judges
  - Chokes
  - Pins
  - Joint manipulation

### Tai Chi Chuan ("Supreme Ultimate Boxing")

- Founder: Disputed—however, origins can be traced to the Chen and Yang families
- Country of origin: China
- Emphasis:
  - Receptive "ying"—slow, repetitive, meditative, slow impact
  - Active "yang"—active, fast, high impact
  - Health—relieve the physical effects of stress on the body and mind
  - Meditation—cultivate focus and calmness to maintain homeostasis and relieve stress
  - Martial art—change in response to outside forces
  - Do not resist a violent force; instead, meet the force and redirect it.
- Techniques
  - Solo form: A slow sequence of movements that emphasize a straight spine, abdominal breathing, and a natural range of motion
  - Pushing hands for training in a practical form
  - Pushes and open hand strikes are more common than punches.
  - Kicks are usually to the leg and torso, never higher than the hip.
  - Fingers, fists, palms, forearms, elbows, knees, and feet are used to strike the eyes, throat, heart, and groin.
  - Joint locks are also used.

## MARTIAL ARTS DISCIPLINES IN ADDITION TO JUDO AND TAE KWON DO THAT FOCUS ON SPARRING AND CONTACT

### Karate ("Empty Hand")

- Founder: Tode Sakugawa or Gichin Funakoshi
- County of origin: Possibly Ryukyu Kingdom of Japan
- There is a push to make this an Olympic sport.
- Emphasis
  - Striking art
  - Forms or techniques that demonstrate combat principles
  - Techniques must be performed with excellent control and good form.
  - Injuring an opponent may result in point deduction.
  - Light contact, but not full contact, is permitted and emphasized.
- Techniques
  - Kicks: Kicks to the head and neck score more points than kicks to the body.
  - Punches: Punches to the back and back of the head score more points than punches to the head and trunk.
- Competition types
  - Kumite: Sparring against an opponent and based on points as described in the techniques section
  - Kata: Detailed choreographed patterns of movements practiced either solo or in team

### Brazilian Jiu Jitsu

- Founder: Carlos Gracie
- Country of origin: Brazil
- Emphasis:
  - Ground fighting
  - Grappling
  - Leveraging opponents into a position of submission
- Techniques
  - Submission holds including joint locks and choke holds
  - Guard position—an advantageous grappling position wherein one opponent has his or her back to the ground and controls the other opponent with gripping and his or her legs
  - Side control—controlling an opponent on the ground from the side of the body
  - Full mount—controlling an opponent from the top while sitting on their chest; most dominant position
- Competition types: Gi (with kimono) and No-Gi (inability to use clothing as leverage)

### Kickboxing

- Founder: Unknown; the term was created by Japanese boxing promoter Osamu Noguchi as a variant of Muay Thai and karate
- Emphasis
  - Strikes with punches and kicks while standing up
  - Ground techniques and grappling are *not* permitted.
- Techniques
  - Strikes above the hip with fists and feet
  - Using elbows and knees is forbidden, and use of shins is seldom permitted.
  - Spinning back-of-the-fist punches are permitted as well as traditional punches.

### Tang So Do ("Way of the Chinese Fist")

- Founder: Hwang Kee
- Country of origin: Korea
- Emphasis: Empty hand-and-foot fighting forms with an emphasis on the development of discipline, self-confidence, and self-defense
- Techniques
  - Emphasis is placed on kicking techniques, particularly turning and spinning kicks to the torso and head of the opponent.
  - Katas are used as a method of training for practitioners away from the dojo.
  - Techniques drawn from ancient Chinese and Japanese martial art traditions

### Thai Boxing (Muay Thai) ("The Art of Eight Limbs")

- Founder: Unknown—traced to Buddhist Monks
- Country: Thailand
- Emphasis: A distinguished art of fighting of attrition that employs strikes with fists, elbows, shins, and knees
- Techniques
  - Strikes: Fists, elbows, shins, feet, and knees are all permitted to strike an opponent.
  - Punching techniques score lesser than other striking techniques.
  - Emphasize entire body movements and hip rotation with strikes to increase strength
  - Core body strength is essential.
  - Kicking techniques are encouraged to connect with the shin.
  - Grappling or clinching (Thai clinch): A clinch is not broken up as in traditional boxing because this can be an advantageous position from which knee strikes can be employed.

### Krav Maga ("Contact Combat")

- Founder: Imi Lichenfeld
- Country of origin: Israel
- Emphasis
  - Counterattacking as soon as possible
  - Targeting attacks to the body's most vulnerable points
  - Maximum effectiveness and efficiency in order to neutralize opponent by any means
- Techniques
  - Kicks, punches, gouges, elbows, and headbutts
  - Throws
  - Joint manipulation—less emphasized

### Mixed Martial Arts (MMA)

- Combat sport in which participants use a multidisciplinary approach to fighting that includes jujitsu, judo, boxing, wrestling, karate, kickboxing, and other fighting disciplines in a supervised bout between two individuals (see Chapter 89, Mixed Martial Arts).

## SUMMARY

- Incidence of martial arts injuries varies with the quality of instruction and the quantity and type of combative training practiced by the participant.
- Injury rates greatly vary, depending on style of martial arts practiced, age, sex, and experience level of the participant.
- Injuries are very uncommon during practice sessions that primarily involve teaching and/or practicing techniques that do not involve full-speed contact.
- There are millions of martial artists practicing in the United States who do not compete or spar and therefore have a very low incidence of injury.

- Participation in martial arts training continues to grow, partly because of the emphasis on personal growth and improved self-confidence as well as physical development and self-defense.
- Martial artists should be encouraged to use protective gear whenever possible because the use of protective gear decreases the incidence of lacerations, contusions, and abrasions and may decrease the peak acceleration forces to the head during kicks and strikes.
- Sports medicine providers should familiarize themselves with the rules and practices of different styles of martial arts as well as the terminology used to describe the types of training and the various kicks, strikes, and attacks used by martial artists.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 87 BOXING

Brian A. Jacobs

## HISTORY

- Boxing is one of the most ancient sports.
- Marquis of Queensbury modified the rules in the early 1800s.
- The 1904 Olympic Games in St. Louis saw men's boxing introduced as a competition sport and women's boxing as an exhibition sport.

## AMATEUR BOXING

- Divided by age class (Box 87.1)
- Weight classes (Table 87.1)
- Experience and skills of boxers: details can be found at www.usaboxing.org.

## INJURY PREVENTION

### Equipment

Headgear: Headgear properly fitted by trained coaches is required for amateur competition. Fitted headgear reduces eye injuries, facial fractures, lacerations, "cauliflower ear," and tympanic membrane perforations.

Gloves: Gloves, thumbless and those with thumbs attached, reduce both eye and thumb injuries. Gloves with mobile thumbs are not authorized for use. Heavier gloves have decreased hand injuries and reduce impact forces. Two-toned gloves are easier to see for scorekeepers and referees. Gloves must remain clean and as close to "like new" condition as possible.

Hand wrap: The hands must be wrapped under the supervision of an appropriate official. Wrapping prevents injury to the hands and further reduces the force of blows delivered.

Mouth guard: Custom-fitted mouth guards prevent dental and temporomandibular joint injuries and are required for the competition. Mouthpieces are also employed to allow the athlete to "set" his or her jaw, reducing the likelihood of knockdowns by reducing the intensity of the blow.

Breast protectors: Breast protectors, when used, must be well fitted and not interfere with the boxer's ability to fight. They should not extend beyond the clavicles or the xyphoid.

### Physical Examinations

- In boxing, an annual physical examination is required for each athlete, performed by a qualified physician. The examination certifies that the athlete is free from injury, disability, or infection, which could jeopardize either the boxer or opponents. The boxer must present, at the physical examination and at weigh-ins, an up-to-date USA Boxing Competition Record Book with correct information and sign-offs by either the Secretary General or the Executive Director of the boxer's national federation and is not allowed to compete without this document.
- A prefight physical is intended to reduce risk of injury by identifying medical problems prior to the competition and by monitoring recovery from previous injuries.

### Physician Approval, Qualification, and Disqualification

Athletes are required to have approval from their physician prior to competing.

#### Disqualifying Conditions

- Acute and chronic infections—examples
  - Illness causing fevers
  - Chest infection
  - Untreated tuberculosis
  - GI conditions with dehydration/malabsorption
  - Hepatitis
  - Open skin conditions; e.g., MRSA, zoster, herpes
  - Mononucleosis within the past month
- Severe blood dyscrasias, conditions requiring anticoagulation, sickle cell disease
- Infection with HBV, HCV, or HIV
- Refractive or intraocular surgery, cataracts, retinal detachment
- Presence of myopia > 3.50 diopters, uncorrected vision worse than 20/200, or corrected vision worse than 20/60
- Significant cardiovascular or pulmonary abnormalities, including
  - Severe COPD, uncontrolled asthma with potential for hypoxemia
  - Severe aortic or pulmonic stenosis, myocarditis, pericarditis, recent embolic disease, 3rd degree heart block, atrial or ventricular tachycardia, coarctation of aorta, PDA, corrected surgical conditions unless cleared by cardiovascular surgery, resting BP >160/100 (if >140/90, the boxer may compete if other BP measures are normal, but physician follow-up is advised for >135/85)
- Congenital/acquired musculoskeletal problems; e.g., spondylolysis, spinal fractures, atlantoaxial instability, unstable joints, loss of thumb or great toe
- Unresolved concussion
- Significant intracranial mass lesions or bleeding, history of craniotomy, cerebral palsy, hypoxic brain injuries, neuropathies causing balance/coordination problems (benign smaller problems can be cleared by neurosurgery)
- Seizure within the past 3 years
- Hepatosplenomegaly, splenomegaly, ascites
- Pregnancy
- Uncontrolled diabetes mellitus or thyroid disease
- Implantable devices interfering with physiologic process/enhancing performance
- Banned substances: In amateur and Olympic competitions, banned substances such as ergogenic aids and steroids are disqualifying if used. Refer to the appropriate authority (e.g., www.wada-ama.org) for comprehensive lists of banned substances, medications, and practices.

#### Nondisqualifying Conditions

- Deafness—judges/referees must be made aware of conditions, and the referee may tap the boxer on the shoulder to signal "break" or "stop."
- Dental braces/orthodontics if the Permission to Box with Braces or Orthodontic Appliances form is attached to the boxer's passbook.
- Breast implants if the Permission to Box with Breast Implants form is the attached to boxer's passbook
- Sex reassignment in accordance with IOC regulations
  - Prior to puberty: athlete regarded as gender assigned after surgery
  - After puberty: athletes are eligible to participate in new gender provided that

**BOX 87.1 AMATEUR BOXING AGE CLASSES***

- 8–14 years-Prep
  - 8–10 years-Peewee
  - 11–12 years-Bantam
  - 13–14 years-Intermediate
- 15–16 years-Junior
- 17–18 years-Youth
- 19–40 years-Elite and Senior
- 40–45 years-Senior/Elite or Masters

*Age group class determined by year of birth; i.e., age of boxer on December 31 determines age group.

**TABLE 87.1 AMATEUR BOXING WEIGHT CLASSES**

| | |
|---|---|
| **Males:** 10 weight categories in each Elite, Senior, and Youth category | Up to 46–49 kg, 52 kg, 56 kg, 60 kg, 64 kg, 69 kg, 75 kg, 81 kg, 91 kg, 91+ kg |
| **Females:** 10 weight categories in each Elite and Youth category | 45–48 kg, 51 kg, 54 kg, 57 kg, 60 kg, 64 kg, 69 kg, 75 kg, 81 kg, 81+ kg |
| **Elite Women Olympic Boxers**: 3 weight categories | 48–51 kg, 57–60 kg, 69–75 kg |
| **Boys and Girls:** Junior Boxers–13 weight categories | 44–46 kg, 48 kg, 50 kg, 52 kg, 54 kg, 57 kg, 60 kg, 63 kg, 66 kg, 70 kg, 75 kg, 80 kg, 80+ kg<br>Prep boxers—Peewee, Bantam, and Intermediate are all arranged similarly; details can be found at www.usaboxing.org |

- All surgical and anatomic changes are completed
- Legal recognition of new gender is conferred by proper authorities.
- Hormonal therapy appropriate for the assigned gender has been administered and verified for sufficient length of time to minimize gender-related advantages
- Eligibility begins no sooner than 2 years after gonadectomy.

## RINGSIDE PERSONNEL AND EQUIPMENT

### Coaches

- One coach and one assistant are allowed for each fighter.
- Must remain seated during each round
- Must not interact with fans or ringside officials
- Should have first aid supplies, two clean white towels, sterile gauze pads, sterile cotton, cotton swabs, ice, and bags for ice
  - First aid equipment does not include ammonia, ammonia inhalants, or smelling salts, which are banned.

### Cutman

- Manages bleeding injuries that could otherwise disqualify a boxer during a bout
- Amateur boxing prohibits the use of any medication to treat bleeding during a bout, but professional boxers are allowed any medication or topical treatment that the cutmen or trainers may have at their disposal.

**BOX 87.2 FEMALE BOXING RULE 101.9**

Female boxers are limited to participation under additional medical restrictions. If any of the following conditions exist, the athlete is not allowed to participate:

- Confirmed pregnancy; suspected pregnancy must be confirmed or denied before starting or continuing boxing
- Painful pelvic disease states, such as symptomatic endometriosis
- Abnormal vaginal bleeding of undetermined etiology
- Recent secondary amenorrhea of undetermined cause
- Recent breast bleeding
- Recently discovered breast masses
- Recent breast dysfunctions previously not present

From McCrory P. Cavum septum pellucidi—a reason to ban boxers? *Br J Sports Med.* 2002;36(3):157-161.

### Controlling Injury and Bleeding

- One minute is allotted to get the bleeding under control.
- Any swelling of the eye or face is managed by pressure applied with an enswell, which is an iced metal spatula applied to the hematoma that milks the blood to the surrounding tissues.
- The prevention of bleeding injuries can be accomplished through the use of petroleum jelly applied to the face.
- Medications—when allowed—to control bleeding include Avitene, Surgicel, Gelfoam, adrenaline chloride, and thrombin; these are frequently mixed with petroleum jelly and applied with cotton swabs.

### Physicians

- Serve as a neutral advocate for the boxers
- Responsible for the care and safety of fighters before, during, and after the match
- There are one or more physicians at ringside at all times during competition.
- Two physicians are preferable; this allows for one to attend to an injured boxer after the completion of that boxer's match, while the other physician attends to the current bout.

## COMPETITION

### Prefight

- All contestants should be thoroughly examined immediately before and after each bout.
- Female participants must now provide a signed Declaration of Non-Pregnancy, as noted in appendix G of the USA Boxing Official Rule Book (previously, signature of an explicit disclaimer stating that the boxer has read Rule 101.9 was required) (Box 87.2).
  - The waiver states the fighter is not pregnant to the best of her knowledge.
    - The team officials bear the responsibility of ensuring that females under 18 years old are certified not to be pregnant and to obtain proper documentation.
  - At the prefight physical, physicians are to note history regarding menstruation, pregnancy, and breast and gynecologic disease/surgical histories.

### Tournaments

- All fighters should be examined before each bout to check for new injuries that could impair the safety and ability of the boxer, precluding further participation.

### Disqualification During the Competition

- The following injuries result in disqualification from the bout:
  - Excessive swelling of the face or eyes that impairs vision
  - Suspected or proven fractures of nose, face, or metacarpals
  - Presence or history of a retinal detachment (permanent disqualifier)
  - Lacerations or wounds requiring dressings for control of bleeding

### Surveying the Venue

- The ringside physician must be familiar with the venue, the location of the training room, the location of the emergency medical system (EMS) providers and ambulances, the layout of the facility, the ring, the condition of the padding at the turnbuckles, and the condition of the ropes and mat.
- The physician should meet with EMS and first aid personnel and be aware of the location of first aid equipment, spine boards, oxygen, cervical collars, advanced cardiac life support equipment (including defibrillators, if available), and ensure adequate proximity to a phone or cellular connections.
- Physicians at ringside are mandated to have an unobstructed view of the ring and of the matches.

### Physicians' Medical Supplies at Ringside

- Physicians at amateur bouts are allowed the following at ringside:
  - Flashlight
  - Oral airway
  - Gloves
  - Vaseline
  - Adrenaline chloride (1:1000) to control nosebleeds and cuts
  - Thrombin for dry cuts
  - Avitene (microfiber collagen hemostat) for active bleeding
  - Ice and ice bags
  - Clean sponges
  - Gauze pads
  - Enswell pressure plates for control of hematomas
  - Two clean white towels
  - Scissors
  - Water in a clear plastic container
  - Two buckets, one containing ice and the other empty
  - Adhesive athletic tape
- Physicians at professional matches may have additional supplies as they deem necessary.

### Evaluation During the Bout

- When called into the ring, the physician should always:
  - Assume a cervical spine injury has occurred and stabilize the athlete as necessary
  - Abide by the "airway–breathing–circulation" principles of basic life support
  - Adhere to appropriate precautions when near blood or body fluids
  - Notify the athlete of his or her presence and title
  - Have athletes not requiring EMS-transfer return for re-evaluation

## THE BOUT

### Rounds and Standing Eight Counts

- Amateur bouts are characterized by up to four rounds of 2 to 3 minutes in length separated by a 1-minute rest interval.
- The referee can institute a standing eight count whenever it is believed that a fighter has been stunned.
- Should a fighter incur three standing eight counts in a given period or four in a given match, the fight is discontinued as referee stop contest–head (RSCH) injury.
- In the case of an unconscious boxer:
  - Referee signals the doctor to enter the ring.
  - Only the referee, physician at ringside, and any assistants the physician deems necessary are allowed into the ring.
- The referee can stop a fight for other reasons/injuries that may put a fighter at risk:
  - RSC (O)—Fighter is outclassed by opponent and is thus at unreasonable risk.
  - RSC (I)—Fighter is unable to compete because of an injury suffered during the bout.
    - Example: excessive swelling or bleeding that cannot be adequately controlled
- The injured fighter is referred to physicians for immediate evaluation after the bout.

### Scoring

- In amateur boxing, points are awarded for blows to the upper body and head regardless of the force applied and whether the struck contestant was knocked down or knocked out.
- Scores in professional boxing are also tabulated as points scored, but greater weight is given to blows of greater force, knockdowns, and knockouts. Subjective bias can play a role in scoring.

## THE REFEREE, THE PHYSICIAN, AND STOPPING FIGHTS

### Referee

- The referee serves as a safety official and operates in the best interests of the fighters in the contest:
  - Ensures that the rules of fair play are strictly observed
  - Is responsible for maintaining control of the athletes at all times
  - Is responsible for preventing unnecessary punishment of a weaker opponent
  - Ensures that the gloves, mouthpiece, and dress adhere to the appropriate regulations

### Ringside Physician

- The physician should pay close attention at all times to the match since doing so will allow for evaluation of injury mechanisms, fatigue, and other factors that contribute to injury.
  - If it is necessary for the physician to stop the match, the physician should be firm in doing so. Reasons for stopping a match include but are not limited to:
    - Cuts with arterial bleeding that cannot be controlled or that extend with further blows
    - Bleeding of the mouth, ear, or of the eyelid that obstructs vision
    - Any trauma that renders an athlete unable to defend him- or herself

### Stopping the Bout

- The ringside physician may, at any time, suspend a bout by mounting the ring apron but can enter the ring only at the behest of the referee.
- If the physician believes that a boxer is no longer capable of fighting, that a boxer is in danger of further injury, or that a boxer is outclassed, the physician can signal the referee to terminate the bout.
  - The referee can call Referee Stop Contest (RSC).

- If the fight is stopped because of concern of concussion or another head injury, the designation is RSCH, indicating that the fight has been stopped due to head injury.
  - The fighter will then be evaluated immediately after the fight has been declared RSCH.

## POSTBOUT EXAMINATION

- Restrictions affidavit: If a bout has ended by a knockout or RSCH, the injured boxer receives a restrictions affidavit requiring signatures of the athlete, the physician at ringside, the referee, and the coach.
- The boxer's coach is responsible for ensuring the boxer gets to appropriate accommodations safely after the fight.
- After any bout, it is imperative that the boxer's physician be contacted immediately if any of the following symptoms are present:
  - Headache or dizziness lasting for more than 2 hours
  - Increasing drowsiness or a decreasing level of or the lack of consciousness following a bout
  - Uncontrolled or persistent vomiting
  - Blurring of vision
  - Irrational behavior or persistent confusion
  - Seizure
  - Inability to move a limb
  - Agitation or excessive restlessness
  - Oozing of bloody or clear fluid from the nose or ears
  - Incontinence of bowel or bladder
- A medical survey is performed after the bout
  - All fighters should be assessed upon the completion of a contest, with special attention focused toward neurologic and orthopedic status.
  - All injuries suffered in a bout should be recorded appropriately.
  - Any future medical suspension that results from a bout should be noted on the athlete's fight card.

## RESTRICTION FROM BOXING

If a decision was rendered as RSC, the following parameters and restrictions apply:

RSC: No restrictions apply. Examples of this include matches in which fighters are clearly mismatched in skill (i.e., a fighter who is outclassed and taking an excessive number of body blows) or a fighter receiving an injury (e.g., a dislocated shoulder) that does not require RSC.

Single occurrence of knockout or RSCH: If a boxer suffers a knockout as a result of blows to the head or if the bout is stopped by the referee because the boxer received heavy blows to the head, the boxer may not box or spar for at least 28 days after the event.

Double occurrence of knockout or RSCH: If a boxer is knocked out twice or if two bouts have ended as the result of suffering heavy blows to the head or if one instance of each occurs within a 3-month period, then, the boxer may not box or spar for 3 months after the second occurrence.

Triple occurrence of knockout or RSCH: If during a 12-month period a boxer has been knocked out three times or has had three bouts stopped because of receiving heavy blows to the head, then the boxer may not box or spar for 1 year after the third occurrence. Any combination of knockouts or RSCHs that equal three under these circumstances also meets the threshold for the 1-year suspension.

Other: Any boxer who loses a difficult bout as a result of many blows to the head or who is knocked down in several successive competitions may be barred from boxing or sparring for 28 days after the last contest on the recommendation of the Medical Jury.

Medical certification after the end of the suspension period: After the suspension period is completed, the boxer must be passed as fit by a neurologist. Ideally, a specialist examination should be conducted, and CT or MRI scans of the brain should be performed.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Comparing Injury Rates to Those From Other Sports

- Boxers suffer injuries at rates comparable to and, in many cases, lower than, those found in other contact and noncontact sports (Tables 87.2, 87.3, 87.4, 87.5).

### Head and Brain Injury (See Chapter 45, Head Injuries)

- Rate of head injuries suffered in boxers ranges from 27% to 94% of all injuries.
- Most are minor, with contusions and lacerations being most common.

#### Concussion

- A study of 175 amateur college-aged fighters reported no findings of acute cognitive impairment.
  - Athletes were tested and evaluated pre- and postbout and participated in as many as four bouts in a 2-week period in some cases.
  - The athletes demonstrated improvements in performance of a learning speed task following bouts, suggesting practice effects caused by repeated test administration over a relatively short time—in spite of participation in the boxing tournament.

#### Intracerebral Hematoma

See Chapter 45: Head Injuries; Fig. 87.1.

#### Intracranial Epidural Hematoma

- The injured boxer may suffer an immediate brief alteration or loss of consciousness, followed by arousal and, potentially, even a normal level of consciousness (see Fig. 87.1), which is then followed by rapid deterioration in mental status with ipsilaterally dilated pupils (due to uncal herniation with compression of the oculomotor nerve), contralateral weakness, and progressive decline.

#### Subdural Hematoma

See Chapter 45: Head Injuries; Fig. 87.1.

#### Second Impact Syndrome

- Believed to occur when an athlete, who suffered an initial head injury, sustains a second head injury before the first injury has fully resolved
- The second injury may be relatively minor and even may occur in whiplash fashion.
- The injured boxer may appear initially stunned.
- Boxers usually remain conscious and may remain standing but then suddenly collapse and become comatose, presenting rapidly dilating pupils, a fixed gaze, and imminent respiratory failure.
- The condition is the result of rapidly developing brain edema with herniation of the brainstem; usually catastrophic and fatal
- Severity is suspected to be due to the loss of autoregulation of the brain's blood supply, leading to vascular engorgement within the cranium, increased intracerebral pressure, and uncal herniation.
- The usual time for brainstem failure appears to be between 2 and 5 minutes.

## TABLE 87.2 INJURY STUDIES IN BOXING

| | |
|---|---|
| **Amateur Boxing** | A study of 1,094 amateur fighters at the 1981 and 1982 USA/ABF championships observed:<br>• 85 injuries of varying degrees of severity, 52 were considered notable<br>• 48 matches were discontinued because of head blows, which occurred at a rate of 4.38%.<br>• All other injuries occurred at a rate of 4.75%.<br>• Soft tissue injuries of the hands accounted for the next largest category of injury in this study, comprising sprains of the first MCP ligaments, subungual hematomas, contusions, and sprains of the carpus. Fractures of the hands and wrist accounted for three injuries; likewise, nasal fractures also accounted for three. |
| **Olympic Boxers** | A study conducted at the United States Olympic Training Center over a 15-year period from 1977-1992 showed the following injury trends:<br>• Of 1,776 reported injuries, only 6.1% met criteria for "serious injury," which was defined as a condition "which could not be remediated through the typical services of an athletic trainer at the USOTC" (concussions would fall within this definition).<br>• Of all injuries, contusions, muscle strains, joint sprains, and tendonitis occurred with the greatest frequency, accounting for 71.9% of all injuries in this study.<br>• Fractures were categorized as a group rather than by body region and accounted for 4.9% of all injuries. Lacerations accounted for another 4.1%, "neural disorders," including concussions, for 1.4%, and all others for the remaining 17.7%.<br>• The study also listed injuries by body region: upper extremity injuries accounted for 24.8% of the total, head and facial injuries for 19.4%, lower extremity injuries for 15.0%, and spinal column injuries for 9.4%.<br>• Multiple examiners, with many specialties and many different levels of training, were involved in data collection. |
| **Professional Boxers** | In a study of professional boxers conducted over a 16-year period in Victoria, Australia:<br>• Face and head trauma made up 89.8% of all injuries reported, with the eye, eyelid, and eyebrow injured most frequently.<br>• Concussions accounted for 15.9% of injuries, with all other injuries accounted for only 10.7% of the total.<br>• It is notable that in the Victoria study, most concussions were not thought to be severe, and the majority of head injuries were lacerations, abrasions, and hematomas.<br>• Next to injuries of the head and face, injuries to the hands and fingers accounted for the next largest number of injuries, accounting for 2.7% of all injuries.<br>• Other sites of injury mentioned include sprains of the ankle, muscular strains of the low back, perforations of the tympanic membrane, dental fractures, fractures of the ribs, and contusions to the lungs. |

Data based on studies from Estwanik JJ, Boitano M, Ari N. Amateur boxing injuries at the 1981 and 1982 USA/ABF national championships. *Phys Sportsmed.* 12(10):123-128; Zazryn TR, Finch CF, McCrory P. A 16 year study of injuries to professional kickboxers in the state of Victoria, Australia. *Br J Sports Med.* 2003;37(5):448-451; Timm KE, Wallach JM, Stone JA, Ryan EJ. Fifteen years of amateur boxing injuries/illnesses at the United States Olympic Training Center. *J Athl Train.* 1993;28(4):330-334.

## TABLE 87.3 DEMOGRAPHIC CHARACTERISTICS OF INDIVIDUALS WITH BOXING INJURIES, US EMERGENCY DEPARTMENTS, 1990–2008

| | Estimated No. of Cases (95% CI) | %[b] | Estimated Annual Average | Injury Rate Per 1000 Participants[a] |
|---|---|---|---|---|
| **Age (Years)** | | | | |
| 6–11 | 4,724 (3,363, 6,085) | 2.9 | 249 | - |
| 12–17 | 42,985 (34,493, 51,476) | 26.0 | 2262 | 14.0 |
| 18–24 | 61,280 (48,518, 74,042) | 37.0 | 3225 | 16.4 |
| 25–34 | 39,333 (31,219, 47,446) | 23.8 | 2070 | 13.6 |
| 35–44 | 12,500 (9,222, 15,778) | 7.5 | 658 | - |
| ≥45 | 4,780 (3,032, 6,529) | 2.9 | 252 | - |
| **Gender** | | | | |
| Male | 150,571 (123,260, 1,777,882) | 90.9 | 7925 | 13.3 |
| Female | 15,025 (10,846, 19,204) | 9.1 | 791 | 8.9 |
| Total | 165,602 (134,891, 196,313) | | 8716 | 12.7 |

[a]Injury rates were not calculated for categories with less than 20 unweighted cases. Injury rates were calculated from 2003 data only.
[b]Percentages may not total 100% because of rounding error.
From Potter MR, Snyder AJ, Smith GA. Boxing injuries presenting to U.S. emergency departments, 1990-2008. *Am J Prev Med.* 2011;40(4): 462-467.

### Chronic Traumatic Encephalopathy

- There are countless anecdotal reports that boxing contributes significantly to late and debilitating chronic brain injury; long-term manifestations include dementia resembling Alzheimer's and movement disorders resembling Parkinsonism.
- These reports are reinforced by retrospective studies conducted on retired boxers. However, the scientific literature lacks conclusive evidence regarding any association of these chronic brain injuries with boxing-related injuries.

#### EFFECT OF REPETITIVE TRAUMA

- Prolonged exposure to repeated concussive and subconcussive head impacts is the major risk factor cited for chronic traumatic brain injury (CTBI).

**TABLE 87.4 SELECTED DIAGNOSES AND INJURED BODY REGIONS FOR BOXING INJURIES: INJURIES RELATED TO PUNCHING BAGS COMPARED WITH INJURIES UNRELATED TO PUNCHING BAGS**

| | *Injuries Related to Punching Bags* | | | *Injuries Unrelated to Punching Bags* | | |
|---|---|---|---|---|---|---|
| | Estimated No. of Cases (%)[a] | Average No./Year | Injury Rate[b] | Estimated No. of Cases (%)[a] | Average No./Year | Injury Rate[b] |
| **Diagnosis** | | | | | | |
| STI | 17,995 (29.6) | 947 | 1.3 | 25,019 (23.9) | 1,317 | 1.8 |
| Fracture | 19,528 (32.1) | 1,028 | 1.4 | 25,941 (24.8) | 1,365 | 2.6 |
| Laceration | 2,550 (4.2) | 134 | - | 11,975 (11.5) | 630 | 0.7 |
| Strain/sprain | 16,134 (26.5) | 849 | 1.4 | 20,309 (19.4) | 1,069 | 1.0 |
| **Body Region Injured** | | | | | | |
| Head/neck | 1,491 (2.4) | 78 | - | 35,800 (34.2) | 1,884 | 2.8 |
| Wrist | 13,543 (22.2) | 713 | 1.0 | 8,298 (7.9) | 437 | - |
| Hand | 33,051 (54.3) | 1,740 | 2.5 | 21,647 (20.7) | 1,139 | 1.5 |
| Finger | 7,340 (12.1) | 386 | - | 9,989 (9.5) | 526 | 0.9 |
| Shoulder | 2,204 (3.6) | 116 | - | 10,204 (9.7) | 537 | 1.0 |
| Total[c] | 60,881 | 3,204 | 4.6 | 104,721 | 5,512 | 8.1 |

[a]Percentage of injuries in each of the two categories of injury, those related and those unrelated to punching bags.
[b]Per 1000 participants per year. Injury rates were not calculated for categories with less than 20 unweighted cases. Injury rates were calculated from 2003 data only.
[c]Only commonly occurring diagnoses and injured body regions are included in the table. Therefore, numbers of injuries do not sum to the totals and percentages do not sum to 100.0%.
STI, soft tissue injury
Reprinted with permission from Potter MR, Snyder AJ, Smith GA. Boxing injuries presenting to U.S. emergency departments, 1990-2008. *Am J Prev Med.* 2011;40(4):462-467.

**TABLE 87.5 PERCENTAGE OF BOXING INJURIES ACCORDING TO BODY REGION INJURED, US EMERGENCY DEPARTMENTS, 1990–2008**

| Body Region | % of Injuries |
|---|---|
| Hand | 33.0 |
| Head and neck | 22.5 |
| Wrist | 13.2 |
| Finger | 10.5 |
| Shoulder | 7.5 |
| Trunk | 6.3 |
| Arm and elbow | 3.6 |
| Lower limb | 3.3 |
| Other | 0.1 |

- Fighters at risk of CTBI appear to be those who have suffered a larger number of knockouts, those possessing inferior fighting and defensive skills, those who cumulatively suffered more powerful blows, and/or those suffering repeated concussions.
- There are compelling anecdotal data regarding the risks and development of CTBI in older boxers; many boxers from the early 1900s had long careers spanning many decades and more than 1,000 bouts.
  - Many boxers of that era also sparred and competed with fighters not matched by size or skill, and most fought with 6-ounce gloves, resulting in a greater force of impact per blow delivered. When sparring, there were no strict enforcement regulations regarding use of headgear.
  - Many fighters, after their professional careers ended, became sparring partners who fought as many as 30 to 40 fights daily, adding to the total injury burden suffered in their lifetimes.

### BIOLOGIC MARKERS FOR CHRONIC TRAUMATIC ENCEPHALOPATHY

- There are biologic markers, such as the apolipoprotein E-ε-4 genotype, that may indicate a person is at higher risk of CTBI.
- The effects of this genotype, coupled with a greater number of heavy blows to the head, may exert a multiplicative rather than additive contribution to the pathogenesis of CTBI.

### MONITORING BOXERS FOR CHRONIC TRAUMATIC ENCEPHALOPATHY

- When considering the presence of chronic brain injury, it is vital that the physician is attuned to subtle signs, such as loss of skill or behavioral alterations.
- An experienced and skilled fighter should be able to avoid a majority of punches.

### CAVUM SEPTUM PELLUCIDUM (CSP)

- Normal variant CSF space between leaflets of the septum pellucidum
- Boundaries
  - Anterior: genu of corpus callosum
  - Superior: body of corpus callosum
  - Posterior: anterior limb and pillars of fornix
  - Inferior: anterior commissure and rostrum of corpus callosum
  - Lateral: leaflets of septum pellucidum
- In boxers, this condition may be a marker of significant atrophy; presumed related to trauma.
- Believed to be the result of suffering a large number of knockouts
- Also associated with schizophrenia and post-traumatic stress disorder
- John D. Spillane, MD, FRCP, was the first to note CSP in 1962.
  - Identified clinical and pneumoencephalographic findings in five retired professional boxers who had chronic neurological problems in later life
  - None of these fighters had fought fewer than 200 bouts.

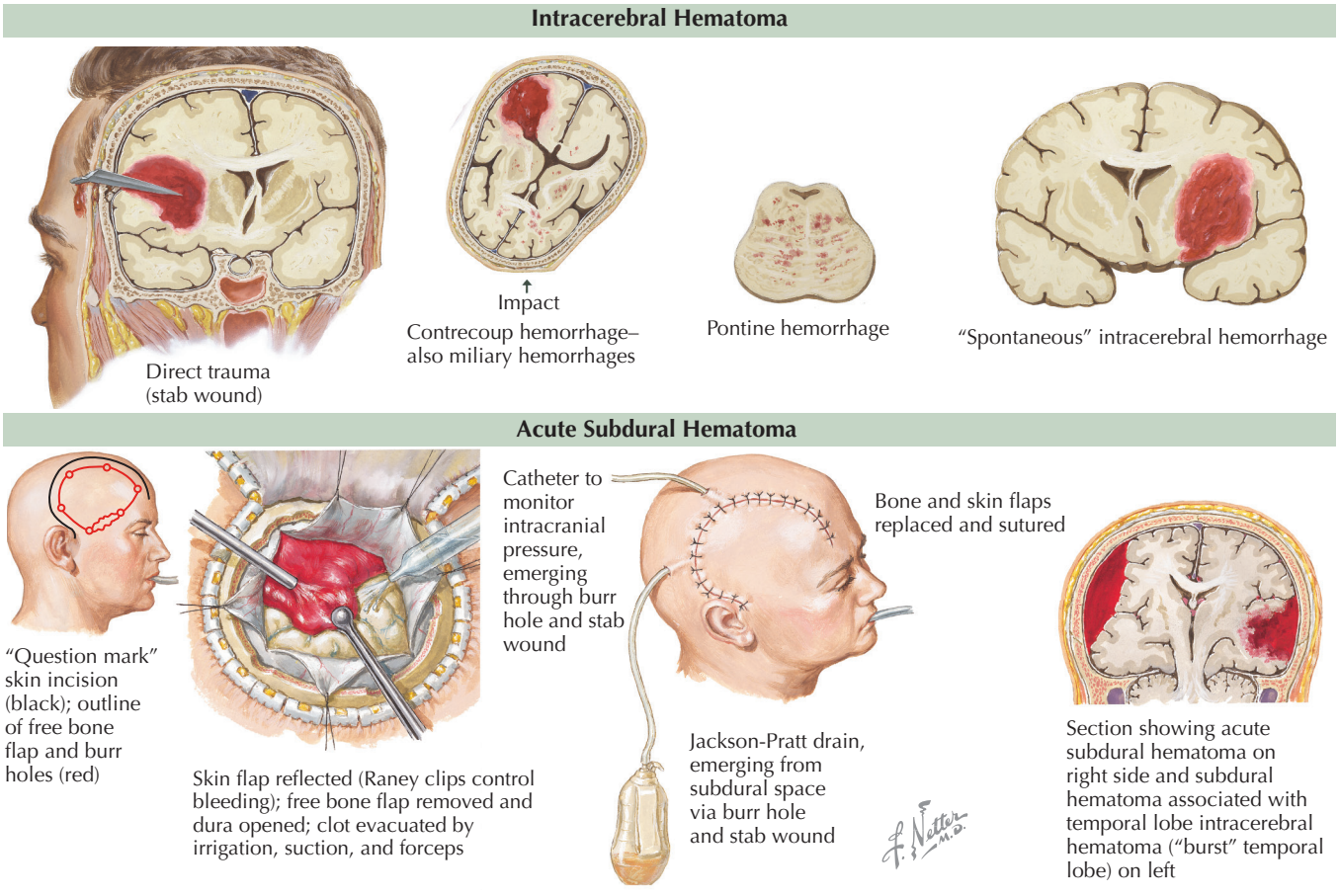


**Figure 87.1.** Acute traumatic brain injury.

- Performed an autopsy study of 15 boxers who fought between 1900 and 1940
  - The number of bouts per fighter ranged from 400 to 700.
  - Observations noted as markers of chronic traumatic brain injury included
    - abnormalities of the septum pellucidum
    - scarring of the cerebellum and other areas of the brain
    - degeneration of the substantia nigra
    - the regional appearance of neurofibrillary tangles
- There is no clear information regarding the prevalence of CSP in the general population at any time after the neonatal period; the condition is found incidentally in between 0.7% and 37% of brain imaging studies.

## Nasal Fracture

- Common occurrence
- Assume a fracture has occurred if there is a contusion or tenderness over the bridge of the nose.
- Imaging is often unnecessary. If required, CT is preferred over plain film because of better visualization of details.

## Nasal Septal Hematoma

- Typically a consequence of a nasal fracture
- If untreated, can be associated with necrosis of the septum
  - May lead to saddle deformity of the bridge of the nose

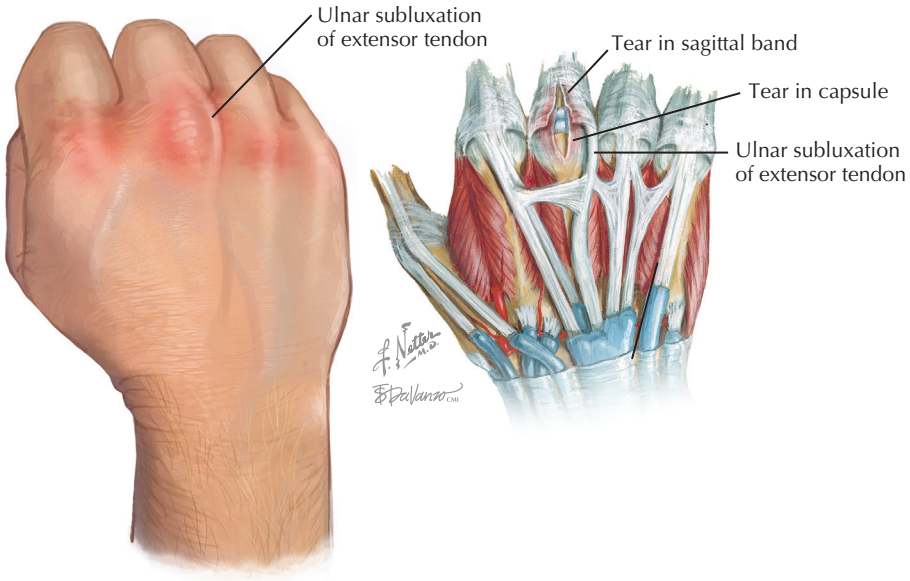


**Figure 87.2.** Boxer's knuckle.

- Should be evacuated ASAP
  - Simple incision and expression of the clot, if present, followed by anterior packing
  - Packing remains in place 2–3 days.
- Infection is a concern.
  - May occur within 3 days—close follow-up with ENT is recommended

## Hand Injuries

### Boxer's Knuckle (Fig. 87.2)

**Examination:** Observation of marked swelling at the metacarpophalangeal joint (MCPJ) and loss of range of motion, frequently w/ extensor lag; ulnar subluxation of the extensor tendon (obvious when flexing MCPJ); tenderness at ruptured sagittal band

**Imaging:** X-ray may show subchondral cysts, which may indicate metatarsophalangeal joint osteochondral fracture.

**Treatment:** Direct repair is performed through a curvilinear incision, avoiding the prominence of the knuckle. Repaired in 60–70 degrees flexion; capsular tears, if present, are debrided but frequently are not repaired. Immobilized postoperation at 60–70 degrees for 6 weeks; occupational/hand therapy post immobilization

**Return to sport:** Patients are usually ready to return to punching at 8–12 weeks after immobilization when wounds are completely healed and pain-free, and full range of motion has been restored with normal strength.

### Traumatic Carpal Boss

**Examination:** A painful, bony prominence at the carpometacarpal joint (CMCJ) either with or without a cyst, usually present in digits two and three.

**Imaging:** X-ray—look for a hypertrophic bone adjacent to the CMCJ, and possibly widened CMCJ space. If the condition is chronic, the obliteration of CMCJ should be noted.

**Treatment:** Arthrodesis of affected joints, with removal of all ganglion cysts (if present), and the removal of hypertrophic bone; K-wires for stability. The injured region is immobilized for 6–8 weeks, or until evidence of radiographic union. OT begins when the cast and K-wires are removed.

**Return to sport:** It usually takes at least 6 months to achieve pain relief and regain the satisfactory range of motion and strength needed for competition.

## NEUROIMAGING

Positron emission tomography (PET) scans: Hypometabolism in the frontal and parietal lobes has been observed in boxers with CTE, similar to the patterns seen in Alzheimer's disease.

Magnetic resonance imaging (MRI): Useful in delineating contusions, subdural and white matter lesions, arachnoid cysts, and brainstem and cerebellar anatomy

Computed tomography (CT): Useful in sorting acute bleeding injuries

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 88 WRESTLING

Robert Kiningham • Aaron Monseau

## INTRODUCTION

- Widely considered to be the world's oldest sport, dating back to the ancient Greeks
- It is a worldwide sport, with particular popularity in Eastern Europe, the Middle East, Asia, and North America.
- Men's wrestling was an original Olympic sport, whereas women's wrestling was added to the Olympics in 2004.

## EPIDEMIOLOGY

- Ranks sixth among high school boys in participation, with almost 260,000 participants in 10,597 schools (2014–2015)
- 220 National Collegiate Athletic Association (NCAA) and 53 National Association of Intercollegiate Athletics (NAIA) collegiate wrestling programs in the US
- The injury rate among high school boys who wrestle is 2.50 per 1000 athletic exposures, which is the second highest injury rate in high school male sports and is approximately the same as that in soccer.
- The injury rate in male college athletes is 7.25 injuries per 1000 athletic exposures as per the NCAA Injury Surveillance System.
- The most common site of injury differs depending on the competition level.
  - Youth wrestling: Hand, wrist, and finger
  - High school wrestlers: Shoulder
  - College wrestlers: Knee
- Approximately 7% of injuries result from illegal moves.
- Skin infections are the most common reason wrestlers seek medical attention and account for most of the time lost from competitions and practices.

## GENERAL PRINCIPLES

### Wrestling Styles

- International wrestling: Freestyle and Greco-Roman
  - Greco-Roman: Wrestlers are not permitted to attack an opponent's legs.
  - Freestyle has both men's and women's divisions.
- United States youth, high school, and college wrestling: Folkstyle
- Greco-Roman and Freestyle reward throws more than Folkstyle, which leads to more high-velocity mat injuries, including head and neck injuries and concussions.
- Folkstyle allows wrestlers to stay in a defensive down position for longer, which leads to more defensive injuries such as shoulder strains and subluxations.

### Equipment and Safety Issues

- Most wrestling matches are contested in a singlet, which is a tight, one-piece uniform, while most wrestlers practice in shorts or sweatpants and a T-shirt.
  - Looser-fitting practice clothes can cause injuries such as finger dislocations when they are loose; hence, most wrestlers will tuck their shirts into shorts and even tuck their pant legs into socks.
- Wrestling shoes provide a light but supportive point of traction on the wrestling mat, which can be slippery when wet.
- Wrestling mats have greatly improved over the past 25 years and have become much more durable.
- Headgear is required in college and high school wrestling but is almost never worn in international wrestling events.
- Supportive braces will have to be approved by the referee and will likely be denied if there are any metallic pieces palpable owing to the possibility of injury to the opponent.

### Rules

- Governing bodies of wrestling in the US
  - High school
    - The National Federation of State High School Associations (NFHS) is the national advisory organization for state high school athletic organizations. Most states adopt the NFHS rules. However, individual state high school athletic associations retain the power to determine their own state's rules. Physicians should check with their specific state's high school athletic association to see if their rules differ from the NFHS rules.
  - College
    - NCAA Wrestling Rules Committee
    - The NAIA and National Junior College Athletic Association (NJCAA) both defer to the NCAA rules for wrestling.
  - International styles (Greco-Roman and freestyle)
    - USA Wrestling is the governing body for international-style events
- Injury time during matches (Table 88.1)
  - Injury time-outs are stoppage of wrestling requested by a coach, wrestler, or referee.
  - Injury time is defined as the cumulative time spent under evaluation and recovery from an injury sustained during the match, including overtime.
- Recovery time
  - Time spent under evaluation and recovery from an injury that resulted from an illegal action by the opposing wrestler
- Bleeding time during matches
  - Time spent stopping the bleeding and cleaning blood from mats, wrestlers, and equipment; distinct from injury or recovery time
  - The NCAA does not limit the bleeding time. The referee, in consultation with medical personnel, has the authority to stop the match and declare the nonbleeding wrestler the winner by medical default if the bleeding becomes excessive or causes an inordinate number of time-outs.
- Concussions
  - The referee has the responsibility to stop the match if he/she suspects a concussion according to the NFHS, NCAA, and USA Wrestling rules.
  - The NCAA makes a distinction between injury time and time spent evaluating a concussion. Medical personnel have unlimited time to evaluate a wrestler suspected of sustaining a concussion. This time does not count against the wrestler's injury time or recovery time.
- Skin infections (see the Skin Infection section)

### Specific Training and Physiology Issues

#### Weight Classifications

- Competitors are matched by weight classes in all styles:
  - Men's freestyle: 6 weight classes
  - Women's freestyle: 6 weight classes

**TABLE 88.1 INJURY ALLOWANCES PER WRESTLING MATCH***

| | NFHS | NCAA | USA Wrestling |
|---|---|---|---|
| Injury Time-Outs | 2 | 2 | Unlimited |
| Cumulative Injury Time | 90 seconds | 90 seconds | Unlimited |
| Blood Time | 5 minutes | Unlimited | Unlimited |
| Concussion Evaluation | Counts toward injury time | Unlimited | Unlimited |
| Recovery Time† | 2 minutes | 2 minutes | Unlimited |

*Exceeding indicated values results in the wrestler losing the match by injury default.
†Time to recover from the opponent's illegal action; if the injured wrestler cannot resume wrestling within 2 minutes, he is declared the winner and the opponent is disqualified.
NFHS: National Federation of State High School Associations; NFHS 2015–2016 Wrestling Rule Book.
NCAA: National Collegiate Athletic Association; 2015–2016 NCAA Wrestling Rules and Interpretations.

**BOX 88.1 NCAA AND NFHS WEIGHT MANAGEMENT PROGRAMS***

- Preseason establishment of minimum weight based on lean weight assessment
  - NFHS: 7% body fat (boys), 12% body fat (girls)
  - NCAA: 5% body fat (men)
- Urine specific gravity (Usg) at initial weight certification
  - NFHS: Usg < 1.025
  - NCAA: Usg < 1.020
- Not more than 1.5% body mass loss per week
- Weigh-ins must occur within an hour of the start of dual meets (2 hours for tournaments)
- The use of laxatives, diuretics, emetics, excessive food and fluid restriction, self-induced vomiting, impermeable (plastic or rubber) suits, hot rooms, hot boxes, steam rooms, and saunas is prohibited.
- NFHS: The weight management program should include "a nutritional component developed at the local level."

*The NFHS is an advisory organization. Each state athletic association determines its own specific rules that may or may not incorporate the NFHS recommendations.
NFHS: National Federation of State High School Associations: NFHS 2015–2016 Wrestling Rule Book.
NCAA: National Collegiate Athletic Association; NCAA Wrestling 2015–2016 and 2016–2017 Rules and Interpretations.

- Men's Greco-Roman: 6 weight classes
- USA collegiate men folkstyle: 10 weight classes
- USA collegiate women folkstyle: 8 weight classes
- USA high school folkstyle: 14 weight classes

### Weight Management

- Wrestlers have traditionally wrestled in the lowest weight class possible in the belief that this will provide them a competitive advantage.
- 85% of US high school wrestlers lose over 10% of body weight to achieve their competitive wrestling weight.
- In-season weight loss occurs through frequent weight cycles involving rapid weight loss and then weight gain after a competition.
- Surveys have found that high school and college wrestlers lose an average of 3.5–5.5 kg of weight in the week preceding a match.
- Most commonly used method of rapid weight loss is sweating combined with fluid and caloric restriction.
- Diuretics, laxatives, and vomiting are less frequently used, with 1%–5% of wrestlers using these methods.

### Weight Loss and Health Concerns

- Rapid weight loss of >5% of body mass in 1–2 days is cited as an acute health risk. During 1997–1998 season, three collegiate wrestlers died from complications of rapid weight loss.
- Long-term consequences of frequent weight cycling are unknown. Impaired growth, eating disorders, and obesity have been proposed but not consistently found in longitudinal studies.

### Weight Loss and Performance

- Impact on performance dependent upon level of dehydration, glycogen stores, time for replenishment (i.e., time between the weigh-in and the competition), and number of same-day matches
- Dehydration of 2%–3% has limited effects on muscle strength and anaerobic power.
- Glycogen-depleted wrestlers reach a performance limiting level of glycogen within 7 minutes of wrestling. Replenishment of glycogen stores takes 24–48 hours. Therefore, glycogen depletion can have a profound impact on performance during a multiple-match day (e.g., a tournament).
- Weight loss of 3%–6% body fat over 3–4 days even without dehydration significantly reduces the average work performance for tasks as short as 6 minutes.

### Weight Management Guidelines and Rules (Box 88.1)

- 1964: NFHS Athletic Associations urged states to pass regulations that included "weight control plans" that would decrease potentially dangerous rapid weight loss methods.
- 1976: American College of Sports Medicine Position Stand on Weight Loss in Wrestlers called for weight certification based on a preseason measurement of body composition.
- 1991: Wisconsin becomes the first high school athletic association to implement a mandatory preseason body composition as the basis for weight certification. Minimum weight was based on 7% body fat. A nutrition education program for wrestlers and coaches was included. Subsequent studies found significant decreases in rapid weight loss and weight cycling in Wisconsin high school wrestlers. Michigan instituted a similar rule in 1997.
- 1997–1998: Deaths of three collegiate wrestlers while attempting rapid weight loss
- 1998: NCAA implements five rule changes for the 1998–1999 competitive season, including a minimum weight based on 5% body fat and moving weigh-ins to at most 1 hour before competition (see Box 88.1).
- NFHS calls for each individual state high school athletic association to develop a program to "discourage excessive weight reduction and/or wide variations in weight." This recommendation became a national mandated rule for the 2006–2007 season. However, the NFHS is an advisory organization. Each state athletic association sets its' own rules and regulations.
- The NJCAA and NAIA both mandate that their member institutions adhere to the NCAA Weight Management Program for wrestlers.
- NCAA and NFHS weight management programs both allow for an exception to the minimum body fat certification for athletes who have <5% body fat (NCAA) or <7% body fat

(NFHS) at the preseason body composition assessment. These wrestlers are allowed to wrestle at their preseason weight with a statement from a physician that this weight is their "normal" healthy weight.
- Very unusual for postpubescent wrestlers to have <7% body fat in high school or <5% body fat in college. Studies of wrestlers at NCAA championships found no wrestlers with >5% of body fat, and <5% of wrestlers had <7% of body fat.
- USA Wrestling governs international-style wrestling tournaments for all age groups. USA Wrestling prohibits several rapid dehydration techniques such as saunas and "hot boxes" and vapor-impermeable suits at their non-Senior level competitions. However, there are no other weight loss rules and there is no minimal weight certification.

### Impact of Weight Management Rules in Wrestling

- Average weight gain the day after weigh-ins at NCAA championships has decreased from 3.7 kg before implementation of weight management rules to 1.7 kg after implementation.
- Surveys in Wisconsin and Michigan performed before and after implementation of the corresponding weight-monitoring programs revealed a significant decrease in average weekly weight loss, frequency of weight loss cycles, and use of potentially harmful weight loss methods.
- Moreover, studies have revealed that US high school wrestlers will use rapid weight loss methods at even international-style tournaments in which there are no weight loss rules; this indicates that despite educational efforts, high school wrestlers will revert to previous weight loss patterns if rules are not in place.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Skin Infections

**General considerations:** Wrestlers are at an increased risk because of two factors:
- Repetitive skin trauma causes a breakdown in the natural skin barriers to infection.
- Wrestlers have close and prolonged exposure to potential infectious agents of the skin.

**Locations:** Most commonly infected areas are the head, neck, and face, which are the areas of maximum skin-to-skin contact. Primary transmission is via skin-to-skin contact and not from mats and other environmental sources.

**Infectious agents:** In college wrestlers, herpes simplex virus (HSV) is the skin infection responsible for the most lost wrestling time, followed by bacterial and fungal skin infections. Wrestlers with skin infections should be withheld from contact with other wrestlers until it is determined that they are no longer infectious. Both the NCAA and NFHS have specific criteria for return to competition after a wrestler has been diagnosed with an infectious skin condition (Table 88.2 and below). The designated on-site physician or certified athletic trainer for a competition may exclude any wrestler deemed infectious even with documented appropriate treatment from the wrestler's personal or team physician (NCAA and NFHS).

**TABLE 88.2 SKIN INFECTIONS: RETURN TO WRESTLING**

| Infection | NFHS | NCAA |
|---|---|---|
| HG Primary | No competition for 10 days (uncomplicated) or 14 days (systemic sx) | No new lesions for 72 hours |
| HG Primary and Secondary | No systemic symptoms<br>All lesions scabbed over<br>No new lesions for 48 hours<br>Minimum 120 hours oral antiviral | No systemic symptoms<br>All lesions scabbed over<br>Minimum 120 hours oral antiviral |
| Molluscum contagiosum | 24 hours after curettage | Lesions curetted or removed and covered |
| Bacterial | All lesions dry<br>No new lesions for 48 hours<br>Oral antibiotic for minimum 3 days<br>MRSA: ***minimum 10 days oral antibiotics*** | All lesions dry<br>No new lesions for 48 hours<br>Oral antibiotic for minimum 3 days |
| TG | Oral or topical antifungal for minimum of 72 hours (14 days for tinea capitis) | Oral or topical antifungal for minimum 72 hours (14 days ***oral*** therapy for tinea capitis)<br>Participation allowed if active lesions are adequately covered |

*HG,* herpes gladiatorum; *sx,* symptoms; *TG,* tinea gladiatorum
NFHS: National Federation of State High School Associations; NFHS 2015–2016 Wrestling Rule Book
NCAA: National Collegiate Athletic Association; NCAA Wrestling 2015–2016 and 2016–2017 Rules and Interpretations

### Herpes Gladiatorum (HG)

**Description:** Cutaneous infection in athletes caused by herpes simplex virus-1 (HSV1)

**History:**

**Primary infection:** Often associated with mild systemic symptoms (fever, fatigue, and myalgia) that occur 1–2 days before the appearance of vesicles. Vesicles usually resolve within 10–14 days. After the primary infection, HSV1 lives dormant in the trigeminal ganglion for facial lesions or dorsal root ganglion for lesions in other areas.

**Secondary infection:** Burning or itching usually occurs at the outbreak site for 2–3 days before the appearance of vesicles. Re-activation can be induced by stress, trauma, temperature extremes, and/or immunosuppression.

**Physical examination:**

**Primary infection:** Multiple clusters of vesicles that can spread over several dermatomes; facial lesions often associated with cervical adenopathy

**Secondary infection:** Vesicles localized to a specific dermatome; herpetic lesions in wrestlers are often not vesicular because the lesions get deformed and abraded by local trauma induced during wrestling.

**Diagnostic considerations:** Tzanck staining from a freshly unroofed vesicle is relatively insensitive. Viral cultures taken from the base of an unroofed lesion; most sensitive when performed within 24–48 hours of appearance (can take up to 5 days to grow). Polymerase chain reaction (PCR) testing, the most sensitive and specific test, can detect asymptomatic viral shedding. However, detection of HSV particles on PCR testing does not necessarily indicate infectivity.

**Treatment:** Oral antiviral treatment should be initiated as soon as infection (or re-activation) is suspected. Valacyclovir 500 mg twice a day for 7 days. Topical antiviral treatments have not been shown to reduce viral shedding or transmission;

use in wrestlers is not recommended. Immediate referral to ophthalmologist if ocular involvement suspected because of the risk of dendritic keratitis and corneal scarring

**Return to play** (Table 88.2)**:**

- Primary outbreak
  - **NFHS:** No competition for a minimum of 10 days if uncomplicated and for 14 days if general body signs and symptoms (fever and adenopathy) are present. All lesions must be scabbed over with no oozing. No new lesions for at least 48 hours
  - **NCAA:** Appropriate dosage of systemic antiviral therapy for at least 120 hours before and at the time of competition. No new blisters for 72 hours before competition, and no general body signs or symptoms; all lesions must have a firm adherent crust with no evidence of secondary bacterial infection.
- Recurrent outbreak
  - **NFHS:** A minimum of 120 hours or 5 full days of oral antiviral treatment; no new lesions for at least 48 hours; all lesions scabbed over
  - **NCAA:** Minimum of 120 hours of oral antiviral treatment; all lesions must be scabbed over
- Covering an infectious herpetic lesion does not make the athlete eligible to participate (NCAA and NFHS).

**Prevention:** Herpes gladiatorum (HG) spreads very rapidly between wrestlers. It is estimated that a wrestler exposed to another wrestler with active HG has a 32.7% chance of contracting HG. Time from exposure to outbreak ranges 4–11 (average 6.8) days. Viral shedding occurs before the appearance of vesicles. Therefore, it is important to educate wrestlers, parents, and coaches about the signs/symptoms of primary and recurrent HG infections so that infected wrestlers can be excluded as soon as possible to minimize transmission to other wrestlers. Valacyclovir 500–1000 mg/day during the wrestling season has been shown to be highly effective in reducing recurrent outbreaks in wrestlers with a history of HG infection. Consider a course of valacyclovir for a wrestler exposed to another wrestler with active HG.

### Common Warts and Molluscum Contagiosum

**Description:** Viral skin infections that can be transmitted by skin-to-skin contact

**Physical examination:** Molluscum appears as a round papule with a central keratotic plug, giving it an umbilical appearance.

**Diagnostic considerations:** Diagnosis is usually based on characteristic appearance. Lesions can be biopsied or excised and sent to pathology for confirmation of diagnosis.

**Treatment:** Molluscum: Curettage, cryotherapy with liquid nitrogen, or chemical destruction; lesions usually spontaneously resolve within 6 months without treatment.
Common warts: cryotherapy, salicylic acid, and hyfrecation

**Return to play** (see Table 88.2)

**NFHS:** Participation allowed 24 hours after curettage

**NCAA:** Lesions must be curetted or removed before the event and covered.

**Prevention:** Good skin care; both molluscum and common warts tend to grow in areas of cracked or otherwise damaged skin.

### Bacterial Skin Infections

**Description:** Classified by the depth of the infection and presence of systemic symptoms (e.g., fever, myalgia, and fatigue)

- **Impetigo**
  - Superficial infection characterized by honey-colored crusting; can have associated bullae
  - Nonbullous impetigo: *Staphylococcus aureus* and *Streptococcus pyogenes* (Group A strep, *S. pyogenes*)
  - Bullous impetigo: Always caused by *S. aureus*
- **Folliculitis**
  - Infection of a hair follicle that remains superficial and localized to the epidermis
  - Most commonly caused by *S. aureus*
  - "Hot-tub" folliculitis: *Pseudomonas aeruginosa*
- **Furuncle**
  - Infection that has spread from the hair follicle deeper into the dermis and subcutaneous tissue, forming a perifollicular abscess
  - Almost always caused by *S. aureus*
- **Carbuncle**
  - Group of interconnected furuncles
  - Often associated with systemic symptoms (fever, myalgia, and fatigue)
- **Cellulitis**
  - Diffuse infection of the connective tissue with inflammation of the dermal and subcutaneous skin layers
  - Triad of erythema, edema, and warmth, with absence of a discrete focus of infection
  - *S. aureus* or *S. pyogenes*
- **Erysipelas**
  - Superficial infection of the dermis that produces intracutaneous edema, causing palpable skin margins
  - Usually *S. pyogenes*
- **Methicillin-resistant staphylococcus aureus (MRSA) infections**
  - Infections caused by *S. aureus* species that have the mecA gene, which confers resistance to most beta-lactam antibiotics
  - Typical presentation is a pustular lesion with central necrosis.
  - Can also present as a cellulitis, folliculitis, furuncle, or carbuncle

**History:** Presence of fever, lethargy, and/or hypotension indicates deeper infection; hospitalization should be strongly considered. Athletes with MRSA infections will often complain of pain out of proportion of the size and appearance of the lesion. Complaint of a "spider-bite" associated with a 65% chance that the lesion is caused by MRSA.

**Diagnostic considerations:** Fluctuant lesions should be incised and drained, with purulent material sent for culture.

**Treatment:** Definitive treatment of fluctuant lesions, even MRSA lesions, is incision and drainage (I&D). Giving antibiotics after I&D does not improve the cure rate but may decrease the formation of subsequent lesions. Nonfluctuant lesions should be empirically treated with a first-generation cephalosporin or amoxicillin clavulanate unless MRSA is *strongly* suspected. Overuse of antibiotics leads to the creation of resistant organisms. If MRSA is strongly suspected, clindamycin, doxycycline, or trimethoprim/sulfamethoxazole should be prescribed. MRSA resistance can develop to these antibiotics as well. Check with local public health or infectious disease experts for the MRSA resistance pattern in your area.

**Return to play** (Table 88.2)**:** Athletes with bacterial skin infections should be checked daily for their response to treatment. Wrestlers with bacterial skin infections should be prohibited from all contact wrestling activities until determined to be noninfectious.

**NFHS:** No drainage from lesions or new lesions for preceding 48 hours; oral antibiotics for at least 3 days; if MRSA diagnosed or suspected, at least 10 days of oral antibiotics before returning to competition

**NCAA:** All lesions must be dry, with no new lesions for 48 hours; oral antibiotics for at least 3 days; covering an infectious lesion does not make the athlete eligible to participate (NCAA and NFHS).

**Prevention:** Most bacterial infections in wrestlers are thought to be transmitted by person-to-person contact; hence, the most

important preventive measure is to identify infections early and immediately exclude athletes with infectious lesions from wrestling. MRSA has been transmitted by equipment and towels in other sports. Mats should be cleaned daily. Discourage towel and equipment sharing. All wrestlers should shower with soap after every practice and competition. Protocols for MRSA eradication in carriers have been published. However, the effectiveness in wrestlers has not been studied. Prophylactic use of antibiotics has been shown to promote antibiotic resistance. A local infectious disease specialist should be consulted before instituting an eradication program in identified or suspected MRSA carriers.

### Tinea Gladiatorum (TG)

**Description:** Cutaneous fungal skin infections (tinea corpora) in athletes; a survey reported that 60% of college wrestlers and 52% of high school wrestlers developed a fungal skin infection during the season

**History:** Dermatophytes infect the outer layer of the epidermis. Arthrospores with extended survival time are shed in dead skin scales.

**Physical examination:** Classically described as an erythematous annular plaque with surface scale at the periphery and central clearing; tinea gladiatorum (TG) in wrestlers can have an atypical appearance with more irregular borders and less central clearing. Also check scalp for tinea capitis. Scalp can be an asymptomatic reservoir of fungus.

**Diagnostic considerations:** Diagnosis is usually made clinically based on appearance. KOH prep from a scraping of the outer border of the lesion can be performed to confirm diagnosis, but it is not very sensitive. Fungal cultures are more sensitive and specific than KOH prep examination but can take 1–2 weeks to grow.

**Treatment:**

- Topical antifungal creams are the first line of treatment for TG.
  - Allylamines are fungicidal and thus more effective than the fungistatic imidazoles.
  - Treatment should be administered for a minimum of 2 weeks or until a week after the lesion has faded (whichever is longer) to prevent recurrence.
- Oral antifungal agents can be used if there are an extensive number of tinea lesions or if there is a failure to clear the lesions with topical antifungals.
- Tinea capitis: Two weeks of an oral antifungal such as terbinafine 250 mg/day for 2 weeks

**Return to play** (see Table 88.2)**:** Wrestlers should be withheld from activities involving physical contact until lesions have faded.

**NFHS:** Oral or topical treatment for at least 3 days for TG and 2 weeks for tinea capitis.

**NCAA:** At least 3 days of topical therapy for TG and 2 weeks of oral antifungal treatment for tinea capitis; the NCAA allows participation if active lesions are in a body location that can be adequately covered with a wrap that cannot be dislodged during the match.

**Prevention:** TG primarily transmitted by skin-to-skin contact; hence, the keystone of a prevention program is to identify and exclude wrestlers with active lesions. Prophylaxis with oral antifungal agents (itraconazole and fluconazole) has been found to effectively decrease TG outbreaks in wrestlers. The risk–benefit profile of prevention of an essentially benign condition (HG) with a potentially liver toxic agent (oral antifungals) must be considered.

### Auricular Hematoma (Fig. 88.1)

**Description:** Blunt trauma to the ear can cause accumulation of blood in the subperichondrial space between the perichondrium and the auricular cartilage, resulting in a hematoma. The perichondrium carries the blood supply to the auricular cartilage. The formation of a hematoma separates the perichondrium from the cartilage, thereby interrupting the blood supply. If persistent, an auricular hematoma can result in cartilage death. Over time, new abnormal cartilage with fibrosis will form, resulting in the characteristic deformity known as cauliflower ear.

**Treatment:** Goals of treatment: remove the blood and prevent re-accumulation; for acute hematomas, blood can be removed by aspiration with an 18-gauge needle. Hematomas may have to be evacuated through an incision. Removal of the blood leaves a potential space into which blood will rapidly re-accumulate with even minimal trauma. Therefore, treatment should include a technique to prevent blood re-accumulation.

Presentation of auricular hematoma (cauliflower ear).

Protective headgear is worn to prevent auricular hematomas.

**Figure 88.1.** Auricular hematoma ("cauliflower ear"). (Photograph © Del Brown Photography, LLC.)

Most effective technique uses absorbable mattress sutures to close the space. Outcomes have been excellent, and wrestlers may immediately return to wrestling.

**Prevention:** The NFHS and NCAA mandate wearing a headgear while wrestling to prevent direct auricular trauma, but this rule is generally only enforced during official matches. International-style wrestling (freestyle and Greco-Roman) does not require headgear.

## Head and Face Injuries

### Brow Lacerations

**Description:** Eyebrow lacerations make up the largest proportion of lacerations and are typically the result of the two wrestlers striking heads. A knee or elbow to the brow is also a common cause of lacerations.

**Physical examination:** Assess for concussion and facial fractures. Assess depth of wound, presence of arterial bleeding, and neurologic function.

**Treatment:**

- During match
  - Control bleeding and protect wound by placing gauze over wound and tightly wrap head with an elastic tape under an athletic tape.
  - Ferric subsulfate (Monsel) solution and/or skin glue can control bleeding. However, these substances can complicate subsequent repair and healing. Ferric subsulfate solution creates necrotic subcutaneous tissue that potentially increases the risk of infection if sutured over.
- After match
  - Suturing the wound will allow the wrestler to continue wrestling and minimize subsequent bleeding.
  - Vertical mattress sutures are particularly useful to withstand large amounts of tension placed on a wound during wrestling.

### Epistaxis

**Description:** Usually unilateral and anterior involving Kiesselbach's plexus

**Physical examination:** Examine to ensure that there is no septal hematoma

**Treatment:** Within a match, bleeding can usually be controlled with insertion of a folded cotton roll to apply compression to the bleeding septum. NFHS allows 5 minutes of cumulative blood time per wrestler per match, beyond which the wrestler is disqualified. No blood time limit in NCAA wrestling. After the match, persistent bleeding can be stopped by vasoconstriction with 0.05% oxymetazoline nasal spray that can be directly applied to the bleeding or used to soak a cotton roll that is placed in the nose for 4–5 minutes. Directly visualized bleeding vessels can also be stopped by gentle cautery with silver nitrate. Septal hematoma needs immediate drainage to prevent cartilage death. Refer to an otolaryngologist if the on-site healthcare provider is not experienced in management.

**Prevention:** Petroleum jelly can be spread on the septum to reduce dryness.

## Lower Limb Injuries

### Prepatellar Bursitis

**Description:** The prepatellar bursa lies directly anterior to the patella and can be easily injured with direct trauma, most commonly from repeated striking of the knee on the mat. The bursa can fill with fluid and/or blood.

**Physical examination:** Swollen and tender area just anterior to the patella; may be associated erythema and warmth of the skin. Typically, the knee is tender but not painful. Range of motion is normal, with pain only in full flexion because of stretching of the bursa; must distinguish infectious bursitis from traumatic bursitis. Infections occur through a breakdown in the overlying skin. Traumatic bursitis should not be aspirated because of the risk of introducing an infection. However, consider aspiration if infection is suspected. Bursal fluid should be sent for Gram stain and culture.

**Treatment:** Focuses on reducing the irritation of the bursitis through suspension of activity and wearing a knee pad; typical neoprene knee pads used often in wrestling will likely not provide enough padding once traumatic bursitis has developed; hence, a large, volleyball-style knee pad is usually required. Off the mat, an elastic bandage or a simple neoprene or elastic knee sleeve will maintain compression and reduce the swelling and pain of bursitis. A week or two of immobilization and avoidance of direct trauma has been recommended to shorten the course of the injury, but many wrestlers will choose to practice and compete. Once the swelling of the bursa has resolved, the wrestler should continue to wear a knee pad to prevent recurrence. Surgical bursectomy can be considered for wrestlers who develop chronic bursitis.

**Return to play:** Wrestlers may compete with a knee pad if bursal swelling is not excessive and there are no signs/symptoms of infection.

**Prevention:** Floors around wrestling areas must be covered with mats to prevent knee trauma.

### Medial Collateral Ligament (MCL) Sprain

**Description:** Medial collateral ligament (MCL) of the knee is the major stabilizer against valgus force and is frequently placed under great stress in the sport of wrestling.

**Physical examination:** See the Prepatellar Bursitis section

**Treatment:** Treatment protocols for MCL injuries that emphasize early controlled motion and protected weight bearing have been found to be successful in returning athletes to full function. A hinged brace can be used to protect against valgus stress and external rotation.

**Return to play:** Return to wrestling is based on a functional assessment of the athlete more than a specific time frame. When a hinged knee brace has been utilized after a sprain, it may be used in wrestling competition if appropriately padded, which must be approved by the referee before a match.

## Upper Limb Injuries

### Shoulder Subluxation/Dislocation

**Description:** Shoulder injuries are common in wrestling due to extreme anterior flexion, abduction, and external rotation that occurs frequently. These positions may lead to subluxation or dislocation.

**History:** A wrestler with a glenohumeral dislocation will present in acute pain with the arm typically held in a fixed position in slight internal rotation and abduction.

**Physical examination:** Important to assess neurovascular status
Axillary nerve: sensation over lateral aspect of shoulder
Brachial plexus: hand strength and sensation

**Treatment**: The ease of reduction is related to the amount of spasm of the surrounding shoulder musculature, which is often directly related to the amount of time since the injury. Therefore, prompt reduction has the advantage of avoiding muscular spasm and decreasing neurovascular compromise. The choice of reduction technique should be based on the familiarity and comfort level of the physician. Certain older techniques, such as the Kocher and Hippocrates methods, have been associated with more postreduction complications and should be avoided. Immobilization in a sling for 3 weeks recommended by certain orthopedic surgeons after a first dislocation; physical therapy for rotator cuff strengthening and neuromuscular training; surgery after an acute dislocation has been shown to reduce

the risk of recurrence; referral to orthopedic surgeon to discuss the potential risks and benefits of surgery should be considered.

**Return to play:** Full rotator cuff strength and absence of apprehension sign

**Prevention:** Shoulder flexibility: Shoulder joint stiffness may increase the risk of injury in wrestlers; rotator cuff strengthening and neuromuscular control

### Cervical Strain/Sprain

**Description:** Cervical injuries can occur when a wrestler tries to resist a force placed on his/her neck by the opponent or while being returned to the mat during a throw by the opponent.

**History:** The wrestler may recall the exact instant that pain began or may simply notice pain after a match. Wrestlers who compete in several matches in 1 day in a tournament may complain of neck pain by the end of the day because of overuse and fatigue. Numbness, tingling, paresthesia, and radicular pain should be investigated because these findings indicate irritation or injury of nerve roots.

**Physical examination:** Assess for spinous process tenderness; assess active range of motion. Do not attempt to forcibly move the neck if pain or muscle spasm occurs. Imaging studies are indicated only after significant trauma, spinous process tenderness, or neurologic findings.

**Treatment:** Cervical immobilization with hard cervical collar indicated when there is a concern for cervical spine injury associated with nerve injury; spinal immobilization in the absence of lower extremity neurologic signs is generally not recommended. Once significant cervical spine and nerve injury are ruled out by examination and/or imaging, treatment consists of pain control with analgesics, stretching, and mobilization techniques.

**Return to play:** Return to wrestling is allowed when there is painless neck range of motion and full strength on resisted neck muscle testing.

**Prevention**: Neck strengthening; adherence (and enforcement) of rules regarding safe return to the mat of a controlled wrestler

### Elbow Dislocation (Fig. 88.2)

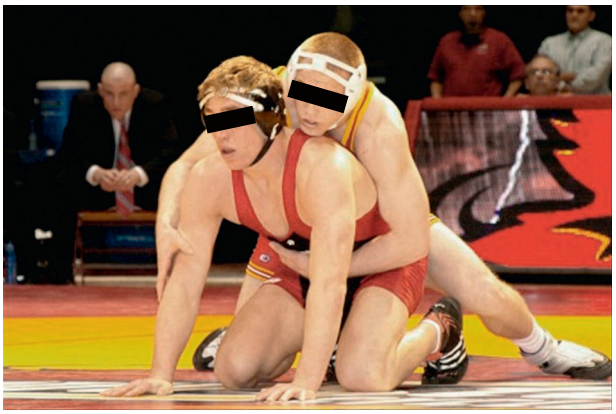
**Description:** Most elbow injuries in wrestling occur when a wrestler has been lifted off their feet then attempts to catch himself with an outstretched arm as he is returned to the mat. Approximately 80% of elbow dislocations are posterior and are associated with significant disruptions of the supporting ligaments; commonly associated with fractures

**Physical examination:** The elbow appears swollen with a disruption of the normal bony landmarks. Examine the hand for vascular or neural compromise by testing the pulses of the radial and ulnar arteries, capillary refill in the fingers, and sensation and motor functions of the radial, ulnar, and median nerves.

**Treatment:** Imaging of the elbow with radiographs should be performed as soon as possible due to the high association with fractures. Relocation may be attempted before radiographs if there is a sign of neurovascular compromise.

**Return to play:** Painless range of motion of elbow; full strength of elbow flexion/extension and forearm pronation/supination

Elbow injuries often occur while attempting to break a fall.



The "referee" position, also known as the *parterres* or *on-the-ground position.*

**Figure 88.2.** Elbow dislocation.

### Finger Dislocation

**Description:** The proximal interphalangeal joint is the most common dislocated finger joint. Medial-to-lateral plane dislocation: One or both of the collateral ligaments of the finger are damaged. Volar-to-dorsal plane dislocation: Volar capsular ligament is injured.

**Physical examination:** Neurovascular examination; assess for phalange shaft fracture

**Treatment:** Reduction can be attempted before radiographs if no bony tenderness or notable deformity of the phalanges; buddy tape or splint after reduction

**Return to play:** May return with buddy taping if no significant fracture or neurovascular injury

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# MIXED MARTIAL ARTS

# 89

Bryant Walrod

## GENERAL PRINCIPLES

### Overview

- The popularity of mixed martial arts (MMA) has grown tremendously in the last 20 years.
- Despite this incredible growth, there is still a paucity of information about the common injuries and proper ringside management of these athletes.
- Often, sports medicine physicians will be asked to cover an MMA competition.
- It can be overwhelming without any basic understanding of either the sport or the ringside considerations for the evaluation and management of MMA competitors.

### Pertinent History

- The Ultimate Fighting Championship is the largest organization in MMA.
  - It was founded in 1993 and has continued to grow in size and influence, having acquired former rivals such as World Extreme Cagefighting, Pride, and Strikeforce.
- Other organizations actively promoting MMA are Bellator, World Series of Fighting, One Fighting Championship, and Invicta FC.
- MMA is defined as a combative sport between two individuals occurring in a ring or cage that utilizes fighting techniques from a variety of martial arts, including jujitsu, wrestling, boxing, kickboxing and other martial arts.
- MMA must follow the Unified Rules of MMA, which were developed to give some standardization to the competition and to hopefully make the sport safer for its participants.
  - These rules mandate that there are two contestants in each match who are paired by promoters and approved by the state commissions.
  - Bouts will typically be three or five rounds, with championship bouts most often being scheduled for five rounds.
  - Each round is 3 or 5 minutes in duration.

### Terminology

- Bouts can be declared by KO (knockout), TKO (technical knockout), submission, referee stoppage, doctor stoppage, corner stoppage, disqualification, or a judge's decision.

  **KO:** A competitor is knocked down and deemed unconscious or disoriented.

  **TKO:** The referee stops the bout when one competitor is unable to intelligibly defend himself/herself.

  **Submission:** One of the fighters signals to the referee, via either a tap or a verbal cue, that he or she no longer wants to continue.

  **Referee Stoppage:** The referee calls an end to the competition for any reason other than a TKO.

  **Doctor Stoppage:** The ringside physician determines that it is unsafe for one of the combatants to continue due to an injury.

  **Corner Stoppage:** The combatant's corner "throws in the towel" to signify that they no longer want their competitor to continue fighting.

  **Disqualification:** One of the combatants is disqualified due to a rules violation.

  **Decision:** The specified number of rounds is successfully completed. The decision is based upon the ringside judges' scorecards.

### Rules

- There are considerable variations in the rules and ringside physician's responsibilities from state to state; it is critical to be familiar with the rules specific to the area in which the competition is held.
  - Clarification on specific state rules, as well as the Unified Rules of MMA, is provided by the Association of Boxing Commission (www.abcboxing.com).
- Most states will require that an individual obtain a separate license from the medical board to serve as a ringside physician.
- Although there is now a new additional certification from the American College of Sports Medicine/Association of Ringside Physicians, states do not mandate any proof of knowledge or experience to sit as a ringside physician. Most simply require an additional fee.

### Weight Classes

- MMA competitors are divided into seven weight classes:
  - Flyweight—up to 125 lbs
  - Bantamweight—over 125 to 135 lbs
  - Featherweight—over 135 to 145 lbs
  - Lightweight—over 145 to 155 lbs
  - Welterweight—over 155 to 170 lbs
  - Middleweight—over 170 to 185 lbs
  - Light Heavyweight—over 185 to 205 lbs
  - Heavyweight—over 205 to 265 lbs
  - Super Heavyweight—over 265 lbs

### Equipment and Safety Issues

- Competitors wear 4–6 oz open-finger gloves, which allow for greater punch velocity and lesser impact dissipation than 16 oz gloves (Fig. 89.1).
  - It is thought that these gloves do more to protect the hands of the striker and decrease the risk of lacerations.
- Competitors are also instructed to wear a protective cup and a mouth guard.
- Shoes and a "gi" are not permitted (Fig. 89.2).

### Regulation

- It is important for the covering ringside physicians to understand the rules and regulations that are specific to the state in which they practice.
- Currently, New York and Connecticut do not sanction MMA competitions.
- There can be variations in the prefight examination requirements from state to state.
- The ringside physician can consult the Association of Boxing Commission's website at the following address for specific state requirements: www.abcboxing.com/medical_requirements.html
- Specific state contacts can be found at the following address: www.abcboxing.com/commission_contacts.html

Figure 89.1. Gloves.

Figure 89.2. Mixed martial arts gi: this type of uniform is not permitted in the UFC.

## Prefight Coverage and Examination

- Most often, prefight physical examinations are completed the day prior to the competition at the time of weigh-ins. However, sometimes the logistics of coverage mandate that the examination be completed the day of the competition.
- When preparing to cover an MMA competition, it is important for the physician to review the contents of the medical bag.

## Unique Environmental and Nutritional Issues

- Dehydration is an important consideration during the prefight physical examination.
  - The Committee Report on Unified Rules of MMA states, "Athletes shall be examined at the prefight examination to screen for an excessive or extreme weight loss practices leading to dehydration."
  - There are no more specific guidelines that are stated on how to accomplish this, and specific practices are not defined; it also does not specify who is charged with doing this screening.
- There are no programs in place like in college wrestling to prevent and monitor excessive weight loss/dehydration.
- There is no section on the prefight physical examination addressing fluid status and weight loss.

**BOX 89.1 HYDRATION STUDY IN MMA FIGHTERS***

- Body mass increased 4.4% in the 22-hour period before the competition.
- Similar to a body mass increase in college wrestlers before new rule implementation (4.9%).
- After the rule change, college wrestlers now have 1.2% weight gain.
- 39% significantly dehydrated 2 hours before event (USG >1.021)
- 11% seriously dehydrated (USG >1.030)
- 23% well hydrated (USG <1.010)
- It is yet to be determined if these findings will influence rule changes with respect to hydration and weight loss.

*Fighters were weighed at the official weigh-in (24 hours before the bout) and then re-weighed about 22 hours later (2 hours before the bout). Fighters provided a urine sample at each of these times and had a skin fold analysis to determine body density during the official weigh-in.
Data from Jetton AM, Lawrence MM, Meucci M, et al. Dehydration and acute weight gain in mixed martial arts fighters before competition. *J Strength Cond Res*. 2013;27(5):1322-1326.

- Same day weigh-ins are difficult in MMA because of the potential financial losses from pay-per-view audience reduction if an individual does not make weight and is thus removed from the fight card the day of the fight.
- See Box 89.1 for the hydration study in MMA fighters.

## Day-of-Fight Considerations, Match Coverage, and Injury Patterns

- Important considerations for an Emergency Action Plan:
  - The physician needs to ensure that they have a reserved a seat ringside with an unobstructed view and easy access to the ring if they are called to enter.
  - Medical team: If you do cover a competition by yourself, it may need to be temporarily suspended if you need to attend to an injured fighter. This delay may not be acceptable to ringside fans or the television audience.
  - It is imperative for the physician to introduce themselves to the EMS team and cover specific responsibilities.
  - If the physician provides laceration repair or intravenous fluid replacement therapy, it is important to arrange for appropriate follow up after the competition.
  - Where is the nearest hospital?
- It is important to meet with the referee before the competition to review responsibilities and to provide an opportunity to ask any questions.
- During the competition, the physician may be asked to enter the ring, but one may not do so unless summoned by the referee.
  - If the physician is asked to enter the ring during the competition, the physician may examine the fighter but not treat him/her.
  - For example, the physician can wipe away blood to examine a cut, but one may not actively treat the laceration.
  - The physician can only determine if the fighter is able to continue or if he/she should be disqualified for medical reasons.
- It is important to take control of the situation.
  - Often, the competitor's corner men will attempt to enter the ring to assist their teammate who is down.
  - This action must be clearly prohibited as moving an injured fighter carries risk if not paying attention to potential cervical spine precautions.
- The establishment of a unified team approach, where each individual clearly knows his/her responsibilities, is paramount to the maintenance of fighter safety.

### Laceration Management

- Some teams and/or promoters will provide a "cutman" for the competitors.
  - The experience of cutmen varies greatly.
  - Cutmen do not replace a physician's medical expertise with respect to deciding whether or not a fighter is qualified to continue or whether or not a fighter should be disqualified due to a laceration.
- If the physician is asked to tend to a laceration, the physician may only treat the laceration in-between rounds.
  - Applying pressure with sterile gauze or a large cotton swab is the best first-line treatment.
  - Also consider petroleum jelly.
  - The only substances that are permitted for administration are a solution of adrenaline 1:1000, Avetine, or Thrombin.
  - An enswell is another helpful instrument to have in your medical bag (Fig. 89.3); it is a frozen piece of steel that can help reduce swelling and contusions on the face or around the eyes.
- The presence of periorbital lacerations or contusions that obstruct vision form potential grounds to disqualify a fighter.
- Epistaxis is also common.
  - Application of direct pressure is typically effective; also, consider petroleum jelly.
- Observe for presence of septal hematoma or nasal fracture (Fig. 89.4)

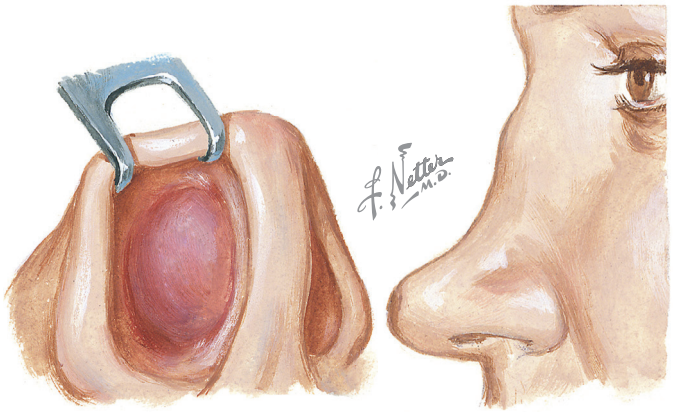
## COMMON INJURIES AND MEDICAL PROBLEMS

- For a video analysis of 642 MMA matches, see Box 89.2.
- For a retrospective review of ringside physician reports from 171 Matches, see Box 89.3.
- For a retrospective review of 635 MMA matches, see Box 89.4.
- For a retrospective review of injury trends while training for MMA events, see Box 89.5.
- For data from 711 MMA bouts (2178 rounds of exposure), see Box 89.6 and Table 89.1.
- For data the Nevada Athletic Commission obtained from UFC fights, see Box 89.7.

### Head Trauma

For video records of 844 UFC matches for the identification of the principle mechanisms and situational factors associated with KOs and TKOs secondary to repetitive strikes, see Box 89.8.

**Figure 89.3.** Enswell.




Examination for septal hematoma.

Cartilaginous deformity secondary to untreated septal hematoma.

**Figure 89.4.** Nasal fracture.

### BOX 89.2 VIDEO ANALYSIS OF 642 MMA MATCHES

- 72% ended secondary to injury.
- 28% of competitors suffered closed head trauma.
- Severity of injuries and concussion rate were difficult to ascertain secondary to video analysis.

Data from Buse GJ. No holds barred sport fighting: a 10 year review of mixed martial arts competition. *Br J Sports Med.* 2006;40(2):169-172.

### BOX 89.3 RETROSPECTIVE REVIEW OF RINGSIDE PHYSICIAN REPORTS FROM 171 MATCHES

- Overall injury rate: 28.6/100 participants
- Facial lacerations: 47.9%
- Hand injuries: 13.5%
- Nose injuries: 10.9%
- Eye injuries: 8.3%
- Risk of injury increased with:
  - Age
  - Losing a match
  - Losing a match by KO or TKO
  - Match going into rounds 4 or 5
- Outcome of match:
  - TKO: 39.8%
  - Tap out: 30.4%
  - Decision: 18.1%
  - KO: 6.4%
  - Choke: 2.3%
  - Disqualification: 1.8%
  - Draw: 1.2%

*KO,* Knockout; *TKO,* technical knockout.
Data from Bledsoe GH. Mixed martial arts. *Combat Sports Medicine*. 2008;323-330.

### BOX 89.4 RETROSPECTIVE REVIEW OF 635 MMA MATCHES

- Injury of 23.6/100 fight participants
- Lacerations and upper extremity injuries were the most common.
- Age, weight and fight experience had no impact on injury rate.
- The losing fighter was 2.53 times more likely to be injured.

Data from Ngai KM, Levy F, Hsu EB. Injury trends in sanctioned mixed martial arts competition: a 5-year review from 2002 to 2007. *Br J Sports Med.* 2008;42(8):686-89.

## POSTFIGHT ANALYSIS AND CONSIDERATIONS

- Ringside physicians must be proficient in basic life support, cervical spine evaluation, and assisting with c-spine immobilization and transport.
- He/she should also able to provide care for contusions and basic lacerations.
  - Certain lacerations may be referred out based upon the individual physician's comfort.
  - Typically, lacerations involving the medial aspect of the eye and those that cross the vermillion border of the lip should be referred for subspecialty care.
- Knowledge of basic fracture/dislocation and splint care is critical.
- Finger and elbow dislocations (Figs. 50.7, 51.4, 51.5, 51.8) from striking and arm bars are common.
- An attempt to reduce the joint should be performed ringside with subsequent postreduction films.
- Fractures of the metacarpals, carpals, metatarsals, tarsals, and orbital floor also occur commonly.

### Concussion Management

It is imperative to understand concussion management (see Chapter 45, Head Injuries); the official statement with respect to concussions from the unified rules of MMA is:

*"Before allowing a fight to continue, the Referee should consult with the Ringside Physician in all cases involving concussive head fouls. The Referee, in conjunction with the Ringside Physician, will determine the length of time needed to evaluate the affected athlete and his or her suitability to continue."*

### BOX 89.5 RETROSPECTIVE REVIEW OF INJURY TRENDS WHILE TRAINING FOR MMA EVENTS

- Males were more likely to be injured.
- 78% occurred in training; 22% occurred in competition
- The most common areas to be injured were:
  - Head and neck: 38%
  - Lower and upper extremities: 30% and 22%, respectively
- The most common injuries were:
  - Contusions: 29%
  - Miscellaneous strains: 16%
  - Concussions: 1.8%
- MMA styles associated with the highest injury rates in descending order:
  - Jujitsu, freestyle wrestling, wrestling, and then kickboxing
- A lower belt rank correlated with an increased risk of injury.
- Professional fighters were three times more likely to get injured than amateur fighters.

Data from Rainey CE. Determining the prevalence and assessing the severity of injuries in mixed martial arts athletes. *N Am J Sports Phys Ther*. 2009;4(4):190-99.

### BOX 89.6 DATA FROM 711 MMA BOUTS (2,178 ROUNDS OF EXPOSURE)

The most common injuries were:
- Lacerations and abrasions: 38%
- Altered mental state: 21.5%
- Fractures: 16.5%.

Injuries were more common in:
- Professionals vs. amateurs
- Later rounds of the fight
- Bouts ending in KO/TKO

Injuries were less likely in bouts that ended in submission, decision, or disqualification compared to a KO/TKO.

The rate of injury in bouts ending in strikes was 9.1%, whereas the rate of injury in bouts ending by decision, choke, or lock was 4.0%.

A diagnosis of altered mental status was made in:
- 0.2% of bouts ended by submission, decision, or disqualification
- 4.2% of bouts decided by KO/TKO

A diagnosis of altered mental status was made in:
- 3.5% of bouts ended by strikes
- 0.2% of bouts ended by submission, choke, or lock

*KO,* Knockout; *TKO,* technical knockout.
Data from McClain R. Injury profile of mixed martial arts competitors. *Clin J Sport Med.* 2014;24(6):497-501.

### BOX 89.7 DATA FROM THE NEVADA ATHLETIC COMMISSION OBTAINED FROM UFC FIGHTS

- 60% reported no injury
- 40% complained of injury
- Overall injury rate: 39.7/100
- The face, as well as the lower and upper extremities, was the most common location of injury.
- The types of injuries recorded were:
  - Facial laceration/soft tissue: 19.2%
  - Facial fractures: 4.6%
  - Eye injury: 2%
  - Knee, shoulder, and hand injury: 1%
- 10% of fighters were sent to the ED or were instructed to obtain x-rays.
  - 11% had CT scans
  - 52% were negative
  - 38% revealed a new fracture (facial bones)
  - 1% soft tissue injury
  - No CT scan revealed any intracranial soft tissue or vascular abnormalities.
- An increased injury rate was observed for losses via tap out by arm bar, TKO, or a decision.
- A loss via KO/TKO increased the risk of having a facial fracture 20-fold.

*KO,* Knockout; *TKO,* technical knockout.
Data from Otten MH. Ultimate Fighting Championship injuries: a two-year retrospective fight injury study. *Osteopath Family Physician*. 2015;7:13-18.

### TABLE 89.1 REASONS FOR ENDING A BOUT BY GENDER AND LEVEL

| | KO/TKO | Submission | Decision | Referee Stoppage |
|---|---|---|---|---|
| **Overall Bouts** | 29% | 55.3% | 14.9% | 0.7% |
| **Men** | **28.6%** | **56.6%** | **14.0%** | **0.8%** |
| **Women** | 35.2% | 38.9% | 25.9% | 0.4% |
| **Amateur** | 29.5% | 58.1% | 12.1% | 11.9% |
| **Professional** | 27.7% | 45.2% | 25.2% | 1.0% |

*KO,* Knockout; *TKO,* technical knockout.
Data from McClain R. Injury profile of mixed martial arts competitors. *Clin J Sport Med*. 2014;24(6):497-501.

### BOX 89.8 VIDEO RECORDS OF 844 UFC MATCHES FOR THE IDENTIFICATION OF THE PRINCIPLE MECHANISMS AND SITUATIONAL FACTORS ASSOCIATED WITH KOs AND TKOs SECONDARY TO REPETITIVE STRIKES*

With respect to matches:
- 55% ended before their scheduled time
- 12.8% ended in KO
- 21.2% ended in TKO
- 90% ending in TKO were the result of repetitive strikes

They also found:
- Incidence of KO: 6.4/100 athlete exposures (AE)
- Incidence of TKO: 9.5/100 AE

Increased risk of KO was associated with:
- Previous KO/TKO: odds risk (OR) 1.30
- Age >35 years: OR 1.94

Decreased risk of KO was associated with:
- Middleweight class: OR 0.44
- Undercard match: OR 0.51
- Each additional minute of the round: OR 0.69
- Additional rounds: OR 0.36

Increased risk of TKO was associated with:
- Heavyweight class: OR 2.12
- Age >35 years: OR 1.96

Decreased risk of KO was associated with:
- Each minute of competition: OR 0.76
- Each additional round: OR 0.64

In KOs:
- The fist was the striking implement in 84% of the KOs.
- The head was the body part struck in all KOs.
- The most common region of head to be struck:
  - Mandible: 54%
  - Maxillary/temporal regions: 20% each
- 63% of those who lost by KO sustained a secondary head impact with the fighting surface (floor, cage, or post).
- The average time between KO strike and match stoppage was 3.5 ± 2.8 seconds; during that time, the competitors sustained an average of 2.6 ± 3.0 (range 0-20) additional strikes to the head.
- In the 30 seconds preceding the KO strike, the losing competitor sustained an average of 6.2 ± 7.3 strikes (range 0-35), with 82% of these strikes being to the head.

With respect to TKOs:
- The losing competitor sustained and average of 18.5 ± 8.8 strikes (range, 5–46) in the 30 seconds preceding TKO, with 92% of these strikes being to the head.

*It is critical that the ringside physician be aware of these findings and the significant head trauma that occurs in the moments preceding a KO/TKO. This should influence postfight evaluations and return to competition considerations.

Data from Hutchenson MG. Head trauma in mixed martial arts. *Am J Sport Med*. 2014;42(6):1-7.

### BOX 89.9 RETROSPECTIVE SELF-REPORTED SURVEY ABOUT MMA TRAINING REGIMENS AND MEDICAL RELATED ISSUES FROM 119 INDIVIDUALS CURRENTLY TRAINING IN MMA

- 15% reported a KO
- 28% reported a TKO
- 13% of those reporting concussion-like symptoms during training sought medical attention.
- 60% with postconcussive symptoms returned to training in 2 days or less.
- This is an area of MMA ringside medicine that certainly must be addressed for the improvement of fighter safety.
- There must be improved regulations, especially with respect to concussion management, affecting return to play considerations after MMA competition to ensure that fighter safety is paramount.

Data from Heath CJ, Callahan JL. Self-reported concussion symptoms and training routines in mixed-martial-arts athletes. *Int J Res Sports Med*. 2013;21(3):195-203.

MANDATORY TIME OFF—UNIFIED RULES OF MMA

- **Technical knock out:** Any fighter losing by way of a TKO resulting from head blows shall receive a medical suspension and shall not participate in any boxing activity for a minimum of 30 days.
- **Knock out**: A fighter losing by way of a KO shall receive a medical suspension and shall not participate in any boxing activity for a minimum period of 60 days. At the discretion of the physician, longer suspension periods may be issued for either the TKO or KO.
- Boxers shall receive a mandatory 7-day rest period after competing in an event.
  - Day 1 of the mandatory rest period shall commence on the first day following the event.
- A physician may issue a medical suspension any time he/she believes it to be in the best interest for the safety of a boxer (i.e., high blood pressure at the prefight physical examination).
- In any/all cases, the decision by the physician to issue or extend a suspension is final.
- Returning to practice and competition can be logistically difficult for a fighter.
- If you are the covering ringside physician, you can provide recommendations with respect to return to practice. However, this can be difficult to enforce.
- As a ringside physician, one typically only covers the event, and follow-up is difficult if the competitors are from different cities and states.
- MMA fighters do not typically have a "team physician" who can administer a baseline or a postfight SCAT 3, follow daily symptom scores, or oversee return to play considerations.
- The gym owner and promoter are not qualified to make these recommendations, and often, the fighter will return with no real guidance, which can put him/her at risk of injury.
- For a retrospective self-reported survey about MMA training regimens and medical related issues from 119 individuals currently training in MMA, see Box 89.9.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:
- eAppendix 89-1 Scoring
- eAppendix 89-2 Prohibited actions
- eAppendix 89-3 State of Ohio prefight physical examination requirements
- eAppendix 89-4 Medical bag

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

# eBOOK SUPPLEMENTS

## eAppendix 89-1 Scoring

### Scoring Criteria

- The following objective scoring criteria shall be utilized by the judges when scoring a round:
  1. A round is to be scored as a 10-10 round when both contestants appear to be fighting evenly and neither contestant shows dominance in a round.
  2. A round is to be scored as a 10-9 round when a contestant wins by a close margin, landing the greater number of effective legal strikes, grappling and other maneuvers.
  3. A round is to be scored as a 10-8 round when a contestant overwhelmingly dominates by striking or grappling in a round.
  4. A round is to be scored as a 10-7 round when a contestant totally dominates by striking or grappling in a round.

### Scoring System

- All bouts will be evaluated and scored by three judges.
- The 10-Point Must System will be the standard system of scoring a bout.
- 10 points must be awarded to the winner of the round and nine points or less must be awarded to the loser, except for an even round, which is scored 10-10.
- Judges shall evaluate mixed martial arts techniques:
  - Effective striking
  - Effective grappling
  - Control of the fighting area
  - Effective aggressiveness and defense

## eAppendix 89-2 Prohibited Actions

These may vary from state to state, but the following are actions that are typically prohibited in MMA:

- Head butting
- Eye gouging
- Hair pulling
- Biting
- Fish hooking
- Attacking the groin
- Strikes to the back of the head and spinal area
- Strikes to or grabs of the trachea
- Small joint manipulation
- Intentionally throwing your opponent out of the ring
- Running out of the ring/cage
- Purposely holding the ring ropes or cage fence
- Grabbing or putting a hand inside the trunks or gloves of the opponent
- Pulling or holding onto an opponent's trunks or gloves

## eAppendix 89-3 State of Ohio Prefight Physical Examination Requirements

A list from the state of Ohio to give a general understanding of the specific prefight physical examination requirements for MMA, boxing, kickboxing, and all other types of martial arts sports. However, it is again recommended to consult your specific state for necessary prefight examination requirements.

1. Blood work: HIV, Hepatitis B Surface Antigen, Hepatitis C Antibody. Fighters 39 years of age and over need to also submit a Metabolic Blood Profile. Blood work must be submitted within 30 days of being taken. Blood work is valid for the lifespan of the license.
2. Physical: Physicals are performed at the weigh-in.
3. Eye examination: Fighters must submit a dilated ophthalmological examination administered within the last year. This examination must be performed by a licensed ophthalmologist or optometrist.
4. EKG: Applicants 39 years of age and over must submit a stress EKG with cardiology clearance. This test is valid for 1 year.
5. Radiological examinations: Applicants 35 years of age and over must submit an MRI of the brain without contrast. In addition to the MRI, fighters 39 years of age and over need to submit a chest x-ray and a MRA. The MRI and MRA are valid for 5 years. The chest x-ray is valid for 2 years.
6. Neurological examination: Fighters who have had multiple losses, KOs, TKOs, and fighters 35 and over need to submit a neurologic examination performed by a licensed MD or DO. The neurologic examination is valid for 5 years.
7. Urinalysis: Not conducted at this time
8. Female fighters: Female fighters are given a pregnancy test at the weigh-in.
9. Older fighters: Fighters 35 years of age and over need to submit an MRI and a neurologic examination administered within the last 30 days. Fighters 39 years and over also need to submit and MRI/MRA, EKG with echo cardio clearance, metabolic blood profile, and a chest x-ray within the last 2 years.
10. Additional requirements: None at this time

## eAppendix 89-4 Medical Bag Contents

- Oral airway
- Sphygomanometer
- Automated External Defibrillator (AED)
- ET tube
- Ambu bag
- 1% Lidocaine w/ Epi
- Backboard
- Cervical collar
- 1% Lidocaine w/o Epi
- Gloves
- Suture kits
- Epipen
- 4x4s, 2x2s
- Coban
- Sharps container
- Athletic tape
- Nasal plugs
- Albuterol
- Dental rolls
- Splinting material
- Glucagon/glucose tabs
- Finger splint
- Ophthalmoscope
- Tooth saver
- Otoscope
- Stethoscope
- Benadryl
- Syringes
- 18-, 25-gauge needles
- Eye wash
- SCAT 3
- Written instructions
- Saline solution
- Trauma scissors
- Water hand sanitizer
- Pregnancy test kit

# RECOMMENDED READINGS

1. Bledsoe GH. Mixed martial arts. *Combat Sports Medicine*. 2008; 323-330.

2. Buse GJ. No holds barred sport fighting: a 10 year review of mixed martial arts competition. *Br J Sports Med*. 2006;40(2):169-172.
3. Heath CJ, Callahan JL. Self-reported concussion symptoms and training routines in mixed-martial-arts athletes. *Int J Res Sports Med*. 2013;21(3):195-203.
4. Hutchenson MG, Lawrence DW, Cusimano MD, Schweizer TA. Head trauma in mixed martial arts. *Am J Sport Med*. 2014;42(6):1-7.
5. Jetton AM, Lawrence MM, Meucci M, et al. Dehydration and acute weight gain in mixed martial arts fighters before competition. *J Strength Cond Res*. 2013;27(5):1322-1326.
6. Lystad RP, Gregory K, Wilson J. The epidemiology of injuries in mixed martial arts: a systematic review and meta-analysis. *Orthop Sports Med*. 2014;2(1).
7. McClain R. Injury profile of mixed martial arts competitors. *Clin J Sport Med*. 2014;24(6):497-501.
8. Ngai KM, Levy F, Hsu EB. Injury trends in sanctioned mixed martial arts competition: a 5-year review from 2002 to 2007. *Br J Sports Med*. 2008;42(8):686-689.
9. Otten MH, Ghazarian N, Boura J. Ultimate Fighting Championship injuries: a two-year retrospective fight injury study. *Osteopath Family Physician*. 2015;7:13-18.
10. Rainey CE. Determining the prevalence and assessing the severity of injuries in mixed martial arts athletes. *N Am J Sports Phys Ther*. 2009;4(4):190-199.

# 90 GYMNASTICS

Amy Jo F. Overlin

## INTRODUCTION

- Types: acrobatic, artistic, rhythmic, tumbling, and trampoline (Tables 90.1 to 90.4)
- Over 97,000 competitive gymnasts register yearly with USA Gymnastics, and up to 5 million recreational gymnasts in the United States.
- >70% female artistic gymnasts

## EPIDEMIOLOGY

- Injury rates: vary greatly depending on the level of the gymnast and hours spent training
  - 0.687–2.859 injuries per 1000 hours of training
  - 6.07–9.22 injuries per 1000 athletic exposures in collegiate athletes
- Higher incidence during dismounts and floor exercise
- Contributing factors:
  - Poor landing technique including landing short
  - Landing with overly upright posture, decreased knee flexion, and relative joint stiffness
- Sprains most common, followed by strains
- The ankle/foot is the most commonly injured body part, except in men and acrobatics (where it is the hand/wrist)
- The incidence of injury during competition approximately two times the practice incidence.
- Higher incidence of growth plate injuries owing to an immature skeletal system
- Injury risk factors:
  - Larger size
  - Rapid growth
  - Training for >15–20 hours/week
  - Life stress
- Gymnasts are both lower and upper extremity weight-bearing athletes; therefore, injuries incurred during gymnastics participation are comprehensive (see Table 90.5).

## COMMON INJURIES AND MEDICAL PROBLEMS

### Mild Traumatic Brain Injury (MTBI)

**Mechanism of injury:** Hitting the head on the mat/floor or apparatus during a fall or dismount
**Incidence:** A study found a 30% lifelong occurrence
**Return to play:** Several activities are aerial in nature; hence, the graduated return protocol will have to be modified to meet the demands of the gymnast while maintaining their safety until the athlete has been fully cleared.

### Cervical Spine Fracture, Subluxation, and Dislocation

**Mechanism of injury:** Complex aerial and acrobatic nature of gymnastics places athletes at a risk of catastrophic neck injuries. Cervical spine fractures, subluxations, and dislocations can occur through various mechanisms:
- Landing head first in a loose foam pit, on a trampoline, or on a mat
- Failure to complete rotation or over-rotating on aerial or salto maneuver.
- Landing on the upper back with the neck in a hyperflexed position
- Landing on the chin or chest with the neck in a hyperextended position

**Specific consideration to standard evaluation and treatment:**
- Pediatric cervical spine collar availability
- Loose foam pit injuries:
  - Foam blocks that fill the pit are easily disturbed, and the athlete is typically buried in the blocks.
  - Avoid jumping into pit to help an injured athlete because the disruption of foam blocks could worsen the injury and make it more difficult to remove the athlete.
  - Considering the difficulty of removing a gymnast with a cervical spine injury from a loose foam pit, physicians, trainers, coaches, and local paramedics should practice emergency removal as part of an emergency action plan.
  - Gently placing a mat into the pit and then using this as a means to reach the athlete is one method to minimize disturbing the foam blocks.

### Shoulder Injuries

#### Anterior Dislocation, Labral Tears, and Multidirectional Instability

See Chapter 49: Shoulder Injuries.

#### Rotator Cuff Syndrome, Impingement, and Tears

- More common in male gymnasts
- Rings, high bar, and parallel bars all put substantially increased stress on the shoulder.

### Elbow Injuries

#### Dislocation

- Upper extremity weight-bearing activities can be gradually introduced, once a gymnast has full range of motion (ROM) and strength in the upper extremity and is pain free.

#### Ulnar Collateral Ligament (UCL) Sprain

**Mechanism of injury:** Valgus stress to the medial aspect of the elbow causes traction injury to the UCL; may occur acutely due to a fall on an outstretched hand or chronically due to repetitive upper extremity weight bearing.
**History:** Valgus mechanism; may be acute or chronic
**Physical examination:** Findings typical of UCL injuries; evaluate for an increased carrying angle and elbow hyperextension bilaterally, which may be a risk factor for this type of injury
**Imaging:** Radiographs: Check for medial epidondylar apophyseal avulsion fracture or chronic changes consistent with medial epicondylar apophysitis. Magnetic resonance imaging (MRI) arthrogram may be needed to determine the degree of ligamentous tear.
**Treatment:** Surgery is reserved for complete rupture of UCL with resultant chronic instability.
**Complications:** Chronic instability, ulnar neuritis

#### Capitellar Osteochondritis Dissecans (OCD)

**Mechanism of injury:** Repetitive weight bearing causes valgus stress with medial elbow tension and lateral radiocapitellar joint compression.
**History:** Gradual onset, elbow pain with weight-bearing activities; pain relieved by rest; decreased elbow extension; in more

## TABLE 90.1 ACROBATIC GYMNASTICS (MEN AND WOMEN)

| Events | Levels | |
|---|---|---|
| Women's pairs | **Junior Olympic** | **Elite** |
| Men's pairs | Levels 1–10 | Junior |
| Mixed pairs | Competitive levels: 4–10 | Senior |
| Women's group | First competitive level: 4 | |
| Men's group | | |

See www.usa-gymnastics.org.

## TABLE 90.2 ARTISTIC GYMNASTICS

| | Women | Men |
|---|---|---|
| **Events** | Vault<br>Uneven parallel bars<br>Balance beam<br>Floor exercise | Floor exercise<br>Pommel horse<br>Still rings<br>Vault<br>Parallel bars<br>High bar |
| **Levels** | Junior Olympic Program<br>Levels 1–10<br>Competitive levels 2–10<br>First competitive level: 2<br>Elite Program<br>Talent Opportunity Program (TOPS)<br>HOPES (10–12-year-old pre-elite)<br>Junior Pre-Elite<br>Junior and Senior International | Junior Olympic Program<br>Levels I–X<br>Competitive levels: IV–X<br>First competitive level: IV<br>Elite Program<br>Future Stars Program<br>Junior National Team<br>Senior Elite Team |

## TABLE 90.3 RHYTHMIC GYMNASTICS (WOMEN ONLY)

| Events | Levels | |
|---|---|---|
| Rope | **Junior Olympic** | **Elite** |
| Hoop | Levels 1–8 | Junior |
| Ball | Competitive levels: 5–8 | Senior |
| Clubs<br>Ribbon | First competitive level: 5<br>Individual and group competitions | Individual and group competitions |

## TABLE 90.4 TUMBLING AND TRAMPOLINE (MEN AND WOMEN)

| Events | Levels | |
|---|---|---|
| | **Junior Olympic** | **Elite** |
| Double minitrampoline<br>Synchronized trampoline<br>Trampoline<br>Tumbling | Levels 1–10<br>Competitive levels: 1–10<br>First competitive level: 1 (except synchronized trampoline: level 10) | Junior<br>Senior |

## TABLE 90.5 DIFFERENTIAL DIAGNOSIS OF GYMNASTICS INJURIES

| | |
|---|---|
| **Lower Extremity Injuries** | |
| **Foot** | Calcaneal Apophysitis<br>Calcaneal Fat Pad Contusion<br>Calcaneal Stress Fracture<br>Lisfranc Injury<br>Stress Fractures: Navicular and Metatarsals<br>Turf Toe |
| **Ankle** | Anterior and Posterior Ankle Impingement<br>Distal Fibular Salter-Harris I Fracture<br>Ankle Sprains: High, Lateral, and Medial<br>Os Trigonum Fracture<br>OCD of the Talar Dome<br>Osteochondritis Dessicans-Talus<br>Posterior Tibialis Tenosynovitis |
| **Knee** | ACL Tear<br>MCL/LCL Sprain/Tear<br>Meniscal Injuries<br>Osgood–Schlatter Syndrome<br>Osteochondritis Dessicans of the MFC<br>Patellofemoral Syndrome<br>Patellar Subluxation/Dislocation |
| **Hip** | Acetabular Labral Tear<br>Apophysitis<br>Femoral Acetabular Impingement<br>Femoral Stress Fracture<br>Hip Instability/Hypermobility |
| **Upper Extremity Injuries** | |
| **Hand/Wrist** | Fractures related to grip lock<br>Ganglion Cysts<br>Gymnast Wrist<br>Rips<br>Scaphoid Fractures/Stress Fractures<br>Scaphoid Impaction Syndrome<br>TFCC Tears |
| **Elbow** | Elbow Dislocations<br>Medial Epicondyle Apophysitis<br>Medial Epicondyle Avulsion Fractures<br>Osteochondritis Dessicans of the Capitellum<br>Ulnar Collateral Ligament Injuries |
| **Shoulder** | Impingement Syndrome<br>Labral Tears<br>Multidirectional Instability<br>Rotator Cuff Strain/Tears<br>Shoulder Dislocations/Subluxations |
| **Other** | |
| **Head** | Concussions |
| **Cervical Spine** | Cervical Fractures<br>Cervical Strain |
| **Lumbar Spine** | Discogenic Back Pain<br>Facet Syndrome<br>Lumbar Strain<br>Mechanical Lower Back Pain<br>Sacroilitis<br>Scheuermann Disease<br>Spondylolisthesis<br>Spondylolysis |

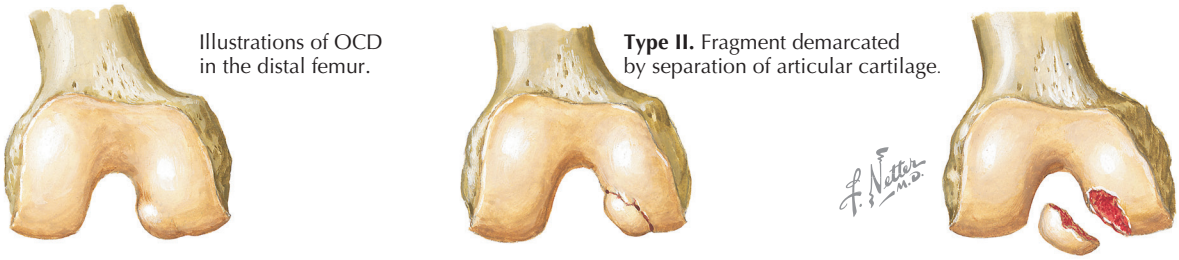



**Figure 90.1.** Capitellar Osteochondritis Dissecans.

advanced cases, mechanical symptoms of catching and locking noted

**Physical examination:** Tenderness to palpation over radiocapitellar joint; effusion may be present; ROM, particularly extension, may be decreased.

**Imaging:** Radiographs: If positive will show a radiolucency or fragmentation within the capitellum, with irregular ossification and crater next to articular surface; MRI arthrogram helps determine integrity of articular cartilage, or if radiographs are negative and there is a high clinical suspicion.

**Classification and treatment of OCD lesions** (Fig. 90.1)**:**

- **Type I:** No displacement of lesion or fracture of the articular cartilage
  - Treatment: Conservative; no upper extremity weight-bearing or strengthening activities until radiographs show evidence of healing and pain resolves completely; consider splint if pain not relieved by discontinuing upper extremity weight-bearing activities or to improve compliance
- **Type II:** Evidence of fracture of articular cartilage or partial displacement of lesion
  - Treatment: Controversial; ranges from conservative to surgical intervention
- **Type III:** Complete detachment of lesion with resulting loose body
  - Treatment: Typically, surgical

**Complications:** Loss of ROM, degenerative changes, and chronic pain; can be career ending; imperative to identify these early in order to minimize complications; 40% of such lesions in female gymnasts noted to be bilateral, highly consider bilateral evaluation given the incidence

### Grip Lock

**Mechanism of injury:** When gymnast performs circling elements on uneven bars or horizontal bar, overlapping of leather grip against itself causes grip to lock or catch in place. Instead of allowing gymnast to circle freely around bar, hand is stuck in position as body continues to swing, causing forearm to "wrap around the bar" and fracture.

**Evaluation and treatment:** Appropriate for type of fracture(s)

**Prevention:** Regular replacement of grips as leather stretches out over time; gymnasts or coaches should make routine checks to ensure that grips are not long enough to overlap around bar. Avoid sharing of grips between gymnasts. Use caution if gymnast has been training on larger-diameter bar and then switches to smaller-diameter bar.

**TABLE 90.6 RADIOGRAPHIC FINDINGS CONSISTENT WITH DISTAL RADIAL PHYSEAL STRESS INJURY**

| Radiograph | Magnetic Resonance Image |
|---|---|
| Growth plate widening | Growth plate widening |
| Haziness within physis | Lack of physeal homogeneity |
| Cystic changes on metaphysis | Physeal cartilage extension |
| Breaking of epiphysis | Metaphyseal "bone bruise"<br>Linear striations |

## Wrist and Hand Injuries

### Distal Radial Physeal Stress Injury (Gymnast Wrist)

**Definition:** Stress injury to distal radial physis

**Mechanism of injury:** Repetitive overuse of wrist as a weight-bearing joint; forces between two to six times the athlete's body weight can load wrist joint during gymnastic maneuvers

**History and physical examination:** Tenderness over distal radial physis, pain reproduced with wrist hyperextension and axial loading, wrist extension may be decreased

**Imaging:** Radiographic and MRI findings suggest chronic stress injury to physis (Table 90.6). Recommend radiographs of contralateral wrist to compare physes for subtle differences. Initial radiographs may be negative. Consider MRI for a more detailed evaluation of physis.

**Treatment:**

- Decrease or eliminate upper extremity weight-bearing activities until pain resolves, which may take up to 12 weeks.
- Casting or bracing may be considered, particularly if the athlete experiences pain outside of gymnastic activities.
- Wrist brace worn during practice can often serve as a "reminder" for both the gymnast and coach to avoid weight-bearing activities.

- Physical therapy to address any flexibility and strength deficits of wrist and finger extensors and flexors; include upper extremity kinetic chain evaluation and therapeutic exercises to correct additional deficits.
- Reintroduce weight-bearing activities gradually once pain subsides.
- Coach should review and correct poor technique.
- Specialized wrist splints (e.g., Ezy Pro brace, Lion-Paws, or Teurlings wrist brace) may be recommended on returning to gymnastics.
- The Ezy Pro wrist brace has been shown in a cadaver study to help decrease wrist hyperextension and ulnocarpal joint forces when an axial load is applied to a pronated and extended wrist.

**Complications:**

- Chronic wrist pain
- Abnormal closure of physis may result in positive ulnar variance
- Triangular fibrocartilaginous complex tears
- Degenerative changes in triquetrum, lunate, and ulna
- Alterations in radioulnar articulation
- Extensor tendon ruptures

### Scaphoid Stress Fractures

**Mechanism of injury:** Repetitive upper extremity weight bearing causing hyperextension and abduction of wrist, leading to increased forces across the radioscaphoid articulation

**History and physical examination:** Chronic radial-sided wrist pain, worse with upper extremity weight-bearing activities; tenderness to palpation over the dorsum of scaphoid or anatomic snuff box

**Imaging:** Radiograph may show sclerosis of scaphoid waist, a fracture, or can be negative. MRI shows edema of the scaphoid consistent with a stress reaction with or without a definitive fracture line.

**Treatment:** Thumb spica cast for 8–12 weeks; after cast removal and physical therapy, begin a gradual return to upper extremity weight-bearing activities. Specialized wrist splints: see Gymnast Wrist above for details. Depending on clinical presentation and/or failure of conservative treatment, an open reduction and internal fixation (ORIF) to place a screw across the scaphoid may be warranted.

### Calluses, Blisters, and "Rips"

**Mechanism of injury:** Several events in gymnastics require hanging, swinging, and support movements on hands while gripping apparatus. Friction between hands and apparatus causes soft-tissue injuries to hands. Even with use of leather grips and chalk to decrease friction, skin develops calluses in response to repetitive forces. Calluses can tear ("rips") or blister.

**Treatment:** Most important principle of treatment is to avoid infection. Torn skin from "rip" should be trimmed away and the area cleaned with antibacterial soap and water. Appropriate protective padding and wrapping should be applied to prevent irritation and infection until the athlete can tolerate friction in area. Consider using DuoDerm Extra Thin CGF Spot Dressing:

- Apply to area of rip
- Athlete may attempt to swing bars with dressing in place, held on with athletic tape under grips.
- Change dressing after each bars practice or every 7 days if bars are not being practiced
- Drawback: treatment can be expensive

**Prevention:** Calluses should be shaved down on a regular basis to decrease the frequency of "rips."

**Complications:** Infection and other injuries related to decreased ability to grip bar ("peeling off") secondary to pain

## Lumbar Spine

- 5.2%–20% of injuries, lower back pain reported in 25%–85% of gymnasts
- Weak core and muscle imbalances despite overall general body strength are often noted
- Maintain a high index of suspicion for significant pathology, particularly those presenting with persistent pain despite reduced load in training

### Spondylolysis and Spondylolisthesis

**Epidemiology:** Female gymnasts have a 10%–16% incidence of pars defects on radiographs. Rhythmic gymnasts may be at a higher risk because of the need for repetitive extreme lumbar hyperextension.

**History and physical examination:** Pain on walking or sitting for long periods, worsening with hyperextension maneuvers, tenderness to palpation over lumbar spine posterior elements, positive stork test, and relative loss of hamstring flexibility

**Treatment:** No gymnastics activities until pain free; bracing is controversial; aggressive core strengthening program with focus on imbalances and flexibility; important to diagnose early in order to maximize the ability to establish a bony union of the stress fracture

### Degenerative Disc Disease/Scheuermann Disease

**Epidemiology:** Repetitive hyperflexion, increasing in incidence due to larger focus on "hollow body" position loading the anterior vertebral bodies

## Hip Injuries

### Pelvic Apophyseal Injuries

**Definitions: Apophysis:** Growth plate that adds contour and shape to bone without contributing to bone length (Table 90.7)

**Apophysitis:** Irritation of apophysis; areas include anterior superior iliac spine (ASIS), anterior inferior iliac spine (AIIS), ischial tuberosity, lesser trochanter, greater trochanter, and the iliac crest

**TABLE 90.7 PELVIC APOPHYSES**

| Apophysis | Attachment(s) | Age of Appearance (Years) | Age of Fusion (Years) |
|---|---|---|---|
| Anterior superior iliac spine (ASIS) | Sartorius | 13–15 | 21–25 |
| Anterior inferior iliac spine (AIIS) | Rectus femoris | 13–15 | 16–18 |
| Ischial tuberosity | Hamstring | 15–17 | 19–25 |
| Lesser trochanter | Iliopsoas | 8–12 | 16–18 |
| Greater trochanter | Gluteus medius | 2–5 | 16–18 |
| Iliac crest | Internal and external obliques<br>Transverse abdominis<br>Tensor fascia lata<br>Gluteus medius | 13–15 | 15–17 |

**Mechanism of injury:**
- **Acute:** Sudden, powerful contraction of involved muscle, avulses apophysis
- **Chronic:** Repetitive tensile forces from eccentric muscle contractions placing traction on involved apophysis

**History: Acute:** Sensation of popping with resultant pain in hip; pain may be severe
- **Chronic:** Insidious onset of pain and weakness of affected muscle group; possible recent growth spurt

**Physical examination:** Apophyseal tenderness; decreased ROM and strength, particularly with acute avulsions; antalgic gait common with acute avulsions

**Imaging: Acute:** Radiographs reveal avulsion
- **Chronic:** Radiographs often normal
- Comparison radiographs of contralateral apophysis may reveal differences in ossification centers. MRI may help confirm the diagnosis.

**Treatment:** Extensive physical therapy may be required for acute avulsions. More significant injuries or those not responding to relative rest may require nonweight-bearing or partial weight-bearing. Surgical treatment reserved for a significant degree of fragment displacement or chronic pain and disability despite adequate conservative treatment.

### Femoral Acetabular Impingement (FAI)

**Mechanism of injury:** Congenital bony malformation of hip that with repetitive use causes breakdown of normal labrum and articular cartilage, leading to pain, dysfunction, and early osteoarthritic changes. Hypermobility syndromes with repetitive trauma to labrum and articular cartilage secondary to extra-physiological ROM causes eventual hip degeneration. Because of the large ROMs required, this can be particularly debilitating to gymnasts.

### Acetabular Labral Tears

**Mechanism of injury:** Repetitive extreme ROMs, including extreme hip flexion, abduction, and extension, increases the repetitive forces placed on the labrum, increasing the risk of tears, can also occur with an acute fall.

### Hip Instability

**Mechanism of injury:** May be secondary to acetabular dysplasia or to chronic laxity of the soft tissues surrounding the hip; can be beneficial in attaining a large ROM required for gymnastics but over time can cause overuse syndromes related to chronic instability as well as labral tears and articular cartilage degeneration

**Physical examination:** Evaluate for hypermobility syndromes

**Complications:** Increases the risk of labral tear and/or FAI

**Treatment:** Physical therapy focused on stabilizing a hypermobile joint; may require a period of rest from exacerbating activities

## Knee Injuries

### Anterior Cruciate Ligament (ACL) Tear

**Mechanism of injury:** Landing without fully completing twisting elements, resulting in valgus, varus, or hyperextension of the knee; pure hyperextension mechanism

### Tibial Tubercle Apophysitis (Osgood–Schlatter Disease)

**History:** Pain with running, jumping, tumbling, and vaulting

**Education:** Important that gymnast and parents understand that the condition is limited and will resolve once apophysis fuses; symptom-guided modifications of training

## Ankle Injuries

### Inversion Sprains

**Mechanism of injury:** Inversion or inversion plantar flexion; landing with ankle/foot rolled under from jumps, saltos, and twists, stepping off landing mats, and landing with foot in seam of mats

**Treatment:** Comprehensive physical therapy program crucial for successful and safe return to gymnastics: ROM, strength, proprioception, with evaluation and correction of entire kinetic chain; ankle taping/bracing may be required during practice/competitions until the injured ankle is completely rehabilitated

**Complications:** Missed diagnoses, including OCD of the talar dome, os trigonum fractures, chronic lateral ankle instability, or distal fibular physeal fracture; consider MRI in recalcitrant cases; recurrent ankle sprains most often secondary to inadequate physical therapy

### Distal Fibular Physeal Fracture

**Mechanism of injury:** Same as inversion ankle sprains

**Imaging:** Recommend bilateral radiographs for comparison; may be negative in Salter-Harris type I fractures; diagnosis based on physical examination

**Treatment:** Nondisplaced, low-grade Salter-Harris fractures are treated with short leg walking cast or walking boot for 3–4 weeks. Once immobilization is completed, treatment is identical to that of inversion ankle sprains.

### Anterior Ankle Impingement Syndrome

**Mechanism of injury:** Abutment of dorsal portion of talus and/or navicular against anterior lip of tibia; results from gymnasts landing "short" (i.e., athletes underrotate backward saltos and lands with ankles hyperdorsiflexed) increasing forces across anterior ankle structures; over time, bone responds by forming exostoses that produce anterior ankle pain with dorsiflexion. Cartilage overgrowths (synovial chondromatoses), chronic synovitis, and articular damage also can occur.

**History and physical examination:** History of repetitive short landings with chronic anterior ankle pain, decreased dorsiflexion, tenderness over anterior tibiotalar joint, and pain to forced dorsiflexion of ankle

**Imaging:** Lateral ankle radiographs confirm exostoses, although they may be negative if impingement consists only of soft tissue (Fig. 90.2).

**Treatment:** Land on soft surfaces; avoid short landings; improve ankle flexibility. Utilize ankle taping that includes prevention of full dorsiflexion. Ankle taping plus utilization of a cut up tennis ball placed on the anterior ankle to prevent full dorsiflexion with landings (Fig. 90.3). In general, conservative treatment provides limited improvement. Consider steroid injection to reduce associated synovitis. Often requires surgical intervention for complete resolution of symptoms.

### Posterior Ankle Impingement

**Mechanism of injury:** Recurrent inversion ankle sprains; repetitively performing skills on ¾ pointe; posterior impingement may be caused by a combination of:
- Hypertrophy or tear of posterior inferior tibiofibular ligament or transverse tibiofibular ligament
- Pathology of os trigonum-talar process, subtalar joint disease, and fractures
- Flexor hallucis longus tenosynovitis

**History and physical examination:** Pain occurs with skills that require gymnasts to be on ¾ pointe or requires rebounding off ¾ pointe. Tenderness to palpation over posterior talus, posterior tibiofibular ligament, or os trigonum; swelling may be noted; pain reproduced by forced plantar flexion

**Imaging:** Radiographs may show presence and/or fracture of os trigonum. Lateral ankle radiograph in full plantar flexion may be helpful to assess any overlapping of posterior bony elements.
MRI: consider to evaluate for stress injury, posterior chondral injury, tenosynovitis (see Fig. 90.2)

**Treatment:** Physical therapy focusing on entire kinetic chain, flexibility, and proprioception; taping of ankle to minimize full plantar flexion; corticosteroid injection around posterior talus; if a nonunion of an os trigonum is the source of pain, consider arthroscopy for removal of os trigonum.

## Foot Injuries

### Calcaneal Apophysitis (Sever's Disease)

**Treatment:** Relative decrease in jumping and running activities; recommend all landings be performed on soft surfaces; utilize tumble track for tumbling and leaps; see the Injury Prevention section. Coaches must evaluate landing technique to ensure that the gymnast does not land back on heels; gastrocnemius–soleus complex stretching; tape on heel cups for practice or use Cheetah brace; work-out in tennis shoes if symptoms are particularly bothersome.

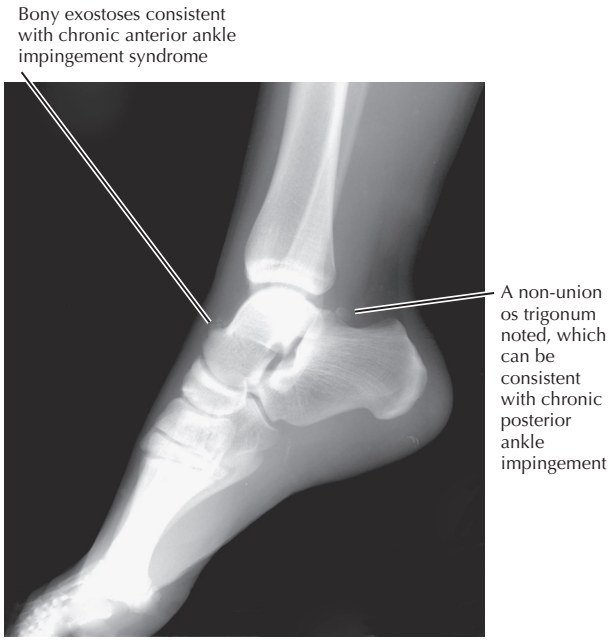



**Figure 90.2.** Ankle impingement.

**Prognosis:** Persistent pain is uncommon, but symptoms could mask an underlying calcaneal stress fracture.

### Calcaneal Contusion

**Mechanism of injury:** Landing over-rotated on dismounts, tumbling, and vaults with body weight back on heels; hitting heels on various apparatuses; performing a skill with poor direction and landing off the side of a mat on a nonpadded gym floor

**History and physical examination:** Mechanism of injury as noted above; pain with ambulation, calcaneus tender to palpation and/or compression, and localized swelling and ecchymosis

**Imaging:** Radiographs if calcaneal fracture or stress fractures are suspected; MRI may be useful to diagnose calcaneal contusion or stress fracture not seen on radiographs

**Treatment:** Ice massage; padding or heel cup for comfort during weight bearing; walking boot and/or crutches (if very symptomatic); heel cups can be taped on for practice, and/or knee pads (such as those used in volleyball) can be worn as heel protectors on uneven, horizontal, or parallel bars; or use basket weave taping of the heel. Correction of poor landing technique and landing on soft surfaces are important to prevent re-injury.

## FEMALE ATHLETE TRIAD

- Epidemiology: The prevalence of female athlete triad in gymnasts is unknown. Studies have demonstrated that gymnasts, along with athletes in other esthetic sports, are at an increased risk of disordered eating and tend to have an increased drive for thinness and high rates of body dissatisfaction.
- Gymnasts have higher bone mineral density because of impact loading forces of the sport compared with other athletes and nonathletic controls. This may offer a certain level of bone protection for gymnasts who suffer from one or more components of the triad.

## GROWTH AND MATURATION

- Female gymnasts are short before beginning gymnastics.
- Studies suggest attenuated growth during training.
- Catch-up growth during reduced training or retirement
- Studies unclear if "catch-up" is complete
- Cause and effect of gymnastics training resulting in stunted growth has not been demonstrated.
- Gymnasts tend to undergo menarche later than general female population.

## Pelvic Floor Dysfunction

- High-impact sports including gymnastics increase risk of urinary incontinence in nulliparous women

Traditional ankle taping. Cut tennis ball placed along anterior ankle joint. Tennis ball secured with Elastikon.

**Figure 90.3.** Ankle taping for anterior impingement syndrome. Photo courtesy of Anna Thatcher, PT, ATC.

  - Caused by recurrent increased intra-abdominal pressure without control of pelvic floor muscles
  - Stress-induced urinary incontinence most common type
  - May utilize coping strategies such as frequent bladder emptying during practices or competition events or using a thin menstrual pad
- Recommend pelvic floor muscle training for gymnasts for treatment and prevention

### Vitamin D Status

- Studies in artistic gymnasts show an increased risk of vitamin D deficiency
- Stress fractures in general are common in gymnastics. Besides over training or complications of female athlete triad, vitamin D deficiencies could be contributing
- Consider checking vitamin D status in gymnasts presenting with stress fractures and treat appropriately.

## INJURY PREVENTION

Areas to focus on for injury prevention include:

- Ankle strengthening, proprioception, and gastrocsoleus stretching
- Core programs to focus on relative muscle weaknesses or imbalances
- Correct hand placement on round-offs and off-loading the elbow joint
  - T hand position versus parallel position
- Wrist and forearm stretching and strengthening
- Utilizing sting mats, resi-pits, and alternate landing surfaces to help prevent overuse syndromes
- Utilize "no crack" training areas
- Teach correct landing biomechanics owing to a higher risk of injury during dismounts
- Track gymnast height, may modify training during peak growth velocity with an aim to reduce incidence of injuries during adolescent growth spurt

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS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# CHEERLEADING

# 91

Theadora Sakata • Susan M. Joy

## INTRODUCTION

- Cheerleading reportedly originated in the 1880s, when Princeton University formed an all-male pep club to support its football team. However, some have called University of Minnesota student Johnny Campbell the first official cheerleader when he organized a crowd of football spectators in 1898.
- Cheerleading involves a combination of dance, gymnastics, and overall athleticism and has evolved largely from the 1980s and 1990s to what we see today.
- Teams are found at recreational, club, and varsity levels.
- All-Star teams are focused primarily on competition against other cheer teams, rather than cheering for another sports team.
- Organized cheer may be found in three scenarios:
  - Competition
    - Routines are performed and judged based on difficulty and execution of maneuvers.
    - Events typically last 1–2 days.
    - Generally held on spring floors or gymnasium hardwood floors
  - Performance
    - Participate in pep rallies or engage in sideline cheering and other events, rather than competition.
    - May occur on a wide variety of surfaces, including grass or turf, gymnasium hardwood, or cafeteria and multipurpose room flooring that ranges from linoleum to tile and carpet
  - Practice
    - Regular practices occur at the high school and college levels, similar to that held for other sports teams
    - Sometimes more than one session per day
- Growth of cheerleading
  - In 1995, there were 30,954 cheerleaders on competition squads registered in the National Federation of High Schools database.
  - This had increased to about 114,400 by 2003.
  - In 2003, there were approximately 3.6 million cheerleaders of all ages.

### Glossary of Terms

Some terms taken from the American Association of Cheerleading Coaches and Administrators (AACCA).

**Base:** A person in direct contact with the performing surface and supporting another person's weight.

**Cradle:** A dismount from a partner stunt, pyramid, or toss, in which the catch is completed below shoulder height by a base or bases with a top person in a face-up open-pike position.

**Dive roll:** A forward roll where the feet leave the ground before the hands reach the ground.

**Drop:** Landing on the performance surface from an airborne position.

**Elevator/sponge toss:** A stunt in which a top person loads in to an elevator/sponge-loading position and is then tossed into the air.

**Flip:** When a person is airborne while his or her feet pass over the head.

**Helicopter:** A stunt in which a top person is tossed into the air in a horizontal position and rotates parallel to the ground in the same motion as a helicopter blade.

**Inverted:** Refers to a body position where the shoulders are below the waist.

**Middle:** A person who is being supported by a base while also supporting a top person.

**Post:** A person on the performing surface who may assist a top person during a stunt or transition.

**Prep:** A stunt in which one or more bases holds a standing top person at approximately shoulder height.

**Prep level:** When a top person's base of support is at approximately shoulder height.

**Pyramid:** A skill in which a top person is being supported by a middle and base layer person.

**Quick toss/partner toss:** A toss technique where a top person begins the toss with at least one foot on the ground.

**Release stunt:** A transition from one stunt to another (including loading positions) in which a top person becomes free from all bases, posts, and spotters.

**Rewind:** A skill in which a top person starts with both feet on the ground is tossed into the air and performs a backward or side rotation into a stunt or loading position. Flips are limited to one rotation and twists are not permitted.

**Spotter:** A person who is responsible for assisting or catching a top person in a partner stunt or pyramid. This person cannot be in a position of providing primary support for the top person but must be in a position to protect the top person coming off of a stunt or pyramid.

**Stunt/partner stunt:** One or more persons supporting one or more top persons off of the ground.

**Top/flyer:** A person who is not in contact with the performing surface and is being stabilized by another person or who has been tossed into the air.

**Toss:** A release stunt in which the base(s) begin underneath a top person's foot/feet and execute a throwing motion from below shoulder level to increase the height of the top person, and the top person becomes free from all bases, spotters, posts, or bracers.

**Tumbling:** Gymnastic skills that begin and end on the performing surface.

### Governing Bodies

- **American Association of Cheerleading Coaches and Administrators (AACCA):** Founded in 1987 with a focus on promoting safety and safety education in cheerleading
- **US All-Star Federation:** Founded in 2003 to promote All-Star-style cheer as a competitive sport; the governing body for cheer teams and competitions that are not associated with schools
- **USA Cheer:** Founded in 2007 as the governing body for sport cheering in the US
- **National Federation of High School Sports (NFHS):** Provides rules for high school cheerleading as it does for other sports

### Safety Rules

- Rules for junior high school-, high school-, and college-level cheer may be found at the CheerRules website (www.cheerrules.org), a joint venture between the AACCA, its CheerSafe initiative, and the NFHS.

- Rules vary based on league and location, especially for recreational cheer teams.
- AACCA provides age-based guidelines for safety, particularly with regard to:
  - Height of builds
  - Allowable maneuvers
  - Surface-specific restrictions
- Youth recreational cheer guidelines (AACCA Guidelines 2015) have the following provisions:
  - For all teams:
    - No basket or elevator tosses
    - No tension rolls
    - Tumbling acceptable but not required
    - No twisting tumbling (Arabians or full twists)
    - No released twists (no helicopters, log rolls, or twisting cradles)
    - Only straight cradles allowed.
    - A spotter is required on all buildings.
    - No inversions allowed in stunts.
  - Flag Level (K-1st Grade):
    - No building above the waist level
    - One foot must be in contact with the base at all times.
    - Thigh stands acceptable.
  - Peewee Level (2nd–3rd Grade):
    - No building above shoulder level
    - Elevator preps, shoulder level liberties, and shoulder stands/sits acceptable.
    - One foot must be in contact with the base at all times, except during a cradle.
  - Junior Level (4th–6th Grade): Top girls must have one foot in contact with the base at all times during any extended stunt.
- USASF provides All-Star competition guidelines; the execution of more complex maneuvers and stunts are allowed at the higher numbered levels.
- NFHS interprets risk-related guidelines set forth by the AACCA for high school squads.

## EPIDEMIOLOGY

- The overall injury rate in cheerleading is 0.9 injuries per 1,000 athlete exposures (AEs), which is lower than that in gymnastics (8.5), soccer (5.3), basketball (4.4), softball (3.5), and volleyball (1.7).
- Injury rates higher in competition than in other settings
  - 0.89–1.40 per 1,000 AEs in competition
  - 0.80–1.00 per 1,000 AEs in practice
  - 0.50–0.80 per 1,000 AEs in performance
  - 0.75–1.00 per 1,000 AEs overall
- Injury rates were not associated with a coach's credentials or his/her completion of cheerleading safety training courses.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Sprains and Strains

- Most common injury type
- Number of days lost due to these kinds of injuries does not differ significantly between athletes on high school, college, and All-Star teams.
- Parts of the body most commonly sprained
  - Ankle (28%)
    - Injury occurrence most frequently associated with tumbling, stunts, and jumps
  - Neck (11%)
  - Low back (11%)
    - Injury occurrence more often associated with spotting/basing another cheerleader than other mechanisms
- Cheer activities most commonly leading to a sprain-type injury included stunting (34%), tumbling (32%), tosses (18%), and pyramids (6%).
- Dancing only implicated in 5% of sprain injuries.

### Sprain and Strain

Most common when moving or tumbling, followed by stunts
**Contusions:** Occur mostly during stunts and tosses
**Fractures:** Mostly caused by moving or tumbling, then tosses

### Concussions

Occur mainly due to tosses, followed by stunting and pyramids

### Catastrophic Injuries in Cheerleading

- Although overall injury rates are relatively low compared to that in other sports, catastrophic injury rates in cheerleading are relatively high (Box 91.1).
- Cheerleading has higher catastrophic injuries than other sports per 100,000 athlete exposures.
- Floor surface types may play a role in the potential for brain injury as different materials have different "critical heights," defined as the height above which life-threatening head injuries are more expected to occur if one is dropped (Fig. 91.1).
  - Most ideal surface: a spring floor, followed by a landing mat over a foam floor.
    - These surfaces have critical heights over 10 feet.
    - Provide sufficient impact absorption for 2-level stunts
  - Rubberized tracks, carpets, vinyl tiles, and concrete are the least ideal surfaces
- An Emergency Action Plan (EAP) should be written, available, and rehearsed for venues hosting cheerleading competitions, performances, or practices.
- It is believed that with safety rules and regulations put in place since 2005–2006, cheerleading has become a safer sport; however, data are still being collected.

### BOX 91.1 STUDY ON CATASTROPHIC INJURIES

Of a total of 134 catastrophic injuries reported in high school athletes from 1982-2013, cheerleading accounted for 64%, even though the sport has only accounted for 4% or less of female high school sports participation in the years included in the National Federation of High Schools sports participation surveys. 86 catastrophic injuries to high school cheerleaders were documented:

- 2 deaths (1 cardiac arrest in 16 year-old female; 1 case of splenic rupture after a flyer landed face-down in the arms of her teammates)
- 34 non-fatal injuries, including skull and vertebral fractures that resulted in permanent paralysis or other deficits
- 50 "serious" injuries that did not result in a permanent functional disability
- 1 college-level death has been noted: A flyer's head hit an unpadded floor during a basket toss in competition.

Data from Mueller FO, Cantu RC. Catastrophic sports injury research: thirty-first annual report, Fall 1982 – Spring 2013. National Center for Catastrophic Sports Injury Research. Available online at https://nccsir.unc.edu/files/2015/02/NCCSIR-31st-Annual-All-Sport-Report-1982_2013.pdf

## Cheerleading and Concussion

- In the published literature, concussions account for about 6% of stunt-related injuries, but actual rates may be as high as 35%.
- A cheer-specific return-to-play sequence after concussion has been proposed.
  - Sequence follows the same principles of return to play in other sports.

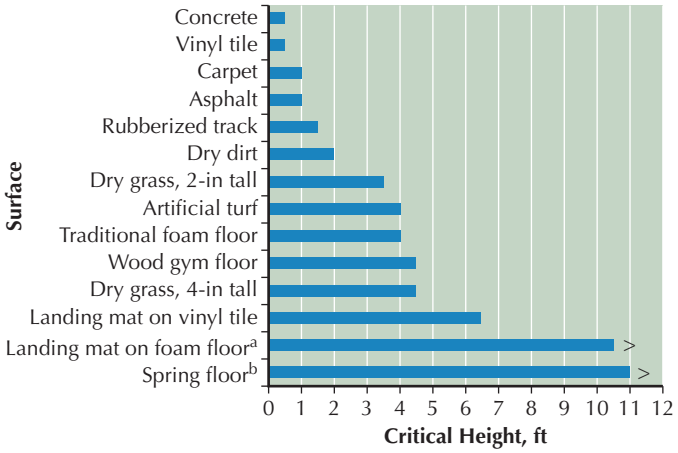


**Figure 91.1.** Critical Height for Cheerleading Surfaces. [a]Landing mat on traditional foam floor. Limits of Triax 2000 were reached before critical height was attained. [b]Limits of Triax 2000 were reached before critical height was attained. (From Shields BJ, Smith GA. The potential for brain injury on selected surfaces used by cheerleaders. *J Athl Train.* 2009;44[6]:595-602. Fig. 2, page 599.)

  - Once the athlete is symptom free with and capable of moderate aerobic activity and light resistance training, he/she may start with moderate jumping such as toe-touches or 15-yard sprints to approximate a tumbling pass without the tumbling.
  - Further progression prior to clearance for full practice may start incorporating roundoffs, walkovers, a single handspring, and non-inverted lifts, eventually progressing to inverted lifts.
  - With progression, it is important to monitor for vestibular-based symptoms, especially with inversions.
- Additional cheer-oriented concussion recommendations, including a sample stepwise return-to-cheer plan, are available on the AACCA at www.aacca.org/concussions.
- See Table 91.1.

## Other Medical Concerns

- Body image dissatisfaction and female athlete triad
  - Body dissatisfaction has been reported by 73% of Caucasian and 50% of African–American female high school cheerleaders.
  - A study of collegiate cheerleaders has demonstrated that flyers not only had the lowest average BMI (20.8 kg/m$^2$) on a cheer squad but also had the highest risk of developing eating disorders compared to bases or back spots.
    - For each BMI unit increase, cheerleaders had an odds increase of 0.368 in their risk of developing eating disorders.
  - Some cheer teams have coaching-imposed weight restrictions, often based on aesthetic beliefs or focused on flyers or top persons.
    - These restrictions, often out of concern for bases and spotters, are not supported by data.

**TABLE 91.1 PROPOSED RETURN TO PHYSICAL ACTIVITY FOLLOWING CONCUSSION FOR CHEERLEADING**

| Stage | Activity | Cheerleading Specific Exercise | Objective of the Stage |
|---|---|---|---|
| 1 | No physical activity; complete physical and cognitive rest | • No activity | • Recovery and elimination of symptoms |
| 2 | Light aerobic activity | • 10–15 min of walking at home or at gym, or stationary bike | • Add light aerobic activity and monitor for symptom return |
| 3 | Moderate aerobic activity<br>Light resistance training | • 20–30 min jogging<br>• Resistance training—body weight squats and push-ups 1 set of 10 reps each | • Increase aerobic activity and monitor for symptom return |
| 4 | Vertical work<br>(No inversion) | • Moderate conditioning jumps (toe-touch, Herkie, double hook), 15 yard sprints (as in tumbling pass), stunting with feet on the ground<br>*no tumbling | • Maximize aerobic activity<br>• Introduce head movements<br>• Monitor for symptoms |
| 5 | Intro level tumbling | • Round-off<br>• Walkovers<br>• Handspring (1)<br>• Light tumbling<br>• Progress from noninverted lifts to inverted lifts<br>• Cradle catch | • Maximize aerobic activity<br>• Add deceleration/rotational forces in controlled setting<br>• Introduce inversions—vestibular stress<br>• Monitor for symptoms |
| 6 | Full practice<br>(after medical clearance) | • Normal training activities | • Reassess for symptoms every 30 minutes throughout the practice<br>• Monitor for symptoms |
| 7 | Unrestricted workouts | • Return to competition | • Assess frequently<br>• Monitor for symptoms |

- Progress to the next stage may occur every 24 hours as long as symptoms do not return.
- It is recommended that you seek further medical attention if you fail more than three attempts to pass a stage.

From May KH, Marshall DL1, Burns TG1, Popoli DM1, Polikandriotis JA. Pediatric sports specific return to play guidelines following concussion. *Int J Sports Phys Ther.* 2014:9(2):242-255. Appendix 1, page 246.

- These practices should be discouraged as they may have detrimental effects on the athletes' body image.

## STRENGTHENING AND CONDITIONING

- Few cheerleading-specific studies have been done; however other sports such as gymnastics and dance may offer a guide to the development of a cheerleading strengthening and conditioning program, given the commonality of many of the movements.
- Given the frequency of strains and sprains in cheerleading, the following are recommended to help promote prevention of injury and re-injury:
  - For ankle sprains:
    - Balance and coordination training, in addition to strengthening and stretching.
    - Consideration of bracing or taping, as well as orthotics and appropriate footwear.
  - For back strains and sprains:
    - Especially of concern for bases and spotters.
    - Training on proper lifting techniques.
    - Total body weight training to improve the strength of abdominal and trunk muscles, in addition to that of the upper and lower extremities.
- A small randomized controlled trial in dancers demonstrated that a conditioning program that included aerobic endurance, strengthening, motor control, and proprioceptive training significantly resulted in fewer lower back injuries than reported in a group randomized to usual training and an educational health promotion program. These exercises may be especially helpful for spotters and bases who need to lift their teammates up in the air.
- Jump training in prepubertal, high-level female gymnasts has been suggested to improve flight time, which may also be beneficial in cheer.
- Arthrometer-measured shoulder laxity in a group of collegiate cheerleaders decreased after a strength training program, suggesting that this may be beneficial for shoulder stability.

## CHALLENGES AND FUTURE DIRECTIONS

- As a relatively new sport, sport-specific research on cheerleading has been limited.
- Data are difficult to collect, especially at the collegiate level where cheer is often formally classified as an activity rather than a sport.
- Information is scarce for athletes prior to reaching high school, at which point they may be captured by the NFHS or the National High School Sports-Related Injury Surveillance Study.
- Cheerleading should be included in ongoing efforts of quantifying sports participation and related injuries given its unique catastrophic injury profile and mix of other athletic disciplines.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# DANCE

# 92

Craig C. Young • Selina Shah • Laura M. Gottschlich

## INTRODUCTION

- Dance is an activity that can be found in most cultures dating back to ancient times.
- Dance is unique in its fusion of art and athletic activity.
- It can increase cardiorespiratory fitness, muscular strength and endurance, flexibility, and bone mineral density.
- Classical ballet provides a foundation for other dance forms, and historically, considerable amount of research has focused on ballet. However, in recent years, interest and research in other forms of dance has increased.
- Other popular forms of dance include contemporary (modern), jazz, hip hop, tap, Irish, folk dance, and ballroom.
  - Although there is some crossover, each of these forms has unique features and injury profiles.

## EPIDEMIOLOGY

A 2011 study of American adolescents found that 20.9% participated in dance, making it the third most common physical activity in girls.

### Ballet

#### Demographics

- Professional female ballet dancers often start classes between ages 4 and 9, with males starting between ages 12 and 16.
- Preprofessional ballet training begins at about age 11 but can start as early as age 8.
  - There is an increase in the duration and intensity of training.
  - Training is conducted 5–6 days per week, ranging from 6 to 45 hours per week.
- The average age for professional ballet dancers is 26 to 27 years.
- The average female body mass index (BMI) is 18 to 19; that for males is 21 to 22

#### Injuries

FREQUENCY

- Injury rate: 75%–95% of ballet dancers suffer at least one injury per year, with an average of 3.0 to 3.2 injuries per dancer per year
- 1.09 to 3.52 injuries per 1,000 dance exposures
- 0.6 to 4.4 injuries per 1,000 hours of training

TYPE

- 53.6%–85% of injuries are from overuse, and 12%–45% from acute trauma
- Most injuries are of muscle strains, followed by ligament sprains and chronic inflammatory processes.

SEVERITY

- The average time lost to injuries is 32.5 days in females and 21.6 days in males.

ANATOMIC LOCATION OF INJURY

- Most common: foot and ankle, low back, hip, and knee

RISK FACTORS

- Intrinsic risk factors for dance-related injuries include: anatomic structure, inadequate strength and flexibility, improper technique, nutrition, previous injury, fatigue, inadequate turnout, higher rate of growth, female gender, and disordered eating behaviors.
- Extrinsic risk factors include: choreography, cold environment, dance floor properties (e.g., surface, resilience).
- Students in summer intensive programs are at high risk of injury due to the sudden increase in hours of activity.

### Modern

#### Demographics

- Professional female dancers start taking dance class at 6.5 years of age, while male dancers start at 15.6 years of age.
- Most professional female modern dancers began their dance careers by studying ballet, whereas men began by studying modern.
- The average age for female professional modern dancers is about 30 years; that for males is 31 years.
- The average BMI for females is 20.6; that for males is 23.6.
- Professional modern dancers study various forms of dance, including ballet, pointe, jazz, tap, hip hop, African, and ballroom, outside the time they spend in rehearsal for their companies.
- They spend an average of 8 hours taking various types of dance classes and about 17 hours in rehearsals for their companies.
- Most dancers also spend about 2 to 3 hours per week doing some form of exercise outside of dance such as yoga, Pilates, Gyrotonics, weightlifting, running, biking, and walking.

#### Injuries

FREQUENCY

- Up to 82% suffer injuries per year
- The annual incidence of injury is 1.2 in males and 1.7 in females.
- Injury rate: 0.6/1,000 hours of dancing
- The majority of injuries occur in class, followed by rehearsal and performance.

TYPE

- Most injuries result from overuse or gradual onset (57%) rather than as a consequence of an acute or traumatic event (43%).
- The most common injury types are: muscle strains, followed by ligament sprains, and then other chronic inflammatory processes.

SEVERITY

- Dancers can return to partial dancing after an average of 2–3 weeks post injury.
- Returning to full dancing can take an average of up to 2 months.
- Modern dancers admit to returning to dance with pain.
- Males miss fewer classes and rehearsals as a result of injury than females.

ANATOMIC LOCATION

- The most common sites of injury in descending order are: ankle, low back, knee, and foot.

RISK FACTORS

- Intrinsic risk factors: self-pressure, ignoring pain and fatigue
- Extrinsic risk factors:
  - The demands of the role and from the choreographer
  - Floor characteristics: surface, resilience, raked or not (floor angled down to audience for better viewing)

INJURY CONSULTATION

- Most dancers will consult with someone within 1 week of injury.
- Fewer than half of the dancers will consult with a physician regarding the injury because they either think that physicians are neither helpful nor understanding of them as dancers; or the dancers do not have health insurance, or are concerned that they will be told to stop dancing for too long. Many dancers consult other healthcare providers such as company physical therapists, chiropractors, massage therapists, and acupuncturists. Many will also discuss their injuries with a member of the company such as the choreographer, company director, or instructor.
- Most dancers actually adhere to the advice given to them.
- Their main reasons for not adhering to the advice given to them include: the lengthy amount of time recommended to refrain from dance, fear of being held out of class or rehearsal if the staff knew about the injury, not agreeing with the advice, and fear of losing their role in the performance to an understudy or rival.

## Irish

### Demographics

- Became mainstream in 1994 following the production of *Riverdance* and subsequent professional touring shows
- The age of professional dancers ranges between 17 and 34 years.
- The mean age of dancers first turning professional is 18.5 years.
- The age of competition-age dancers generally ranges from 4 to 21 years.

### Injuries

FREQUENCY

- Up to 60% professional Irish dancers have suffered injuries.
- Up to 80% of competitive level Irish dancers have suffered injuries.
- The lifetime risk of injury can be as high as 90%.
- 79.6% of injuries are categorized as overuse or chronic.
- 20.4% of injuries are categorized as traumatic or acute.

TYPE

- In descending order of frequency: tendon injury, apophysitis, patella pain or instability, stress injury, strain, and sprain

LOCATION

- 95% of injuries involved the lower extremities. Frequency in descending order: foot, ankle, knee, and hip

RISK FACTORS

- Intrinsic: fatigue or overwork, ignoring early warning signs, improper or lack of warm up and/or cool down
- Extrinsic: accident, unsuitable floor, repetitive movement, unsuitable foot wear

## GENERAL PRICIPLES

### Terminology

**Arabesque:** A pose in which the dancer stands on one leg and raises the other straight behind (at various angles); usually one arm is stretched out in front (Fig. 92.1)

**Class:** Lesson

- Barre—the first part of ballet class conducted using the railing for balance and technique training
- Center—the portion of class in which dancers perform dance movements in the "center of the room" without using the barre for assistance
  - Across the Floor: A series of choreographed steps performed diagonally across the room in small groups, with choreography done on the right side in one direction and on the left side in the other

**Demi-pointe:** The foot is maximally plantar flexed with toes maximally extended—weight on metatarsal heads.

**Foot positions:** (see Fig. 92.1)

- First position—feet turned out with heels touching
- Second position—feet turned out with heels apart
- Third position—feet turned out, overlapping with right heel in hollow of left foot
- Fourth position—feet turned out, apart but with overlapping heels
- Fifth position—feet turned out, touching with right heel in front of left toe

Arabesque.

Demi plié

Grand plié.

Basic foot positions—first to fifth from left to right.

On pointe (during relevé).

**Figure 92.1.** Common terms in dance. (Photographs courtesy of Craig C. Young, MD and Selina Shah, MD.)

**Jeté:** A jump where the legs are in a split position in the air
**Plié:** Bending of the knees and ankles with the legs turned out
- Grand plié—a large or deep plié where the knees are maximally flexed and the feet are in demi-pointe (see Fig. 92.1)
- Demi-plié—a "small" plié where the knees are only partially flexed and the feet are flat on the floor (see Fig. 92.1)

**Pointe:** Dancing while supporting the body on the tips of the toes (see Fig. 92.1)
**Relevé:** To rise up to the tiptoes or full pointe
**Turnout:** A stance in which legs are rotated outward. Turnout is the sum of the external rotations of the hip, knee, tibia, ankle, and foot.

## TYPES OF DANCE

### Ballet

#### Background

- Classical ballet originated in the 1400s in Italy and blossomed in the 1600s in France.
- Traditionally, professional ballet companies perform story ballets. Some of the most famous ones are *The Nutcracker* (usually performed during Christmas), *Swan Lake*, and *Giselle*. They also will perform mixed programs consisting of a variety of choreographed dances.
- Ballet is a choreographed series of specific motions, with specific placement of all body parts from the head to the toes.

#### Technique

- Many positions require extreme external rotation of the hips (see Fig. 92.1).
- Flexibility is required to achieve 180 degree splits of the legs.
- Strength is required to hold the legs in extreme positions of hip flexion and in extension with extended knees and plantarflexed ankles and toes.
- Dancing on pointe consists of dancing while supporting the body on the tips of the toes. This is almost always done by women, but there is one professional male company, as well as a few other choreographed dances, where males perform pointe work.
  - Most young female dancers aspire to go on pointe.
  - Adequate skill, technique (including balance and core stability), alignment, maturity to apply teacher corrections, and at least 90 degrees of ankle plantarflexion to achieve full pointe are the basic requirements. More sophisticated tests that examine the dancer's ability to maintain proper alignment and balance while performing ballet jumps and combinations may also be useful (Fig. 92.2, relevé passé).
  - Though age is not a requirement, most dancers are about 11 or 12 years old before they achieve all of the necessary criteria for pointe.

#### Equipment

- Both men and women wear ballet slippers for the majority of classwork. These are made of soft, supple material and a flexible, thin leather sole. They must fit snugly and are secured with elastic band(s).
- Pointe shoes are handmade from lacquered satin and burlap, with a stiff leather sole, which results in a hard but pliable shank and a rigid, unpadded toe box (see Fig. 92.2).

### Jazz

- Jazz dance originated from the African–American dances of late 1800s and has led to the advent of tap dancing, as well as some forms of ballroom (e.g., the swing and Lindy).
- Traditional jazz shoes—soft leather with a small heel
- Jazz sneakers—soft leather/canvas with a padded sole to increase shock absorption from jump landings
- Some positions require less external hip rotation than ballet.
- Jazz dancing is very free-form and includes kicks, falls, jumps, and slides; generally consists of quicker, sharper movements than ballet.

### Modern (Contemporary)

- Originated at the turn of the 20th century in defiance of the rigidity of classical ballet.
- Unlike ballet, modern dance uses gravity and emphasizes freedom of movement, sometimes taken to bizarre extremes.

Ballet shoes: Pointe shoe cutaway-note lack of padding when compared to running shoe.

Ballet: Relevé passe.

Modern dancers.

Irish dance hard shoes.

Typical Irish dance position (arms tightly adducted to side and elbows locked in extension. Neutral posture is held from the waist up during all dancing and hands are closed into fists).

**Figure 92.2.** Types of dance and equipment. (Ballet photographs courtesy of Craig C. Young, MD; Modern dance photograph courtesy of Blue 13 Dance Company, Ryuichi Oshimoto; Irish dance photographs courtesy of Laura Gottschlich, DO.)

- Most professionals dance barefoot, although a few wear some type of jazz shoe, ballet shoe, foot thong, socks, or simply use duct tape (see Fig. 92.2).

### Ballroom

- Ballroom dancing is a set of partner–dancer dances that were initially developed as social dances.
- Traditionally, the purpose of the dance was to show off the woman, who performs all the fancy moves, while the male acts as support as well as the leader.
- Women wear high heels.

#### Types of Ballroom

- Smooth (waltz, foxtrot, tango)
  - These dances, especially waltz, involve lots of extension and flexion from the knees to create the rise and fall of the dancers.
- Latin (cha-cha, rumba, samba, jive, paso doble)
  - Typically these dances emphasize hip motions.
- Rhythm (East Coast swing, mambo)
- Dance sport—competitive athletic-style dancing based on all of above styles that typically involves more lifts, throws, and slides than traditional ballroom dances
- Other ballroom dances include the polka, two-step, Lindy, and West Coast swing.

### Folk

- A highly variable type of dance—depends on individual style
- Irish is most common subtype.

### Irish

- The earliest recording in literature of Irish dancing was in 1300.
- Became mainstream in 1994 with the production of *Riverdance*
- Originated in Ireland, migrated first to England, and then to Australia, Canada, and the United States
  - Schools are now established in 30 countries across five continents.
- Combines artistic elements of ballet with the rhythm of jazz and tap. Has had an important influence on American dancing—including square dance, clogging, jazz, and tap
- Has a competitive and a professional element
  - Professional/paid dancers are ineligible for competition.
- Shoes consist of a soft leather, lace-up slipper (soft ghille or reel shoes) and a leather shoe with fiberglass tips and heels (hard or jig shoes) (see Fig. 92.2).
  - The extremely snug fit gives a tight toe appearance in plantar flexion.
  - Recent adjustments of neoprene padding has slightly improved shock absorption.

#### Competition

- Competitions exist at local *(Feis)*, regional *(Oireachtas)*, National, and World *(Rince Na Cruinne)* levels.
- Competition may be solo or in groups of up to 16 dancers.
- Dancers compete in multiple dances in both hard (Treble Jig, Hornpipe, Set Dances) and soft shoes (Reel, Slip Jig, Jig, Figures).
- Dancers are judged on timing to music, posture, position of feet and legs during execution of the dance, and style.

#### Biomechanical Principles

- There are some similarities between ballet and tap dancing.
- Most elements of Irish dancing are performed in the sagittal plane.
- Hamstring flexibility must be enough to afford approximately 0 to 135 degrees hip flexion.
- Dancing is performed in external rotation from the hip to approximately 70–80 degrees.
- The front foot is routinely crossed in front of back foot, covering the back toe during most dance moves and in tight plantar flexion (see Fig. 92.2).
- The front knee is routinely covering back knee during most dance moves.
- Most dancing is performed with arms tightly adducted to side and elbows locked in extension. A neutral posture is held from the waist up during all dancing, and hands are closed into fists (see Fig. 92.2).
- Most dance moves are performed with rigid landing in point/demi-point positions and the knees tightly extended.
  - This is different from ballet where jump landings utilize a demi-plié so that shock is also absorbed through the knees.
- Repetitive pounding of the balls of the foot occurs for minutes at a time during rhythm moves with the hard shoes, as well as during landing from jumps.
- Recent studies show many moves in Irish dance produce force of 2–4 times a dancer's bodyweight.
- Turning of the head for the purposes of spotting for jumps and spins is not allowed, unlike most other forms of dance.
- In the late 1980s, dancers began to dance on pointe in their hard shoes, often jumping, spinning, and running on the fiberglass tips.
- In 2002, the governing body of Irish dancing, *An Coimisiun*, banned toe or "block" movements for all children under the age of 12, citing concern over stress on immature bones with pointe movements.

### Aerobic

- Differs from other form of dance because its purpose is primarily for exercise
- Emphasizes quick motions to raise heart rate and tone muscles, rather than artistic form and performance
- Exercisers wear shock-absorbing athletic shoes.

## COMMON INJURIES AND MEDICAL PROBLEMS

### Eating and Nutritional Disorders

- Very common, especially in adolescent dancers
- Professional female dancers often believe that they weigh more than an "ideal" dancer should, even though audience members believe that most appear to be at the weight of an "ideal" dancer.
- Problems may start in preadolescent dancers.
- Increases risk of stress fractures

### Menstrual Disorders

- Primary amenorrhea and secondary amenorrhea are common in dancers.
- Increases risk of stress fractures

### Smoking

- Higher prevalence in dancers compared to other athletic populations
- Especially at risk—female dancers who are having difficulty maintaining weight

### Burnout

- Beware—especially in adolescent folk dancers who present with recurrent atypical injuries

## Osteopenia

- Amenorrheic female dancers at risk
- Eumenorrheic female dancers and male dancers tend to have higher bone densities than nonathletes.

## Musculoskeletal Injuries

### Shoulder

- Rotator cuff problems, particularly in male dancers

### Spine

- Neck and back strains (especially of the trapezius, rhomboid, and latissimus)
- Prevalence of "incorrect" lifting techniques—remember that choreographers and artistic directors often value appearance over optimal lifting technique.
- Other back problems—spondylolysis, spondylolisthesis, scoliosis, and facet syndrome
- Core stability tests (for 30 at least seconds)
  - Pike position—push up with forearms supporting (elbows bent 90 degrees) and on toes with ankles in neutral
  - Side lift—hold trunk linear facing side support on forearms

### Hip and Groin

- Piriformis syndrome
  - Pain deep in gluteal or groin region
  - Pain worsens with stair climbing, standing up, or prolonged sitting
  - Pain with extreme passive hip internal rotation (flexed approximately 90 degrees) or resisted hip external rotation
- Snapping hip syndrome
  - External snapping hip—in most nondancers, snapping hip syndrome is of this type, which is caused by the iliotibial band (ITB) snapping over the greater trochanter.
    - Dancers with external snapping hip syndrome complain of snapping when landing jumps.
  - Internal snapping hip syndrome is common in dancers and is caused by iliopsoas.
    - Usually in the nonweight-bearing gesture leg (e.g., when bringing the hip from an externally rotated, abducted, flexed position to a neutral extended position)
    - Can be reproduced by bringing hip from flexed, externally rotated position to an extension with internal rotation
    - Sometimes reproduced with a resisted straight knee sit-up
  - Both can be clinically diagnosed and, if required, confirmed with ultrasound.
- Hip labral tears and femoral acetabular impingement
  - Sharp, painful catch in groin
  - Can be confirmed with magnetic resonance imaging (MRI) arthrogram
- Sartorius tendonitis
  - From overuse of external rotator and flexor of the hip
  - Pain with external rotation, full flexion, abduction of hip
- Rectus femoris tendinitis
  - From repetitive forward extension of leg (e.g., battement or développé devant)
  - Groin pain, especially with knee extended

### Knee

- Patellofemoral pain syndrome
- Patellar tendonitis

### Leg

- Tibial stress fracture
  - Avoid rodding—this may cause loss of motion at knee; drilling out the fracture is a better option.
- Medial tibial stress syndrome

### Ankle

- Achilles tendonitis
  - Ribbon friction—beware of where ribbons from slippers cross the Achilles tendon (Fig. 92.3).
  - Character shoes—often do not have Achilles tendon notch and may not have optimal sizing because the wardrobe department may have limited costumes (see Fig. 92.3)
  - Certain movements like the "rock" step in Irish dance puts up to 2700 N of force or 14 times a dancer's body weight on the Achilles tendon and the connecting muscles (see Fig. 92.2).
- Ankle sprains
- Ankle impingement
  - Anterior: especially aggravated by pliés; often associated with osteophyte at margin of anterior tibia and dorsum of talus
  - Posterior: especially aggravated by pointe and demi-pointe; often associated with os trigonum or prominent posterior process of talus
  - Evaluation: lateral foot x-ray in maximal dorsiflexion (for anterior impingement) or maximal plantarflexion (for posterior impingement) (see Fig. 92.3)
  - Treatment: although surgery in dancers is often career-ending, surgery for ankle impingement may be career-saving.
- Peroneal tendonitis; subluxation; dislocation; and dysfunction
- Tarsal tunnel syndrome

### Foot

- Cuboid subluxation
  - A plantar subluxation of the cuboid
  - Risk factors include the tendency to be in valgus position of forefoot at metatarsal joints.
  - Can occur secondary to lateral ankle sprain
  - Symptoms: lateral foot pain, weakness with push-off, and inability to jump because of sharp pain
  - Diagnosis: pressing dorsally on cuboid from plantar surface reproduces pain and possibly produces visible depression. Radiographic studies usually unsuccessful
- Subtalar coalition
  - A bony connection that is between the calcaneus and either the navicular or the talus
  - Limits foot motion
  - May cause mechanical pain
  - Usually affects young dancers
- Navicular stress fractures
- Lisfranc joint injuries
  - Lisfranc joint: articulation of second metatarsal with first and second cuneiforms (see Fig. 92.3)
  - The Lisfranc joint has limited mobility
  - Pointe dancers at highest risk
  - Lack of dorsal support and immobility of second metatarsal causes dorsal displacement of second metatarsal with axial load in plantar flexion
  - Sprain: disruption at Lisfranc joint
  - Fracture/dislocation: diastasis between first and second metatarsal seen in standing anterior–posterior radiograph of foot. Line from medial edge of fourth metatarsal to cuboid is disrupted, as seen on standing medial oblique view of foot.
- Spiral fracture of shaft of fifth metatarsal (dancer's fracture) (see Fig. 92.3)
  - Etiology: inversion injury while on pointe
  - Treatment: cast shoe
- Metatarsal shaft stress fractures
  - Usually in second to fourth metatarsals
  - Especially in the longest toe
- Flexor hallucis longus tendonitis (dancer's tendonitis)
  - Usually occurs in demi-pointe

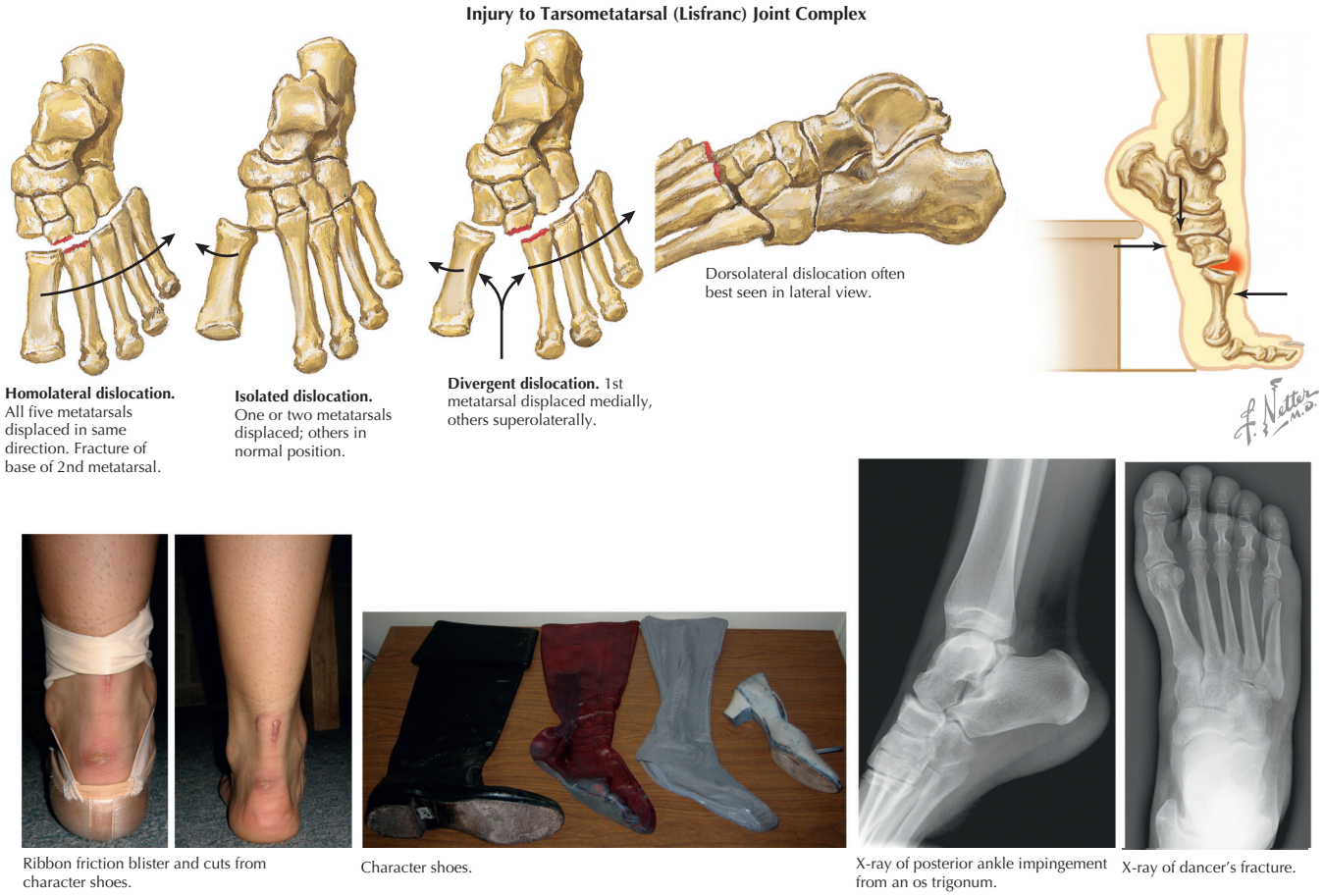


**Figure 92.3.** Musculoskeletal injuries in dance. (Photographs copyright Craig C. Young, MD.)

- Pain posteromedial ankle: tenderness with active or passive motion
- May have painful crepitus with active motion
- Pain with resisted flexion of great toe
- May present with a palpable nodule behind medial malleolus and triggering of the great toe with plantarflexion

- Morton's neuroma and corns
  - Use of silicone spacers; check shoe size, especially pointe shoes; remember that each pair is handmade, thus variable in size.
- Bunion (hallux valgus), hallux rigidus, and hammertoes
  - Bunions are particularly a problem if the great toe is longer than second toe.
  - Avoid bunion surgery—minimal loss of motion may result in the ending of a dance career.
  - Hammertoe usually occurs in second toe, particularly if it is the longest toe.
  - Shoe padding can be used to help spread the load on the toes.
  - Silicone pad (great toe) or sleeve (toes)
  - Orthotic with rigid extension in nondance shoes (rarely fits into dance footwear except some character shoes)
- Paronychia and subungual hematoma
  - The range of appropriate nail length is small; too short—paronychia/ingrown nails; too long—subungual hematoma/nail trauma.
  - File nails daily to maintain optimal length.
- Sesamoiditis and sesamoid fracture
  - Especially common in folk dancers; 27.7% of stress fractures of the foot in Irish Dance are in the sesamoid.
  - Most likely linked to repetitive pounding in demi-pointe and pointe with jumps, landing, rhythm sequences, increased loading with forced turnout, and weak control of external rotation at the hip.
- Plantar fasciitis and turf toe
  - Especially common in Irish dancers
- "Stone bruise"—metatarsal head contusion
- Calluses and corns

## GENERAL TIPS FOR TREATING DANCERS

- Evaluate the dancer's schedule. Many hours are spent in solo class, team class, private lessons as well as cross-training.
- Evaluate the dance floor for overuse injuries.
  - Sprung floors have the best impact absorption.
    - This is particularly important because dance shoes have little or no cushioning.
  - Dancers may use different floors in the class area than in performance areas.
  - Dancers in smaller programs and folk dancers are much less likely to have the luxury of dancing on sprung floors.
    - 50% of Irish dance schools are not in traditional studios.
- Evaluate footwear worn when dancer is not dancing.
  - Alteration of this footwear may allow the dancer to continue dance activities by allowing for more effective relative rest when not dancing.
- If you don't understand the names of the motions, have the dancers describe or demonstrate them.
  - The knowledge of aggravating motions is critical in designing rehabilitation and return-to-activity plans.

- In general, avoid surgery in dancers because even the minimal loss of range of motion may result in the end of a dance career.
  - Exceptions are "excisional operations" (e.g., excision of os trigonum, osteophyte shaving for ankle impingement syndrome).
  - Dancers also recover well from meniscus and hip labral surgery.
- Respect dancers as artists, but also encourage them to think of themselves as athletes who need proper conditioning, nutrition, and rest.
- Injury consultation:
  - Most dancers will consult with someone within 1 week of injury.
  - However, less than half will consult a physician because:
    - They think that physicians are not helpful or do not understand dancers.
    - They are worried that they will be told to stop dancing for too long.
    - They do not have health insurance.
    - Many dancers consult other individuals such as fellow dancers, choreographer, company director, instructor physical therapists, chiropractors, massage therapists, and acupuncturists.
- Most dancers actually adhere to the advice given to them. Reasons for not adhering to the advice given to them include: the lengthy amount of time recommended to refrain from dancing, fear of being held out of class or rehearsal if the staff knew about the injury, not agreeing with the advice, and fear of losing their role in the performance to an understudy or rival.
- Most dance classes have multiple parts (barre vs. center class, warm ups vs. full class, strength training, etc.) and it is often not necessary to have a dancer stop dance class completely. Even the observation of class or a walkthrough of rehearsal is often beneficial and may lead to increased compliance with the treatment plan.

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Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 93 TRACK AND FIELD

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## INTRODUCTION

### Overview

- Track and field often attracts multisport athletes.
- The sport involves year-round competition and training.
- Differing athletic events subject athletes to differing demands. For example:
  - The shot put demands explosive power.
  - Endurance events demand high levels of aerobic conditioning and stamina.
  - Sprint distances (100 m, 200 m, and 400 m) demand explosive conditioning for power, flexibility, and anaerobic conditioning.
  - Middle distance races (800 m or 1500 m) demand a combination of anaerobic and aerobic conditioning to sustain power and stamina.
  - Distance events (3,000 m, 5,000 m, 10,000 m, and marathon) demand stamina, high levels of aerobic conditioning, and sustained power.
  - The 100 m and 400 m hurdles, as well as relays (4×100 m, 4×200 m, 4×400 m, 4×800 m, 4×1500 m, distance medley, and sprint medley), demand mental concentration, power, and stamina.
  - Field events, such as long jump, high jump, triple jump, shot put, javelin, discus, hammer, heptathlon, decathlon, and pole vault, as well as the track event 3000 m steeplechase, demand combinations of technique, stamina, power, and speed.

## EPIDEMIOLOGY

- It is difficult to look at injury statistics given the lack of a common denominator (player hours or athlete exposures) as well as variability in methodology of studies.
- There are no National Collegiate Athletic Association (NCAA) data collected for track and field.
- For large track and field events, injury incidence is the greatest for minor orthopedic injuries (5.7 per 1,000 athletes), followed by minor medical injuries (3.4 per 1,000 athletes). Medical coverage should be planned to accommodate seven major orthopedic injuries and two major medical injuries per 10,000 athletes.
- Male athletes and masters athletes are at greater risk of injury than other categories of athletes.
- Of all participants in the 1985 Junior Track Olympics, 35% reported the need for performance-related medical treatment.
- In general, **most injuries are overuse injuries that occur during training. However, the risk of injury during competition is four times higher than that in training.**
  - Acute injuries are often muscle strains or avulsions or are related to stress fractures; other acute injuries uncommon.
  - Majority of injuries involve lower extremities.
  - There is some risk for head and neck injury during the high jump and pole vault; there is a risk of blunt trauma with javelin, hammer, and discus.
  - Of particular concern are acute medical problems such as cardiovascular collapse secondary to underlying cardiac disease, dehydration, or other abnormalities related to environmental conditions.

### Site of Injury

- The most common injury sites are the lower leg (28%), thigh (22%), and knee (16%).
- The most common injuries are hamstring muscle strains and stress reactions.
- **The knee is the most commonly injured joint in runners** (48% of all joint injuries).
- Overuse injuries are more common in distance runners, whereas acute injuries are more common in sprinters, hurdlers, jumpers, and multievent athletes. Recurrent injuries are common.

### Specific Diagnoses

- Anterior knee pain accounts for 24% of running injuries in men and 30% of running injuries in women.
- Medial tibial stress syndrome accounts for 7.2% of injuries in men and 11.4% in women.
- Iliotibial band syndrome accounts for 7.2% of injuries in men and 7.9% in women.
- Patellar tendinosis accounts for 5.1% of injuries in men and 3.1% in women.
- Metatarsal stress syndrome accounts for 3.1% of injuries in men and 3.8% in women.
- Achilles tendinosis accounts for 4.7% of injuries in men and 2.7% in women.

## MUSCULOSKELETAL ISSUES

### Basic Running Mechanics

- It is important to understand the normal biomechanics of running to recognize abnormal biomechanics and how they can affect incidence of injury.
- **Foot strike:** At lower speeds, occurs with the heel, but at higher speeds, occurs with the forefoot
- Ground strike occurs 800 to 2,000 times per mile for the average runner (5,000 foot strikes per hour of running).
- **Reaction forces at foot strike are usually 1.5 to 5 times the body weight.** Joint shear forces during running increase to almost 50 times that of walking. These reaction forces are augmented considerably by different surface types.
- At the point of initial rearfoot contact, the foot is in supination. This is associated with the "closed-pack" position, a rigid positioning of the tarsal bones increasing stability.
- The foot then pronates with the tarsal joints, assuming an "open-packed" position, which is more accommodating and less rigid, allowing partial absorption of reaction forces. There is internal rotation of the tibia on the talus.
- As runners progress to the push-off, the subtalar joint supinates with external rotation of the tibia. The foot remains in supination during the airborne phase and forward swing of leg.
- Major muscle groups all show increased electromyographic activity during running, and all lower extremity joints show increased motion during running.
- In the stance phase of running, the ankle contributes 60% of the power generation, whereas the knee and hip generate 40% and 20%, respectively.

- **The knee is the principal shock absorber during running, absorbing twice as much energy as the ankle and hip.**
- **Abnormal biomechanics can lead to overload of other structures.** Commonly, gluteus maximus weakness can lead to poor control of the lower limb and reaction forces being transmitted throughout the kinetic chain. As another example, an abnormal amount of rearfoot varus or pronation can abnormally load structures higher in kinetic chain, leading to increased valgus stress at the knee. This is an important etiological factor in patellofemoral dysfunction (see the Patellofemoral Dysfunction section later in the chapter).
- **Orthotics** may help prevent injury if significant biomechanical abnormalities are present. Screen with gait assessment during the preparticipation examination

### Physiologic Issues

- **Demands depend on type of activity**
  - **Sprinters:** Energy requirements provided primarily by anaerobic energy pathways. Glucose is the major fuel source.
  - **Long distance events:** Energy requirements provided primarily by aerobic energy pathways, with fat and glucose derived from glycogen stores
  - **Middle distance and combination events**: Combination of both aerobic and anaerobic pathways
- Specificity of training, including periodization, to demands based on type(s) of energy pathways used.

### Strengthening and Conditioning

- **Sport specificity in training is key in track and field. This differs for each event.** Most training continues year round. There is a need to emphasize variability in training and avoid overtraining.
- **Endurance and sprint athletes:** The selective strengthening of certain muscle fiber types (fast-twitch vs. slow-twitch) demonstrates specificity (i.e., endurance-type training leads to changes in slow-twitch fiber morphology as well as enzyme metabolism specific for endurance-type activities). Similarly, explosive strengthening programs specifically train fast-twitch muscle fibers and the metabolic functions used to sustain these activities. It is more beneficial to train with sport specificity in mind when designing strength and conditioning programs.
- **Use of the entire range of motion (ROM) for strengthening is important.** Strength gains observed are specific to the range and speed at which strengthening exercises are performed.
- **Field events:** The successful transfer of ground reaction force through the foot, ankle, knee, hip, trunk, shoulder, elbow, wrist, hand, and finally, to the implement is critical to success in throwing events. Maintain quadriceps–hamstring balance. Again, sport specificity is helpful. A shoulder scapular stabilization and rotator cuff strengthening program will be helpful for the shot put, javelin, hammer, and discus. Reproduction of shoulder movement with manual resistance. Proprioceptive work is helpful in hurdles, discus, javelin, triple jump, and long jump.
- **Develop smaller supporting muscles:** Strengthening prevents development of medial tibial stress syndrome, plantar fasciitis, patellofemoral dysfunction, and other overuse injuries.

### Flexibility

- Despite a lack of reliable data about the effects of increased flexibility in preventing injury, most agree that the introduction and usage of a flexibility program helps avoid acute muscle strains. If the muscle length at which maximal stretch felt is greater, it takes larger acute overload to "stretch" the muscle past this length, leading to injury. Flexibility remains an essential tool in the treatment of muscle strains and joint protection.
- A flexibility program should be worked into strengthening programs such that strength is improved throughout the ROM without loss of motion.
- Ballistic stretching should be avoided.
- Stretching should be performed after warming up, rather than while "cold" at the start of practice.
- Stretch larger muscle groups first, followed by smaller groups.
- Hamstring flexibility is important in mechanical low back injuries. If hamstring flexibility is limited, a posterior pelvic tilt is created and the trunk flexion becomes limited, increasing stress in the lower back.

## COMMON MEDICAL PROBLEMS

### Preparticipation Physical Examination

- Stresses the importance of cardiac, musculoskeletal, and neurologic systems.
- Detailed family history is conducted, emphasizing any history of heart murmur, sudden cardiac death, Marfan syndrome, hypertrophic cardiomyopathy, or premature atherosclerotic disease.
- Screen for possible anatomic abnormalities that may predispose to injury; consider use of orthotics and a flexibility/strengthening program in the presence of muscle imbalances, especially of the hip abductors and gluteal musculature, assessed both statically and dynamically.
- Assess for nutritional or training errors, including any "self-restricted" food types. Inquire about lactose intolerance.
- Rule out significant medical or orthopedic problems that would preclude activity or require restrictions.
- In female athletes, pay special attention to menstrual dysfunction, hypocalcemic diets, a history of disordered eating, and/or a history of stress reactions or fractures (see the Female Athlete Triad section later in the chapter).

### Nutrition Issues

- It is important to consider nutrition when an athlete presents with symptoms of fatigue, burnout, or recurrent minor injuries (see Chapter 5: Sports Nutrition).
- Zinc, calcium, and iron are commonly deficient in athletes.
- Ideal nutritional intake: 6 to 13 g carbohydrates per kg body weight; 1.2 to 1.7 g protein per kg body weight; with the remainder of calories from fat. This translates into a diet of 60% to 70% carbohydrates, 10% to 15% protein, and 25% to 30% fat. No performance benefit has been shown with diets consisting of 15% fat versus diets consisting of 20% to 25% fat.
- If an athlete eats an adequate caloric intake from a variety of wholesome foods, nutritional needs are often met. Proper food selection, not supplementation, is the ideal form of nutrition.

#### Iron Deficiency

- Common in young athletes. Iron loss can be due to hemolysis with hemoglobinuria, gastrointestinal losses, and excessive sweating. Female athletes are at increased risk of anemia because of the additional loss that occurs with menses. Athletes are not immune from other medical problems, and thus a complete workup of an iron-deficient athlete is important.
- Iron deficiency or decreased iron stores can occur without anemia in as many as 24% to 47% of female athletes and 0% to 17% of male athletes.
- The dietary recommended intake varies based on sex and age. Females aged 19 to 50 years require 18 mg of iron per day, and males aged 19 to 50 years require 8 mg per day.
- Pseudoanemia: an increase in plasma volume of 6% to 25% with training results in hemoglobin and hematocrit appearing falsely low. Typically self-limited

- Screening for iron deficiency is recommended in elite athletes and high risk populations, such as vegetarian athletes, those with a history of iron deficiency, or athletes exhibiting an unexplained decrease in performance.
- Screening hemoglobin with a follow-up examination of ferritin is reasonable for assessing iron deficiency. Ferritin is a storage form of iron but can be transiently elevated in acute inflammation or following vigorous activity. Ferritin levels decrease, however, with longer term aerobic training. Iron and total iron binding capacity can differentiate pseudoanemia from true anemia.
- While anemia has been shown to affect athletic performance, the effect of iron deficiency alone is unclear.
  - Supplementation of 325 mg ferrous gluconate twice daily is recommended if truly iron deficient. Increase intake of iron-rich foods and ensure adequate vitamin C intake.

## Calories

- While obtaining an adequate total caloric intake is obviously essential, this basic ingredient of good nutrition is often neglected.
- In an attempt to eat "healthy," many athletes restrict fat intake. This strategy results in the risk of fat-soluble vitamins (vitamins A, D, E, and K) being deficient. Diets with less than 15% fat have shown no gains in athletic performance or health.
- Some athletes experiment by restricting calories as a means of reducing body weight, thus improving aesthetics and performance. This can increase the risk for developing a frank eating disorder, along with its concomitant medical problems.
- Other deficiencies found in athletes include zinc, magnesium, folate, as well as vitamins B6, C, and B12. Athletes should consider a nutritional consultation to formally review food intake if concerned about vitamin and/or caloric deficiencies.

## Protein Intake

- Often an issue in vegetarian athletes and those that restrict food intake
- Consult a nutritionist to ensure adequate intake.
- If vegetarian, protein complementarity (with legumes and grains) can ensure adequate protein intake, but it is still important to assess iron intake. Minimizing this potential deficit may minimize potential fatigue and diet-related conditions.
- Voluntary protein intake, along with fat and total energy intake, has been shown to be lower in athletes with menstrual irregularities than in normally menstruating athletes.

## Calcium

- Dietary reference intakes (DRI) vary based on age. The most recent guidelines suggest 1,300 mg daily for ages 14 to 18 years, and 1,000 mg daily for ages 18 to 50 (Fig. 93.1).
- Female athletes often consume less than the DRI for calcium.
- Calcium and estrogen are necessary in women for normal bone deposition. If depleted, this can lead to lower bone density. Peak bone density is reached in women in late teens to early 20s; thus, adequate intake in childhood and adolescence is critical.

## Vitamin D

- Vitamin D is essential for bone health and the incorporation of calcium. Primary sources are sunlight and vitamin D–enriched foods such as milk.
- Vitamin D levels have been increasingly associated with generalized musculoskeletal pains.

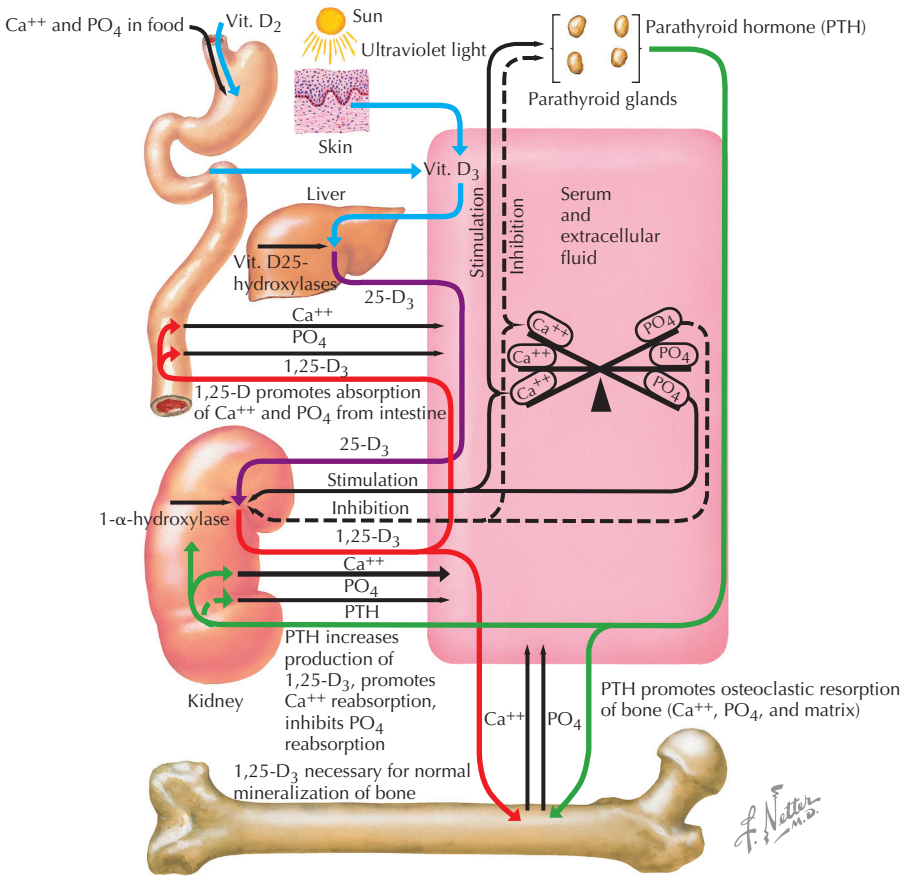



**Figure 93.1.** Normal calcium and phosphate metabolism.

- The DRI is 25 micrograms daily and vitamin D-25-OH levels can be monitored.
- Athletes who practice and compete in indoor sports and those living in Northern latitudes may be at risk**. Consider supplementation if levels are low.**

### Nutritional Supplements and Ergogenic Aids

- Athletes are at risk for use and abuse of supplements, as well as ergogenic aids.
- Some of these are restricted under US Olympic Committee and NCAA drug testing. Examples include ingesting excess amino acids, medium-chain fatty acids, vitamins, minerals, herb extracts, special proteins, and enzyme complexes.
- Often marketed to individual sports
- There are reports suggesting that up to 62% of track athletes use supplements, including multivitamins.
- Specific questions related to pharmacokinetics, interaction with normal foodstuffs and prescribed medications, and side-effect profile should be asked of the pharmacist.

#### SUPPLEMENTS

- The positive effects of supplementation are unproven if the athlete is not actually deficient in a vitamin.
- In the setting of dehydration, amino acid supplementation may be detrimental if kidney function is marginal.
- Protein and vitamin supplementation in great excess can be dangerous.
- Excesses of most vitamins are eliminated from the body.
- Fat-soluble vitamins are stored within the body, and thus toxicity is possible.
- Most methods of supplementation are expensive.

#### ERGOGENIC AIDS

- Erythropoietin, human growth hormone, and anabolic steroids
- Erythropoietin is detected by a urine test; new tests being developed to detect biosimilar formulations
- There is a blood assay to detect the use of human growth hormone.
- Anabolic steroids are detectable with urine drug testing.
- All are associated with significant side effects. Erythropoietin is associated with hyperviscosity syndrome and even death. Hyperviscosity syndrome is made worse with dehydration. Human growth hormone is associated with side effects that include acromegaly-like features and worsening of existing cardiovascular disease. Anabolic steroid side effects are well known.
- For up-to-date information, contact the US Anti-Doping Agency Drug Reference Line (800-233-0393).

## Fluid Considerations

- During prolonged exercise, 2 to 4 pounds of body weight are lost per hour; equivalent to 1 to 2 L per hour.
- The rate of dehydration can be estimated by changes in nude body weight; each pound of weight lost equals 450 to 650 mL (16 to 24 ounces) of dehydration.
- Dehydration can incur physiologic changes: for every liter of water (2.2 pounds) lost while exercising in the heat, there is an increase in core body temperature of 0.3°C, increase in heart rate of 8 beats per minute, and decrease in cardiac output of 1 L per minute.
- Proper rehydration is essential before, during, after, and between events.
- Pre-exercise hydration should consist of 500 to 600 mL of water or sports drink 2 to 3 hours prior to exercise, and 200 to 300 mL 20 minutes prior to exercise.
- To maintain hydration, ingestion of 200 to 300 mL every 10 to 20 minutes is required to prevent greater than 2% body weight reduction.
- Postexercise hydration should aim to restore fluid loss accumulated during the event.
- Thirst is a delayed sensation and therefore is not a good indicator of hydration status.
- Water temperature does not affect body heat storage, and thus water should be consumed at a comfortable temperature (10°C–15°C) to promote hydration.
- Carbohydrates should be replaced if the exercise session lasts longer than 45 minutes or is high intensity.
- For an athlete who weighs 68 kg (150 pounds), the carbohydrate requirement is 30 to 60 g per hour. Carbohydrate and fluid needs can be met by drinking 625 to 1,250 mL per hour of beverages with 4% to 8% carbohydrate content.
- For a glycogen-depleted athlete, a postrace carbohydrate intake of 1.5 g per kg body weight during the first 30 minutes postrace, and again every 2 hours for 4 to 6 hours will effectively replenish glycogen stores.
- Some studies document a decrease in gastric emptying once the glucose concentration is above 6%. Water is still an excellent source for short distance events (<1 hour).

### Nutritional Recommendations Are Different for Females and Males

- Societal influences have made constant dieting acceptable for girls and women, and athletes are even more likely to attempt to change body appearance if they think it will improve performance.
- Females at increased risk of nutritional deficiency, as well as eating disorders.

## Female Athlete Triad

- Three interrelated conditions: low energy availability (with or without an eating disorder), menstrual dysfunction, and altered bone mineral density, each on a continuum from healthy to disease state. (see Chapter 12: The Female Athlete; Chapter 27: Eating Disorders in Athletes) (Fig. 93.2).
- Present in every sport, but most commonly seen in sports that select for a lean body weight (swimming, cross-country skiing, cross-country running) or in sports scored subjectively (gymnastics, figure skating, diving)

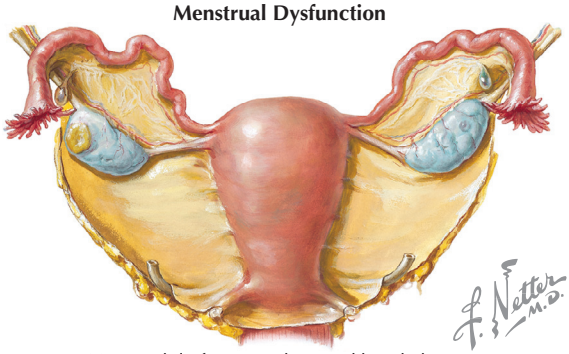
### Low Energy Availability/Disordered Eating

- When energy availability falls below a threshold due to an imbalance in energy expenditure, cellular maintenance, growth, and reproduction are disrupted.
- Origins are multifactorial: genetics, perfectionism, personality traits, identity, self-esteem, family dynamics, coping skills, control issues, alterations of body image, and/or need for control. Patients often have a history of sexual or physical abuse. Societal pressures increase risk.
- Inadequate caloric intake can also result from lack of knowledge of proper nutrition.
- Prevalence estimates of disordered eating among high school and college athletes range from 15% to 62%, compared with 13% to 20% in the general adolescent female population.
- Characteristics associated with successful athletes that lead to overlapping and increased risk for developing eating disorders include perfectionism, goal setting, and overachieving.
- Subtle messages by coach, parents, or teammates (often unintentional) can add to pressures that lead an athlete to experiment with pathogenic weight control behaviors:
  - Misconception that lower body weight improves performance
  - Educating coaches in how to recognize and prevent eating disorders is essential.
- Difficult to identify; a team approach is often necessary for proper treatment

**Eating Disorders**

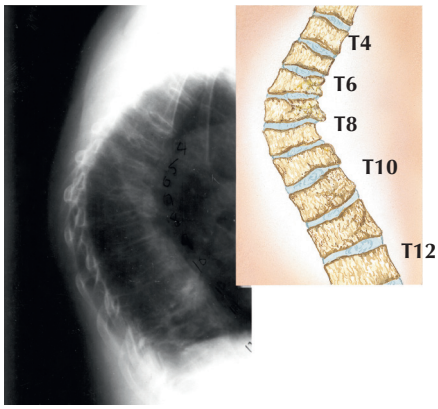
Origins multifactorial: perfectionism, personality traits, identity, self-esteem, family dynamics, coping skills, control issues, alterations of body image, need for control. Patients often have history of sexual or physical abuse. Male and female athletes should be screened for disordered eating. More common in sports in which "lean" aesthetics are favored (gymnastics, cross-county, etc.)

**Menstrual Dysfunction**




Menstrual dysfunction, shortened luteal phase, anovulation, oligomenorrhea, and amenorrhea can occur in response to chronic exercise and are associated with decreased bone mineral density (BMD) or low bone mass. Detailed menstrual history should be obtained.

**Osteoporosis**




Low BMD risk factor for early osteoporosis and stress fractures. Increased incidence of stress fractures seen in runners with amenorrhea. These athletes are at increased risk for decreased lifetime peak bone mass, which can put them at risk for postmenopausal osteoporosis.

**Figure 93.2.** Female athlete triad.

- The treatment team usually includes a physician, psychiatrist, and nutritionist. Additional support system includes coaches, sport psychologists, athletic trainers, and families. Psychological counseling is a cornerstone.
- Treatment usually not very successful, emphasizing the need for prevention through education and early identification.
- Screen for concomitant depression. Preliminary studies suggest a favorable response to antidepressants such as fluoxetine.

### Menstrual Dysfunction

- Common in athletes, especially endurance athletes
- Menstrual dysfunction may include a shortened luteal phase, anovulation, oligomenorrhea, or amenorrhea.
- Strenuous training alone does not cause menstrual dysfunction; requires concurrent dietary restriction.
- Exercise-associated menstrual dysfunction remains a diagnosis of exclusion.
- The physician must rule out other conditions such as pregnancy, thyroid disorders, adrenal disorders, prolactin-secreting tumors, and/or ovarian disorders.
- Important to initiate workup and treatment because of the long-range consequences of menstrual dysfunction. Treatment must be individualized.
- "Progesterone challenge" helpful in functionally differentiating an estrogen-deficient state from an estrogen- and progesterone-deficient state.
- If exercise-associated amenorrhea is diagnosed and no contraindication exists, estrogen and progesterone supplementation should be considered. Many use age 16 as the younger cutoff for initiating treatment.
- The oral contraceptive pill has a good side-effect profile, is well tolerated, and has convenient packaging. If positive progesterone challenge, can use monthly progesterone alone
- Other considerations: decrease training intensity; increase body weight if underweight; assess nutritional intake; maintain high index of suspicion for eating disorders

### Osteoporosis and Stress Fractures

- The bone is constantly remodeling; the process is a balance between osteoblast and osteoclast activity.
- Amenorrheic runners have lower estrogen levels and lower bone mineral density (BMD) than runners with normal menstrual cycles.
- An alteration in homeostasis due to low energy availability and low estrogen levels, which increases osteoclast activity, leads to decreased BMD.
- Stress fractures are caused by increased stress on normal bones (stress fractures), or normal stress on abnormal bones (insufficiency fractures) (Fig. 93.3).
- Low BMD is a risk factor of early osteoporosis and stress fractures. An increased incidence of stress fractures is seen in runners with amenorrhea. These athletes are at increased risk of decreased lifetime peak bone mass, which can put them at risk of postmenopausal osteoporosis.

### Recognition and Treatment

- **Detection and education is critical.**
- A preparticipation physical examination offers a good opportunity to address these issues with female athletes and provides an opportunity to educate young athletes about the importance of maintaining normal menstrual function, risks of eating disorders, and the relationship of both to incidence of stress fractures and early low bone mass.
- Ask athletes who present with stress fractures about current and past menstrual history.
- Supplemental history for female athletes is a helpful screening tool during preparticipation physical examination.

## Male Athlete Triad

- Parallels female athlete triad: inadequate nutrition, hypogonadotropic hypogonadism, and low bone mineral density. There is a lack in the quality and quantity of studies in males.
- Similar to female athlete triad, each condition falls on a continuum of healthy to disease state
- More prevalent in sports that emphasize leanness (running, cycling, wrestling, judo, horse racing)
- Subset of male endurance athletes has been shown to have lower testosterone levels and sperm counts.

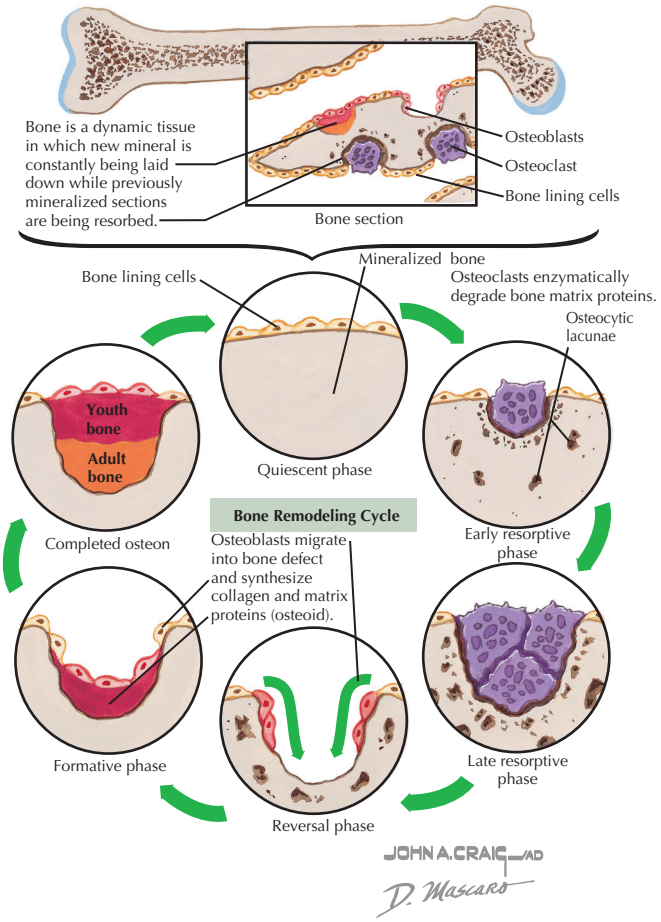



**Figure 93.3.** Bone remodeling.

- Screening for hypogonadism in male athletes has been proposed; however, no current evidence-based guidelines incorporate risk stratification or screening for male athletes.
- Proposed screening for athletes with recurrent stress fractures, especially in the presence of low BMI, includes scanning dexamethasone, 25-hydroxyvitamin D, and free and total testosterone levels.

## Overtraining

**Description:** A spectrum of adaptations that includes functional overreaching, nonfunctional overreaching, and overtraining syndrome, where the balance of energy expenditure and recovery is disrupted (see Chapter 28: Overtraining).
- **Functional overreaching (FOR):** intensified training accompanied by a temporary decline in performance. With appropriate rest periods, the process leads to overall enhanced performance.
- **Nonfunctional overreaching (NFOR):** a *short-term* (several days to several weeks) decrease in performance related to stress from both training and nontraining sources that may lead to maladaptation
- **Overtraining syndrome (OTS):** a *long-term* (several weeks to several months) decrease in performance related to stress from both training and nontraining sources that may lead to maladaptation

**Symptoms:** Nonspecific, insidious symptoms include fatigue, mood disturbance, sleep difficulty, and performance decline.

**Diagnostics:** Generally a diagnosis of exclusion; presence of normal laboratory testing results. Important to assess for anemia, hypothyroidism, infection, collagen vascular disease, glucose abnormalities, and/or hormone deficiencies.

**Treatment:** Decrease in training, increase in carbohydrate intake, and a gradual return to activity. Sports psychologist referral should be made.

**Associated conditions:** Overtraining contributes to burnout, overuse injuries, stress fractures, menstrual dysfunction, iron deficiency, and long-lasting decrements in performance.

## Syncope

**Description: Common medical problem with multiple causes**

**Evaluation:** Need to be rigorous in excluding the possibility of a serious medical problem, especially if syncope occurs in the midst of full exertion. Ask about history of associated chest pain, palpitation, shortness of breath, dizziness.

**Differential diagnosis:**
- Dehydration
- Cardiac sources: long Q-T interval, arrhythmias, hypertrophic cardiomyopathy, anomalous coronary arteries, aortic stenosis or other valvular abnormalities, neurocardiogenic syncope
- Neurologic sources: seizures, arteriovenous malformations, aneurysm
- Hematologic (anemia) or electrolyte abnormalities (red flag for eating disorders).

**Diagnostics:** History is essential because syncope during exertion is much more concerning than syncope occurring after full exertion or standing still (common in neurocardiogenic syncope). Requires further workup and testing as indicated by history, family history, and physical examination. Often difficult to differentiate pathologic cardiac condition from "athlete's heart," which is a physiologic response to training. Both share common symptoms such as: hypertrophy, prolonged electrocardiographic intervals, functional (nonpathologic) flow heart murmurs. Echocardiography, maximal exercise testing, and tilt-table testing are often useful adjuncts to the history and physical examination.

**Return to play:** Guidelines are unclear, but serious, life-threatening abnormalities must be ruled out first.

## Gastrointestinal Problems

**Alterations in gastrointestinal (GI) function:** 25% to 50% of runners experience abdominal cramps, diarrhea, nausea, and abdominal pain. Blood flow to the gut typically decreases 50% during exercise.

**Diarrhea:** Typically self-limited and physiologic, tending not to cause dehydration or electrolyte imbalances. Antidiarrheal medications should be avoided whenever possible.

**Celiac sprue (gluten-sensitive enteropathy):** Can lead to decreased bone mass secondary to poor absorption of calcium. Athletes will typically complain of bloating and/or diarrhea and may have a family history. Consider this condition in athletes with these chronic symptoms. The athlete may also have electrolyte abnormalities and vitamin deficiencies. A diagnosis is made with serologic testing for immunoglobulin A and immunoglobulin G antitissue transglutaminase. If the test is positive, then a small intestinal biopsy is performed to establish a definitive diagnosis.

**GI bleeding:** Severe GI bleeding after endurance running have been reported, with rare occurrences of death secondary to acute hemorrhage, although these are reported as isolated cases. Reports of bright red blood per rectum must be rigorously worked up, and ulcerative colitis and Crohn's disease must be ruled out. Occult forms of GI bleeding are common. Studies have reported anywhere from 8% to 22% rates of clinically detectable bleeding in marathoners after a race. If more sensitive

assays are used, an increase in fecal heme concentrations is seen in 83% of marathoners. Thus, runners have increase in stool heme after race, and in approximately 20% it is detectable clinically. Bleeding is typically self-limited and resolves in roughly 72 hours. Bleeding can be associated with excessive iron loss and resultant iron deficiency, but must be differentiated from Runner's pseudoanemia. Pathophysiology is unclear, but the physician must consider nonsteroidal anti-inflammatory drug (NSAID) use, bowel ischemia, the traumatic shearing effect from running, and underlying GI abnormalities. A postexercise biopsy of colon should be performed to reveal congestion and vascular lesions. The biopsy should be evaluated with appropriate imaging techniques. After GI pathology has been ruled out, treatment issues surround presence/absence of iron deficiency and consideration of antimotility agents for diarrhea. Antidiarrheals inhibit heat dissipation and should be used with caution.

**Reflux/delayed gastric emptying:** A common complaint in endurance athletes, and may result in nausea or vomiting. Exercise increases acid secretion, and may lead to heartburn-type symptoms in some athletes. Symptoms can be exacerbated by precompetition nervousness. Thorough workup indicated to rule out GI pathology. Treatment centers on alteration in eating patterns, trials with magnesium or aluminum hydroxide and simethicone (Maalox), or $H_2$-blocker trial (1 hour before events).

### Exercise-Induced Asthma

**Description:** Common in athletes, especially when exercising in cold. **Should be discussed during preparticipation physicals, including severity and current medications**

**Symptoms:** Variable and occurring with exercise: wheezing, tightness, chest pain, shortness of breath. Symptoms can sometimes be vague: poor exercise tolerance, cough or tightness after exercise. Symptoms aggravated by allergens, upper respiratory infections, and environmental conditions (humidity, cold air).

**Diagnostics:** Provocative testing helps make a diagnosis and assess response to bronchodilators: spirometry before exercise challenge, adequate exercise challenge (use whatever athlete describes as "typical" precipitant), postexercise spirometry. Look for a decrease in forced expiratory volume in the first second ($FEV_1$) and a decrease in $FEV_1$ as a fraction of total forced vital capacity.

**Treatment:** Beta adrenergic receptor agonists, such as albuterol, are often helpful as pre-exercise medication to prevent exercise-induced asthma. Other medications include sodium cromolyn (Intal) or nedocromil sodium (Tilade) as premedication. Inhaled corticosteroids or the three to four times daily use of medications tends to be helpful for acute flaring of symptoms, baseline asthma (not only exercise-induced asthma), or allergen-induced asthma. Avoid known precipitants. Proper cardiovascular warmup can lessen symptoms and allow athlete to "run through" asthma: 15 to 20 minutes at approximately 70% $VO_2$ max, using a series of 40- to 50-yard sprints. Minimize symptoms: use a scarf or other methods to warm inspired air; breathe through nose.

**Drug testing concerns:** The drugs permitted for the treatment of asthma change frequently. Athletes should be made aware of the acceptability of their current medications. All medication questions should be addressed to the US Anti-Doping Agency Drug Reference Line (800-233-0393).

### Renal Issues

**Pseudonephritis:** Proteinuria, hematuria, and presence of cellular elements in the urine after exercise. Exercise is associated with decreased renal blood flow, and when coupled with dehydration, results in a decreased glomerular filtration rate as exercise continues. Proteinuria, hematuria, and pyuria, as well as cellular elements seen after intense exercise, are self-limited. Workup indicated if abnormalities persist after discontinuing exercise, or if other symptoms or risk factors exist.

**Gross hematuria:** Reported in runners and often occurs without warning and without symptoms as painless clots of blood or grossly bloody urine. Possibly due to a traction effect on bladder. Cystoscopy can sometimes detect the source of bleeding but is often negative. Further workup indicated if abnormalities persist despite stopping exercise, or if other symptoms or risk factors are present.

## COMMON MECHANISMS OF INJURY

### Precipitating Factors

- **Training errors account for approximately 80% of injuries** (Fig. 93.4). Changing to a harder running surface, an abrupt increase in training mileage (10%), an abrupt increase in training intensity, hill running, consistent running on crowned roads, previous injury, inadequate rest/nutrition
- **Uncorrected anatomic problems** include hip, pelvis, back, knee, foot, ankle, and leg length discrepancy.

### Overuse

- Most common mechanism in track and field injuries
- Classified according to timing of pain in relation to onset of activity
  - Type 1: Pain after activity
  - Type 2: Pain during activity, not restricting activity
  - Type 3: Pain during activity, restricting activity, which restricts performance
  - Type 4: Chronic, unremitting pain

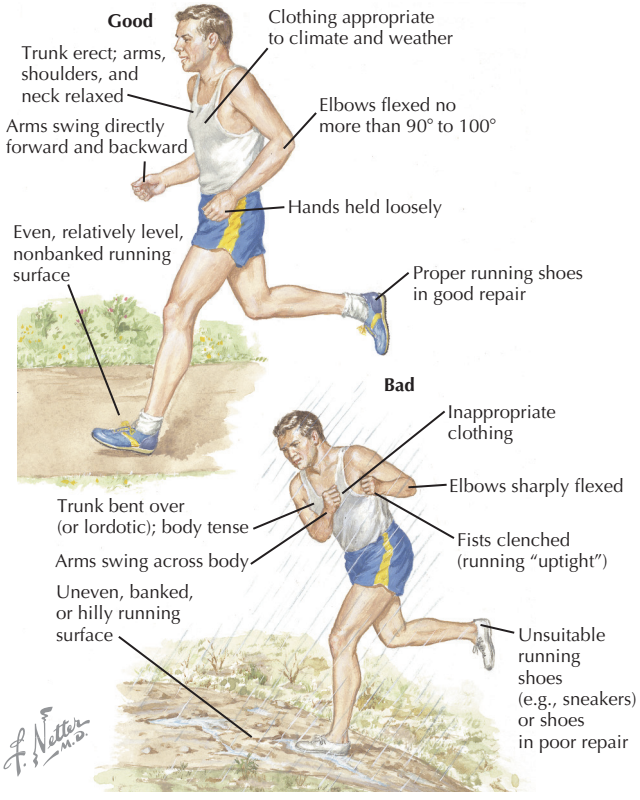



**Figure 93.4.** Good and bad running practice.

## SPECIFIC MUSCULOSKELETAL INJURIES

### Knee

**The knee accounts for 30% to 50% of all track and field injuries.**

#### Iliotibial Band Syndrome

**Description:** Common in runners. Iliotibial band inserts into Gerdy's tubercle along the lateral tibia (Fig. 93.5). Often seen in conjunction with greater trochanteric bursitis

**Presentation:** Lateral knee pain, often in midrange of knee flexion from 20 to 70 degrees, when the iliotibial band rubs across lateral femoral condyle

**Evaluation:** Contributing anatomic factors (should be corrected if possible): leg length discrepancy, abnormal foot biomechanics (especially hyperpronation), tibia vara, scoliosis. Tightness of the musculature and/or muscle strength imbalances often seen; important to treat with corrective rehabilitation

**Diagnostics:** Ober's test is useful for identifying tight iliotibial bands. Can reproduce the pain by resisting knee extension and looking for painful arc. This is sometimes difficult to differentiate from trochlear articular surface or an osteochondral defect.

**Treatment:** Stretching, ice, oral NSAIDs, phonophoresis, iontophoresis. Avoid running on beveled surfaces. Consider the use of corticosteroid and anesthetic agents if other measures fail.

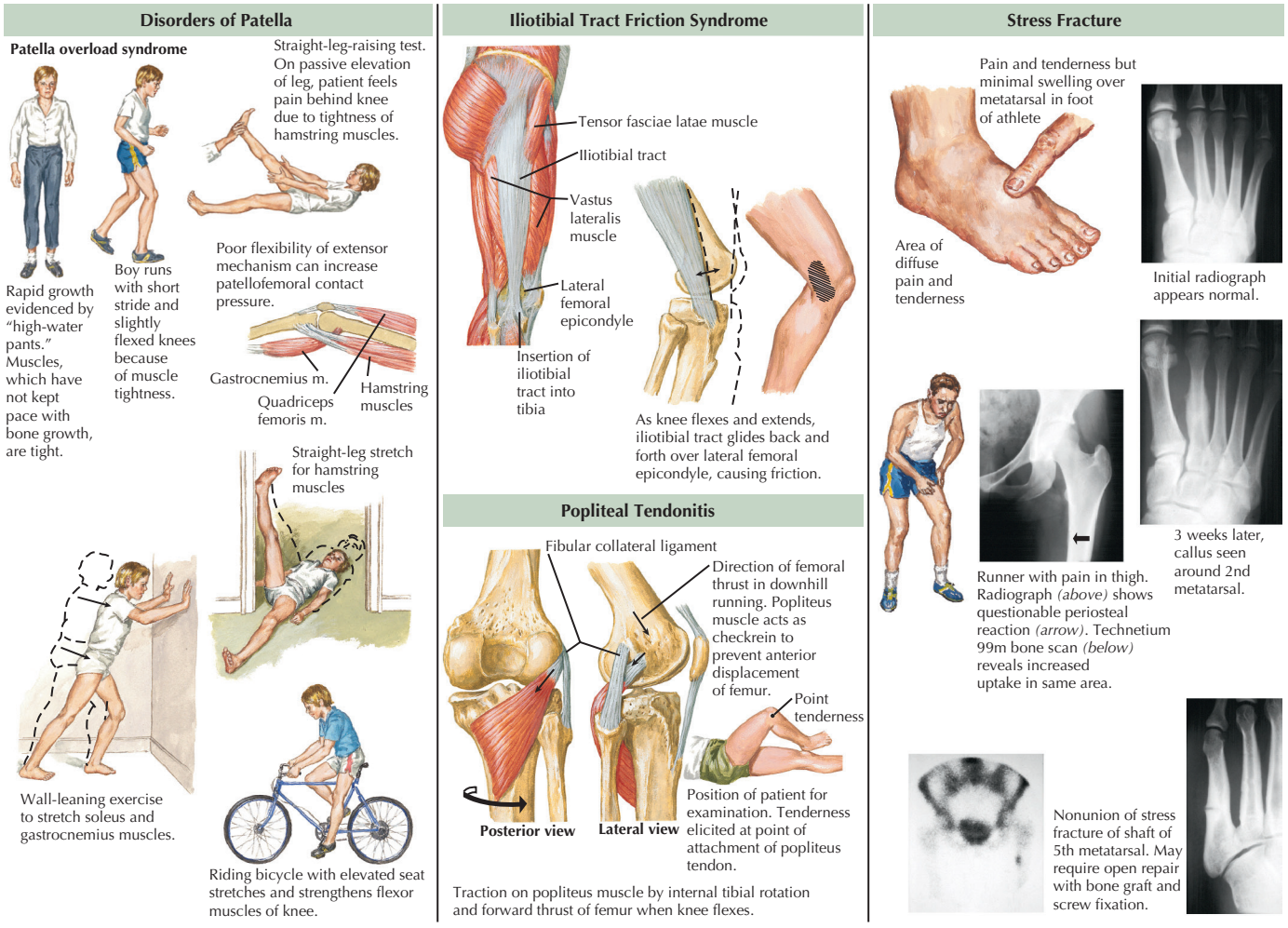



**Figure 93.5.** Lower extremity injuries.

### Patellofemoral Dysfunction

**Description:** Generally thought to be caused by abnormal tracking of the patella with resultant patellofemoral irritation and pain (see Fig. 93.5). The proposed mechanism of patellofemoral dysfunction suggests that it results from activity that pushes the tissues of the knee beyond a "zone of homeostasis" rather than biomechanical alignment problems. Understanding the static and dynamic orientations of the patella is important in understanding the nature of a tracking dysfunction, as well as in guiding rehabilitative treatment.

**Contributing factors:** Increased Q angle, deficient vastus medialis obliquus (VMO) or tight vastus lateralis musculature, patella alta, pronation (increases the functional Q angle), genu valgum, and recurvatum

**Presentation:** Anterior knee pain, which often made worse by climbing or usually descending stairs, or prolonged sitting ("theater sign")

**Treatment:** Mainstay remains the strengthening of the VMO along with improved flexibility of lateral structures. There are various patellar taping methods to allow the patella to track more normally and facilitate pain-free strengthening of the VMO. Unclear how functionally effective taping is. Correction of leg length discrepancy or excessive pronation or other abnormal foot biomechanics, if present. Closed-chain kinetic exercises can used in combination with physical therapy modalities, including ice, to decrease inflammation. Biofeedback is often helpful.

### Patellar Tendinosis

**Description:** Common, especially in activities requiring jumping

**Symptoms:** Pain along the infrapatellar tendon, inferior pole of patella, or insertion of the patellar tendon into tibial tubercle; pain during knee extension, especially terminal extension

**Treatment:** Ice, stretching, NSAIDs, modalities (phonophoresis, iontophoresis), and decreased activity. Can use alternate cardiovascular equipment (bicycle, aqua arc, swimming, aqua-jogging). Avoid activities that aggravate pain, such as jumping and plyometrics.

### Infrapatellar Fat Pad Impingement

**Presentation:** Anterior knee pain, similar in presentation to patellofemoral syndrome, except typically worse with standing and rapid knee extension. Often associated with an acute hyperextension injury

**Examination:** Reveals tenderness at or below the inferior pole of the patella, often worse with passive, ballistic hyperextension

**Treatment:** Patellar taping, often by compressing the fat pad even further

### Hamstring, Adductor, Quadriceps Strains

**Description:** Common, especially in sprinters

**Prevention:** Eccentric hamstring exercise programs have been shown to reduce the risk of hamstring injury; specific exercises include Nordic hamstring exercises and yo-yo curls. Risk factors retrospectively associated with adductor strain include relative adductor weakness and decreased hip abduction range of motion.

**Presentation:** A sudden pain in belly of affected muscle. Athletes often describe "pulling" or "tearing" sensations. Athletes often run through initial discomfort only to experience increasing pain and disability later.

**Examination:** Variable amounts of swelling and ecchymosis, depending on extent of muscle damage as well as exact location of injury. The size of the hematoma and cross-sectional damage can be assessed with MRI, though it does not correspond well with future risk of injury.

**Most common hamstring injury:** Short head of biceps femoris. Most often occurs at the proximal musculotendinous junction. As the athlete's velocity increases, the hamstrings take an increasingly active role in force production, leaving them prone to injury. Injuries typically occur during the eccentric phase of high-velocity movements. Although the data are speculative, incidence of hamstring strains appears to increase with inadequate flexibility, quadriceps-hamstring muscle imbalances, inadequate warm-up, prior injury, poor proprioception, and fatigue.

**Treatment: All of these are extremely difficult to treat. Initial management:** compressive wrap, ice, stretching. **Subsequent management:** use of NSAIDs, possibly ultrasound if scarring occurs. **Rehabilitation** should emphasize eccentric activity, reproduction of sport-specific demands, and maintenance of adequate ROM.

**Prognosis and return to play:** Gradually, as long as athlete has full range of motion, full strength, and normal functional testing. Recurrence is common and often frustrating for the athlete. Progression back to full activity must be slow. Use of sport-specific progression helpful

## Lower Leg, Foot, and Ankle

### Medial Tibial Stress Syndrome

**Description:** Pain over the medial third of the posteromedial tibia; association with excessive pronation

**Presentation:** The area of tenderness is often located in the soleus muscle, which is involved in the inversion of subtalar joint and plantar flexion of ankle joint.

**Diagnostics:** X-ray findings have poor sensitivity (10%) in the early stages of stress reactions. Findings are typically very subtle and include the thickening of medial tibial cortex common on plain radiographs; myositis and tendinosis are evident on bone scan (differentiated by increased blood pool phase). Order further imaging if clinical suspicion is high. Scintigraphy helps identify stress fractures. Plain radiographs may show evidence of stress fracture after 14 days, while bone scan is very sensitive but lacks specificity (statistics depend on site of fracture). Computed tomography (CT) scans with thin cuts over the suspected area are an option. However, false negatives are common and stress reactions are not well visualized. Furthermore, radiation exposure is involved. MRI has several advantages: no radiation exposure, multiplanar imaging, high-sensitivity, precise location of abnormalities, and the ability to detect early pathology. There are some reports of false-positive MRI results in asymptomatic patients. Fat-suppressed or short tau inversion recovery (STIR) images can be ordered. May also have the radiologists use MRI-visible markers to localize the painful area. Cost of bone scan and MRI varies based on institution.

**Associated injuries:** Progression from medial tibial stress syndrome to stress fracture is a major concern; treatment is aimed at preventing this.

**Treatment:** Ice; assessment of biomechanical abnormalities, focusing specifically on the need for orthotics; eccentric strengthening of antagonistic muscle groups and flexibility exercises, relative rest with alternative methods of training is useful (cycle, swim, aqua-jogging, Zuni unloader). Pain serves as a useful guide in injury management. Day-to-day, week-to-week adjustments in training regimen are often necessary. Athletes may continue pain-free activities (swimming, cycling, hand-cycle, pool-running, ellipticals). Return to play is dictated by patient's ability to be pain-free during activities. Avoid NSAIDs, as they are felt to inhibit osteoblast activity.

### Fractures and Stress Fractures

**Description:** Typically associated with mileage of more than 20 miles per week, changes in training volume and intensity, or underlying bone abnormalities (see Fig. 93.5).

**Treatment:** Depends on site, extent, and circumstances. Discontinue the offending activity, unload the affected extremity (walker-boot, crutches, even a wheelchair if bilateral involvement is present). Analgesia: NSAIDs have been shown to inhibit osteoblast activity *in vivo* in a rat model. Therefore, a non-NSAID may be a wiser choice. The same study demonstrated that low-intensity pulsed ultrasound facilitated the healing of stress fractures. Human studies on the efficacy of bone stimulators, via pulsed ultrasound or electrical field stimulation, are less conclusive. Low intensity pulsed ultrasound did not affect the healing of stress fractures in a study of 26 midshipmen. Electrical field stimulation hastened healing in a small subset of acute, severe stress fractures but had no overall impact on healing for the whole study population. Consider use of a sports psychologist and nutritionist for support. Locate and correct any biomechanical abnormalities with orthotics, shoe-type changes, assess shoe age, and consider physical therapy for stretching and strengthening. Bisphosphonates should be used with extreme caution, given the negative impact on reproductive health; the agent has very little role in women of reproductive age. The navicula, tibia, and metatarsals are the most common sites of injury in track athletes. Distance runners are prone to tibial and fibular stress fractures. Studies suggest females are more susceptible than males. Likely multifactorial: the female athlete triad, baseline fitness level differences, training errors, and body geometry differences have all been cited as possible causes.

TUBEROSITY AVULSION FRACTURES

**Description: Most common fracture of the proximal fifth metatarsal** (Table 93.1). Often occurs where the plantar aponeurosis or peroneus brevis attaches. Fracture displacement rare

**Evaluation:** Look for an associated lateral malleolus fracture. Can be differentiated from unfused tuberosity apophysis by the smoothness and orientation of the fracture line (fracture often perpendicular to shaft of bone). Differentiated from os peroneum by smoothness of edges and frequent presence of bilaterality

**Treatment: Primarily symptomatic**. Can immobilize using a cast or molded hard-sole shoe for 1 to 2 weeks, followed by protected weightbearing to tolerance and close follow-up

JONES FRACTURE

**Description: Acute forefoot injury of the proximal 5th metatarsal without prodrome. However, likely an acute-on-chronic phenomenon in an area of consistent microtrauma.** Occurs at the junction of metaphysis and diaphysis; no intermetatarsal extension

**Treatment:** Varies. Conservative management with casting for 6 to 8 weeks. If no improvement is observed, surgical consultation should be ordered.

PROXIMAL FIFTH METATARSAL DIAPHYSEAL STRESS FRACTURE

**Description:** A fracture resulting from repetitive cyclic forces applied to foot

**Presentation:** Can have prodromal symptoms or acute or chronic presentation

**Classification:** Torg classification helps differentiate stress fractures by their potential to heal (see Table 93.1).

Acute, early diaphyseal stress fracture (periosteal reaction)
Delayed union (lucent fracture line and medullary sclerosis)
Nonunion

**Treatment:** Depends on patient needs and expectations.\

- Type I: treat as an acute nondisplaced Jones fracture
- Type II: treat operatively with bone graft or medullary screw fixation
- Type III: treat operatively; some prefer closed cannulated medullary screw fixation, others open methods

OTHER STRESS FRACTURES

**Description:** Runners account for 69% of all stress fractures because of increased stresses on normal bone, as well as normal to increased stresses on weakened bone. Injury can occur throughout the lower extremity: tibia (34%), fibula (24%), metatarsals (18%), femur (14%), and pelvis (6%).

**Evaluation and treatment:** Pay particular attention to the location of femoral neck fractures. Fractures occurring on the superior/tension side of the femoral neck require prompt surgical evaluation. Fractures on the inferior/compression side of the femoral neck can be managed more conservatively.

**Associated injuries:** Associated with gait abnormalities, leg length discrepancy, poor surface, incompletely rehabilitated prior injuries, reduced bone mineral density, training errors, and abrupt changes in the volume or intensity of training

## Exertional Compartment Syndrome

**Description:** Reversible ischemia caused by a noncompliant osseofascial compartment that does not appropriately respond to the expansion of muscle tissue during exercise

**Presentation:** A tight cramping ache over the involved compartment, with onset at a reproducible point during exercise, and resolution with rest. Presentation in the anterior compartment is most common (45%), followed by the deep posterior (40%), lateral (10%), and superficial posterior compartments (5%). Can present with associated weakness and paresthesias

**Diagnostics:** Compartment pressures can be measured by a variety of methods, including a needle manometer, wick catheter, slit catheter, continuous infusion, and solid state transducer intracompartmental catheter. Criteria for diagnosis include a pre-exercise pressure 15 mmHg or greater, 1-minute postexercise pressure 30 mmHg or greater, and 5-minute postexercise pressure 20 mmHg or greater. The more criteria met, the greater the confidence in the diagnosis. Testing may require

**TABLE 93.1 FOOT FRACTURES**

| Fracture Type | Mechanism | Location | Chronicity | Prognosis |
|---|---|---|---|---|
| Avulsion | Hindfoot inversion | Tuberosity | Acute | Excellent |
| True Jones | Forefoot adduction | Metaphyseal-diaphyseal junction | Acute | Good |
| Diaphyseal stress | Cyclical loading | Proximal diaphyses | | |
| Torg type I | | Narrow fracture line, no medial sclerosis | Acute or chronic | Fair-good |
| Torg type II | | Wide fracture line, some medial sclerosis | Delayed union | Variable |
| Torg type III | | Complete medullary sclerosis | Nonunion | Variable |

Adapted from Quill GE. Fractures of the proximal fifth metatarsal. *Orthop Clin North Am*. 1995;26(2):353-361.

sport-specific exercise to reproduce symptoms. Less-invasive alternatives currently being explored include ultrasound, triple phase bone scan, MRI, and near-infrared spectroscopy.

**Treatment:** Treatment can be surgical or conservative. Conservative measures include relative rest (limiting exercise to produce minimal symptoms), anti-inflammatories, manual therapy, stretching, and strengthening. If symptoms persist beyond 6 to 12 weeks despite conservative treatment, or if diagnostic testing demonstrates severely elevated pressures, surgical release is recommended. Surgical remediation involves fasciotomy, with or without fasciectomy, by various techniques. Return to play period is approximately 3 months.

### Peroneal, Anterior Tibialis, and Posterior Tibialis Strains

**Description:** Feature as common overuse injuries, as well as acute overload injuries and strains. **Usually caused by training errors,** increase in training, change in shoes, poor flexibility

**Diagnostics:** Can see avulsions or partial tears, especially of the peroneal tendons. Peroneal tendons run in the common tendon sheath until just posterior to the lateral malleolus, then bifurcate and run in their own tendon sheaths. MRI helps evaluate significant tendon injuries. Tendinosis is rarely detected by MRI. Musculoskeletal ultrasound is increasingly used to diagnose these types of soft tissue injuries, but is not yet standard care.

**Treatment:** Ice, stretching, addressing training errors or foot and ankle biomechanics

### Plantar Fasciitis

**Description:** Microtears caused by traction of the plantar fascia and associated structures at the calcaneal insertion

**Presentation:** Heel or arch pain, often worst with first steps after getting out of bed

**Examination:** Seen in both pes planus and pes cavus, although the latter is more common. A tight gastrocnemius–soleus complex and plantar flexors, as well as excessive pronation, increase the risk of developing plantar fasciitis. Pain to palpation along the plantar fascia, often at insertion into calcaneus. Pain with resisted toe flexion and passive toe extension.

**Imaging:** Radiographs may demonstrate heel spur, but this often presents bilaterally despite unilateral symptoms.

**Treatment:** NSAIDs, ice massage, arch stretches, heel cushioning, counterforce taping, phonophoresis or iontophoresis. Corticosteroid injections can be useful if other modalities are not helpful but may increase risk of rupture. Surgery can be used to release the plantar fascia in severe, refractory cases. **Night splints** are useful to keep the plantar fascia at length by placing the foot at 90 degrees of dorsiflexion. Stretching of the gastrocnemius–soleus complex and hamstrings are also important, as well as intrinsic strengthening of foot musculature. **Arch supports or orthotics can be used if other rearfoot abnormalities present.**

### Heel Pain: Fat Pad Contusion, Heel Spurs

**Description:** Common in hurdlers, jumpers, and endurance runners

**Presentation:** Pain along fat pad or at insertion of the plantar fascia into the calcaneus. Patients often have history of increased training or increased shoe wear and tear without replacement.

**Evaluation:** Consider calcaneal stress fracture in the differential. Presence of an exaggerated heel strike in gait is common.

**Treatment:** Ice massage, NSAIDs, shoe modification: soft cushioning, replacement of worn shoes (recommend changing shoes every six months or 300 to 500 miles)

### Achilles Tendinopathy

**Description:** Common in events involving jumping or landing, as well as sprinting. Tendonitis represents an inflammatory process, whereas tendinosis refers to chronic intratendinous degenerative changes without acute inflammation. Pathology reveals no inflammatory cells involved.

**Evaluation:** Pain often presents approximately 2 cm proximal to the insertion of the Achilles tendon into the calcaneus or distal tendon. Palpation of nodularity may signify mucinous degeneration of the tendon and/or a partial tear. Can palpate crepitus along tendon sheath with a passive or active motion ("squeaking tendon"). Pain along the tendon is accompanied by resisted plantarflexion and passive dorsiflexion. A positive **Thompson test** signifies a complete tear. The patient is placed prone and the gastrocnemius is squeezed, which creates an automatic plantarflexion of the foot. A positive test occurs if no plantarflexion occurs. Test with the knee in full extension, as well as 90 degrees of flexion, to confirm the diagnosis.

**Treatment:** Ice, relative rest, NSAIDs, bilateral heel lifts to put tendon at rest, eccentric rehabilitation program that emphasizes stretching, return to functional activities, slow progression. Modify activities to pain tolerance. Assess for training errors or anatomic precipitants. Treatment of Achilles rupture should be individualized. For a competitive athlete, surgical treatment may be the better option for an earlier functional recovery. Surgical debridement is sometimes considered for recalcitrant tendinosis.

## Hip and Spine

- Hip and spine injuries **account for close to 20% of all injuries** in track and field athletes.

### Muscular Imbalance

**Description: Common cause of muscular strains and overuse injuries.** Unclear cause but can been seen in association with an old injury or as a response to overload, muscular weakness, leg length discrepancy, or other asymmetric anatomic abnormalities. One theory is the "lower crossed syndrome," where tight hip flexors and erector spinae are associated with weak gluteals and abdominals. This can lead to an anterior pelvic tilt and dysfunctional lower extremity biomechanics.

**Treatment:** The most difficult aspect of treatment is the recognition of muscular imbalances. Treatment rests on the flexibility of tight structures, in addition to the strengthening of weak structures. The correction of anatomic or gait abnormalities is essential.

# ENVIRONMENTAL ISSUES

## Heat Injuries

**General considerations:** When an athlete exercises in the heat, as body temperature increases, physiologic mechanisms act to dissipate excess heat into the environment. These thermoregulatory adjustments impart physiologic strain and may lead to dehydration, which in itself can also lead to further thermal and cardiovascular strain.

**Heatstroke:** Body temperature above 104°F, altered mental status, absent sweating, seizures, and coma. Can be fatal, and accounts for hundreds of deaths in the United States annually. Treat as medical emergency; assess renal, neurologic, hepatic systems, and hospitalize.

**Heat exhaustion:** Where body temperature does not exceed 104°F and no mental status changes are observed. Symptoms include profound weakness, dizziness, syncope, muscle cramps, and nausea. Risks are similar to heatstroke if unrecognized. Treatment often similar: assessment by physician, rest in cool environment out of the sun, oral administration of fluids. Activity should be restricted for remainder of day, and athlete should be re-evaluated on the following day.

**Heat cramps:** Cramps often in abdomen and lower extremities; often related to fluid or electrolyte deficiency. Treat with fluid and electrolyte replacement.

**Heat syncope:** A decrease in vasomotor tone and venous pooling (especially immediately after stopping exercise), which leads to syncope; dehydration and increased body temperature contribute to development

**Heat tetany:** Spasm, often in wrist, observed with heat; exacerbated by hyperventilation

**Heat edema:** A swelling of the hands and feet in response to heat; often worst in initial phases of accommodation to new environment. Self-limited; often resolves with cold compresses and elevation. Diuretics not indicated and may exacerbate dehydration

**Risk factors:** Very young or very old age; preexisting dehydration; obesity; a history of previous heat-related illnesses, heart disease, alcohol use, or drugs (tricyclic antidepressants, amphetamines, LSD, PCP, cocaine, anticholinergics [previously discussed], antihistamines, diuretics, beta-blockers); increased humidity, temperature, no clouds or wind; poor physical conditioning; acute febrile illness

**Treatment:** Generally focuses on rapid cooling and timely transport to a medical facility. If rectal temperature is above 104° F, cooling should occur immediately with the goal of reaching a temperature of 102° F within 30 to 60 minutes. Airway protection must be ensured. Electrolyte abnormalities common

**Prevention:**

- Heat acclimatization: athletes planning to compete in heat should train for a minimum of 2 weeks for 60 minutes per day in a similar environment to the competition venue to allow for optimal physiologic adaptations
- Hydration: prior to training or competing in the heat, athletes should consume 6 mL/kg fluid every 2–3 hours. Sodium supplementation may be required during training. Postexercise hydration should aim to replenish 100%–150% of body mass losses.
- Cooling strategies: combination of internal (ingestion of cold fluids) and external (iced garments, cold towels, water immersion, fans)

## Cold Weather Injuries

- Heat production in the body is maintained by:
  - **Basal heat production:** Produced by metabolic processes; ineffective in maintaining body temperature when exposure to cold environment occurs
  - **Muscular thermoregulatory heat:** Produced by shivering. Blood flow is preferentially shunted to vital organs. Can increase heat production three- to five-fold but requires energy; also increases cooling of the distal extremities and skin
  - **Mild-to-moderate exercise-induced heat:** Produced by low-intensity exercise; can increase heat production five-fold. Uses low-energy requirements and can thus be sustained for a long period of time
  - **High-intensity exercise-induced heat:** Can increase heat production 10-fold but cannot be maintained for long periods because of higher energy requirements

### Specific Cold Weather Injuries Include:

**Frostnip:** Reversible without permanent tissue damage; blanching of skin; slow, painless crystal formation on ears, face, toes, and fingertips; common in windy conditions

**Chilblains:** Repeated exposure to cold water or wet cooling; a red, hot, tender swollen extremity with numbness or tingling. Irreversible damage can occur to capillaries, muscles, and nerves innervating the extremity; may progress to gangrene

**Frostbite:** The freezing of soft tissue; commonly involves the nose, fingertips, ears, and toes. Symptoms include local pain, numbness, redness, and superficial blistering. Deeper blistering and the involvement of deep soft tissue and bone occurs with increased severity of exposure. Common in winter months and/or with windy/wet conditions. Frostbite may be permanent if not treated quickly.

**Hypothermia:** Occurs when the core body temperature drops below 95° F; significant cause of mortality

**Treatment of cold injuries:**

- **The best treatment is prevention.**
- After a warm environment is secured, patient should be rewarmed slowly to minimize the risk of tissue damage.
- Removal of all wet clothing; proper measurement of core body temperature using oral, rectal, or auricular thermometer
- Use of warm whirlpool, analgesics, skin protection
- Other preventive measures: layered clothing; covering for head; protection of hands and toes; avoidance of wetness; proper nutrition and hydration; adequate warmup before activity; breathing through nose to warm inspired air; avoidance of alcohol, nicotine, and other drugs. Never train alone

## Guidelines About Environmental Issues

- American College of Sports Medicine Guidelines remain standard.

**TABLE 93.2 COLOR-CODED FLAG SYSTEM**

| Color | Risk | Comments | Restrictions |
|---|---|---|---|
| Black | Extreme risk | WBGT > 82° F | Cancel race |
| Red | High risk | WBGT 73–82° F | Be aware; high-risk athletes should not run |
| Yellow | Moderate risk | WBGT 65–73° F | Heat-sensitive athletes should slow pace |
| Green | Low risk | WBGT 50–65° F | No restrictions |
| White | Risk for hypothermia | WBGT < 50° F | Hypothermia possible, especially if wet or windy |

*WBGT,* Wet bulb globe temperature.

**TABLE 93.3 CORRECTED ESTIMATION OF THE RISK OF EXERTIONAL HEAT ILLNESS BASED ON THE WET BULB GLOBE TEMPERATURE (WBGT) TAKING INTO ACCOUNT THAT WBGT UNDERESTIMATES HEAT STRESS UNDER HIGH HUMIDITY**

| Estimated Risk | WBGT (°C) | Relative Humidity (%) |
|---|---|---|
| Moderate | 24 | 50 |
| Moderate | 20 | 75 |
| Moderate | 18 | 100 |
| High | 28 | 50 |
| High | 26 | 75 |
| High | 24 | 100 |
| Excessive | 33 | 50 |
| Excessive | 29 | 75 |
| Excessive | 28 | 100 |

From Racinais S, Alonso JM, Coutts AJ, et al. Consensus recommendations on training and competing in the heat. *Br J Sports Med*. 2015;49(18):1164-1173.

- Use of wet bulb globe temperature (WBGT) ideal but has limitations
- WBGT = (0.7 Twb) + (0.2 Tg) + (0.1 Tdb); where Twb = wet bulb thermometer temperature, Tg = black globe thermometer temperature, and Tdb = dry bulb thermometer temperature
- Use color-coded flag system (Table 93.2).
- Use correction system to account for humidity (Table 93.3).

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eAppendix 93-1 Upper Extremity Injuries (Shoulder Impingement, Acromioclavicular Injuries, Fractures, Medial Epicondylitis)
- eAppendix 93-2 Knee Injuries (Greater Trochanteric Bursitis, Leg Length Discrepancy, Meniscal Lesions)
- eAppendix 93-3 Lower Leg, Foot, and Ankle Injuries (Ankle Sprains, Chondral and Osteochondral Lesions)
- eAppendix 93-4 Hip and Spine Injuries (Spondylolysis and Spondylolisthesis, Herniated Nucleus Pulposus, Paraspinal Muscular Strains)
- eAppendix Safety and Equipment Issues

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

### eAppendix 93-1 Upper Extremity Injuries (Shoulder Impingement, Acromioclavicular Injuries, Fractures, Medial Epicondylitis)

#### Shoulder Impingement

**Description:** Common in field events: javelin, discus, shot put
**Causes:** Rotator cuff pathology, labral tear, biceps tendinitis, multidirectional instability with muscular compensation, natural adaptations in range of motion, or overuse
**Treatment:** NSAIDs, ice, decrease in training, flexibility, evaluation of biomechanics. If no response is observed, assess the need for further diagnostic studies and/or specialist evaluation.

#### Acromioclavicular Injuries

**Description:** Most commonly occur after a fall on the shoulder with the arm by the side
**Symptoms:** Pain and swelling directly over joint, pain with adduction
**Treatment:** Ice, NSAIDs, relative rest

#### Fractures

**Description:** Usually result from direct falls
**Imaging:** Required for suspected fractures
**Treatment:** Depends on fracture site and type

#### Medial Epicondylitis

**Description:** Seen in throwers
**Evaluation:** Need to exclude fracture, valgus instability, and neurologic involvement
**Treatment:** Biomechanical evaluation, ice, NSAIDs, physical therapy modalities, relative rest. Consider use of an arm brace (reversal of lateral epicondylitis strap)

### eAppendix 93-2 Knee Injuries (Greater Trochanteric Bursitis, Leg Length Discrepancy, Meniscal Lesions)

#### Greater Trochanteric Bursitis

**Description:** Tight lateral structures (iliotibial band syndrome) often compress the bursa and cause increased friction of the iliotibial band over the greater trochanter. Can be any combination of three bursae in the area
**Presentation:** Lateral hip pain; pain with active or passive adduction. No pain with passive hip rotation
**Examination:** Assess the biomechanics of the hip, knee, and foot and ankle. Examine leg length discrepancies and presence of training errors, all of which can lead to increased friction at the greater trochanter.
**Treatment:** NSAIDs, ice massage, stretching of the iliotibial band, external hip rotators, hip flexors, use of phonophoresis, iontophoresis, corticosteroid injection (if not responsive to prior treatments)

#### Leg Length Discrepancy

**Description:** Up to 5 mm difference in leg lengths is normal. Can contribute to iliotibial band friction syndrome, greater trochanteric bursitis, patellofemoral dysfunction, sacroiliac dysfunction, and muscular imbalances
**Examination:** Measurements should be made from the anterosuperior iliac spine to the medial malleolus bilaterally to assess for "true leg length discrepancy" and the umbilicus to the medial malleolus bilaterally to assess for "functional leg length discrepancy." Can also bridge the pelvis to determine if the discrepancy is femoral or tibial. Pelvic obliquity and the reproducibility of measurements can make this method imprecise.
**Radiographic measurements:** Standing postural studies; standing anteroposterior view of the pelvis to include the femoral heads and iliac crests. Then measure from bottom of film to measure discrepancy (more accurate). Does not discern exact location of discrepancy (tibia vs. femur), but this is usually of little consequence in management
**Treatment:** Once a discrepancy has been defined, **heel lift** is incorporated into the shoe to correct. **Usually corrects the condition for approximately 60% to 75% of cases.** If the discrepancy is greater than 1.5 cm, modify the shoe externally.

#### Meniscal Lesions

**Description:** Most common acute mechanism is twisting injury in association with jumping. **Meniscal tears associated with osteoarthritis.** Chronic degenerative meniscal tears occur later in life, usually with little or no associated trauma.
**Symptoms:** Joint line pain, lack of full extension, joint effusion that occurs slowly (vs. immediate effusion in cruciate ligament tears), inability to squat or duck walk
**Examination:** Positive joint line pain, "bounce home" test, hyperflexion, circumduction/scour maneuvers (figure eight movement of foot with knee in hyperflexion), McMurray's maneuver, Apley's maneuver
**Diagnostics:** Magnetic resonance imaging (MRI) is sometimes helpful in determining extent of tear, associated injuries, and likelihood of repair versus meniscectomy
**Treatment:** If not causing mechanical obstruction: nonweight-bearing treatment should be used (weight-bearing if pain free), decrease in activity, NSAIDs, ice, maintenance of quadriceps and hamstring strength. Gradual reintroduction of activity, using pain and swelling, as well as mechanical symptoms (clicking, catching, locking) as guides for further conservative management (as opposed to surgical). Sport specificity and individualization necessary for proper management

### eAppendix 93-3 Lower Leg, Foot, and Ankle Injuries (Ankle Sprains, Chondral and Osteochondral Lesions)

#### Ankle Sprains

**Description:** Common in cross-country runners. Studies suggest that poor proximal neuromuscular control can predispose the athlete to injuries. This can be assessed with single-leg squat balance.
**Mechanism of injury:** Inversion mechanism most common with the involvement of anterior talofibular ligament, calcaneofibular ligament, and posterior talofibular ligament, progressively (see Fig. 65.5)
**Associated injuries:** Watch for associated foot or metatarsal injury. Deltoid ligament less commonly involved. Distal anterior tibiofibular ligament involvement (syndesmotic sprain) portends a more extensive injury with longer recovery time.
**Evaluation:** A "**squeeze" test**, where the examiner squeezes the tibia and fibula together and pain is reproduced, implies a syndesmotic injury ("high ankle sprain"). Radiographs should be obtained based on the Ottawa ankle rules.
**Acute treatment:** Ice, elevation, compression wrap, immobilization (if severe), NSAIDs, weight-bearing as tolerated by pain as long as normal gait biomechanics can be maintained. X-rays, including mortise views, to assess for fracture, generally an avulsion. Consider repeat films 7 to 10 days later if initial films are negative and symptoms persist or worsen in order to avoid missing an occult fracture.
**Chronic treatment:** Rehabilitation is important for an early return of motion and strength, as well as proprioception and functional activity. Bracing or taping often allows early return to sport.

### Chondral and Osteochondral Lesions

**Description:** Occur in conjunction with ankle sprains. Ankle most stable in dorsiflexed position, at risk for injury in plantarflexion. Chondral or osteochondral injuries can occur, most commonly at talar dome, less commonly tibial plafond. Can also develop loose bodies

**Presentation:** Athletes present with pain, swelling, locking with activity. Often discovered when presumed ligamentous injury fails to respond to appropriate treatment

**Imaging:** Plain radiographs often negative initially. Lesions better seen on CT or MRI

**Treatment:** Depends on location and size of lesion or fragments and amount of pain and disability. Arthroscopic debridement, drilling, or removal of loose bodies sometimes necessary

## eAppendix 93-4 Hip and Spine Injuries (Spondylolysis and Spondylolisthesis, Herniated Nucleus Pulposus, Paraspinal Muscular Strains)

### Spondylolysis and Spondylolisthesis

**Description:** Spondylolysis (defect in pars interarticularis) and spondylolisthesis (movement of vertebral bodies in the sagittal plane)

**Presentation:** Acute-onset back pain, usually worse with activity and specifically extension; pain to palpation along spine or often paraspinally; pain with extension aggravated by one-legged extension.

**Diagnostics:** Initial films may be negative. Bilateral oblique films are used to assess the integrity of the pars interarticularis. Flexion and extension views to assess for the presence and severity of spondylolisthesis and stability of the spine. MRI, bone scan with single-photon emission CT, or plain CT with thin cuts through the area of interest can be used if suspicion is high.

**Treatment:** Bracing, if caught before X-ray evidence present; limitation of activity; ice; neutral spine stabilization strengthening with flexion-based exercises.

**Return to play:** Gradual increase in sport-specific activity as tolerated with pain as a guide. Follow-up bone scans not helpful because scans remain positive for several months.

### Herniated Nucleus Pulposus

**Description:** Less common injury in cross-country runners, more common in sprinters

**Presentation:** Central back pain that worsens with flexion; pain over disc space; true nerve root irritation can present with classic radicular pain as a typical presenting symptom, with a typical positive straight leg raise test and signs of neural tension

**Examination:** A careful neurologic examination with thorough manual muscle, deep tendon reflex, and monofilament sensory testing to exclude neurologic compromise

**Diagnostics:** Plain radiographs may show degenerative changes and disc space narrowing. Flexion and extension views assess spondylolysis, spondylolisthesis, or instability. MRI is helpful in assessing soft tissue and the disc itself and can demonstrate central or foraminal stenosis. It is also helpful in ruling out a tumor, multiple sclerosis, or other space-occupying lesions. Electromyography or nerve conduction studies are useful diagnostic tools if radiculopathy is suspected.

**Treatment:** Relative rest, NSAIDs, muscle relaxants, ice, physical therapy modalities. Consider epidural steroid injections, opioids, or oral steroids, depending on individual situation.

**Return to play:** Activity may be allowed if no neurologic compromise, full strength and motion are achieved, and pain is controlled. Every situation must be individualized.

### Paraspinal Muscular Strains

**Description:** Common, especially in hurdlers. Often secondary to acute overuse or injuries related to a strengthening program

**Presentation:** Paraspinal pain

**Examination:** Assess for the presence of other low back pathology.

**Diagnostics:** Additional studies indicated if no response to initial treatment, or guided by history or physical examination

**Treatment:** Initial treatment: ice, NSAIDs, stretching. Heat and ice for symptom reduction. Abdominal, as well as neutral spine strengthening program once acute pain subsides. Flexibility program and trigger point injections may help.

## eAppendix Safety and Equipment Issues

- For all events, maintain proper supervision of surface and runways.
- When multiple events are going on at one time, safety issues are paramount. This is especially true for throwing events, in which risk of injury to spectators (impalement) is significant.

### Track Surface

EQUIPMENT ISSUES

- Tracks are generally oval with the inner length equal to ¼ mile, 440 yards, or 400 m. Knowledge of athlete training surfaces is helpful.
- Cinder, clay, or all-weather synthetic track is often used for the track surface itself.
- Proper maintenance of the surface as well as adequate drainage is important.

SAFETY ISSUES

- The amount of shock absorption and damping characteristics depends on the surface used; these factors may play a role in injury.
- The banking of the track may alter the biomechanics of both inside and outside legs. Banking is increased in smaller tracks. Athletes should alternate directions routinely during training.

### Jumping Events

EQUIPMENT ISSUES

- Ramps for long jump, triple jump, pole vault, and javelin often use rubberized synthetic surfaces.
- Maintenance issues are the same as for track surface.
- Take-off boards must be replaced frequently.
- Landing pits usually contain slightly moist sand, which must be turned and maintained at a minimum of 12 to 18 inches deep.
- Pits extend from 10 feet in front of, and at least 20 feet beyond the take-off board.

SAFETY ISSUES

- Ramps that are not wide enough can cause injury, especially inversion ankle sprains.
- Runways must be kept clear of foreign objects, spectators, and other obstacles.

### Pole Vault (Sprinter With Pole)

EQUIPMENT ISSUES

- Foam rubber padding around the planting box as well as underneath the bar
- Planting box with sawdust or sand inside
- Crossbar is usually fiberglass.
- Poles are usually fiberglass (lighter, more expensive) or metal; up to 18 feet long.

SAFETY ISSUES

- Poles can break during take-off or from contact with vaulting box.
- Landing mats should cover vaulting box on all sides.
- The thickness of the mat should range from 28 to 36 inches.
- Risk of injury to cervical spine or head, and risk of fracture secondary to incorrect fall mechanism. Proper cervical-spine care and backboard usage technique is a must.
- Missing the pole plant into vaulting box can lead to shoulder subluxation, dislocation, and acromioclavicular joint injuries.
- Proper technique and supervision are important.

### Hurdles (Sprint Event With Obstacles)

EQUIPMENT ISSUES

- Hurdle is designed to tip over forward.
- Soft top hurdles minimize contact injuries with hurdle during practice.
- Protection of ankle, heel, and knee with padding minimizes injuries in practice.

SAFETY ISSUES

- Lanes clear of foreign objects, spectators
- Injuries can result from direct contact with hurdles on the inside of trailing leg.

### Throwing Events

- Raise biggest concern about injuries to both athletes and spectators.
- Examples: shot put, discus, hammer, javelin
- Proper technique and supervision essential
- Proper warm-up helps prevent overuse injuries.
- Concern for spectators being struck by discus, javelin, hammer, or shot put

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# ROAD BIKING

# 94

Marc R. Silberman

## GENERAL PRINCIPLES

### Races

#### Road Racing

**Stage races:** Multiday races over consecutive days with daily stage winners and an overall winner based on cumulative time; mass start races where the athletes ride in a peloton.

**Grand tours:** Tour de France, Giro d'Italia, and Vuelta a España; usually include a prologue, flat stages for "sprinters," hilly stages for "climbers," and time trials

**Road races:** Mass start point to point races between 100 km and 298 km in length (Milan–San Remo, the longest professional 1 day race in modern times)

**Circuit races:** Multilap races of 100 km to 140 km, on 5-km to 30-km courses. The World Championship and Olympic road races are circuit races. A kermesse is a common cycling race type in Belgium for amateurs, lasting for 120 to 180 minutes.

**Criteria:** Multilap races of 40–80 km or miles, on 1-km to 1-mile courses containing tight cornered roads. A common cycling race for amateurs in the United States (US), lasting for 60 to 90 minutes. Crashes are common.

**Time trials (TT):** Individual races "against the clock"; riders start at 1- to 2-minute intervals on time trial bikes. Drafting is prohibited. Distances range from 20 km to 50 km. **Triathlons** have TTs in lengths from 20 km (sprint distance) to 180 km (full distance).

#### Track Racing

**Description:** Held on a **velodrome** track (with banking, length 250 m to 333 m); riders use **track bikes** (with no brakes and fixed gears)

**Races: Match sprint, kilometer, pursuit, team pursuit, points race, miss and out, keirin, Madison**

#### Touring

- Self-contained noncompetitive cycling for pleasure, ranging from a day to multiday trips
- Fully loaded or self-supported touring involves cyclists carrying everything they need—clothing, food, cooking equipment, and tents in panniers
- **Cyclosportive (randonnee cyclosportive),** or cyclosportif, is a long-distance, annual, organized, mass-participation cycling event.
- The Italian term **Gran Fondo** is used to name these events in the US.
- The true Italian Gran Fondos are long-distance bicycle races, while in the US, they refer to something in between a race and a tour: a mass participation ride of varying distance on open roads, with some racing for time.
- Both a cultural and a sporting event

### Riders

**Sprinters:** Possess high numbers of fast-twitch muscle fibers for explosive acceleration; can reach speeds of 66.1 ± 3.4 kph (57.1 to 70.6), with peak power output of 1248 ± 122 W (989 to 1443 W). Sprinters take calculated risks in maneuvering through the pack, waiting until the last possible moment to move out of another rider's slipstream and into the wind. Professional sprinters in the last 10 minutes of a race produce 316 ± 43 W, 95 ± 4 rpm and speeds of 50.5 ± 3.3 kph; in the last minute prior to the sprint produce 487 ± 58 W, 102 ± 6 rpm and speeds of 55.4 ± 4 kph; with peak power during the sprint at 17.4 ± 1.7 W/kg

**Climbers:** Lightweight, possessing high levels of aerobic power, a high power-to-weight ratio, a $\dot{V}O_2$ max of 75 to 85 mL/kg/min, and power output measurements of 7.4 W/kg over 5 minutes, 6.5 W/kg over 20 minutes, 6.1 W/kg over 30 min, and 5.7 W/kg over 1 hour

**Lead out riders:** Break the wind for their sprinters until the very last possible moment by sustaining high speed for a kilometer; generally lack the finishing top end speed of sprinters

**Time trialists:** Ride at a steady state for long periods; physically larger cyclists who can push a big gear and produce greater absolute power outputs

**Team leaders:** Riders for stage race overall classification must be able to climb and time trial.

**Domestiques:** Sacrifice themselves for the sprinters and leaders by carrying water, blocking the wind, or even giving a wheel

### Organizations

**USA Cycling (USAC):** National governing body for bicycle racing in the US

**Union Cycliste Internationale (UCI):** World governing body for cycling. Issues licenses, enforces disciplinary rules, manages the classification of races and points ranking systems, and oversees the World Championships.

**International Olympic Committee (IOC):** Organization that oversees the Olympics

**United States Anti-Doping Agency (USADA):** A nongovernmental agency responsible for implementation of the World Anti-Doping Code in the US. The World Anti-Doping Code, which lists drugs and methods that are prohibited in sports, was developed by the World Anti-Doping Agency (WADA).

**WADA:** Independent foundation created through a collective initiative led by the IOC. In November 1999, the WADA was created to promote and coordinate the fight against doping in sports. In 2004, the **World Anti-Doping Code** was implemented by sports organizations prior to the Athens Olympics, standardizing regulations governing antidoping.

**Court of Arbitration for Sport (CAS):** International institution independent of any sports organization to facilitate the settlement of sports-related disputes through arbitration or mediation.

**Mouvement Pour un Cyclisme Credible (MPCC):** A union created on July 24, 2007 by professional road cycling teams to defend the idea of clean cycling according to a strict code of ethics.

### Epidemiology and Injury Statistics

- Traumatic injuries occur in 38% to 48.5% of professionals.
- Overuse injuries occur in 51.5% to 62% of professionals.
- Two-thirds of traumatic injuries involve the upper extremity.
- Two-thirds of overuse injuries involve the lower extremity.
- Touring cyclists on a 500-mile, 8-day ride, sustained 57.2% bicycle contact injuries (32.8% buttock, 9.1% groin, 10% palmar, 5.3% foot) and 42.8% overuse injuries.
- A 4-year study of 51 top level professionals found that 43 athletes sustained 103 injuries (50 traumatic and 53 overuse). eight remained free of injury, 22 (43%) sustained both traumatic and

overuse injuries, 13 (25.5%) sustained only traumatic injuries, and 10 (19.5%) sustained only overuse injuries. Twenty-nine (67.4%) sustained more than one injury.

- A survey of 81 cyclists in a well-established masters cycling club found:
  - 81% had a racing license; average number of racing years 9.5, with an average annual mileage 6,000 miles.
  - 79% were seen in an emergency room, 33% had been admitted to hospital, with 15% to the intensive care unit.
  - 54% had sustained fractures: clavicle, 22 cyclists; upper extremity, 20 cyclists; ribs, 20 cyclists; lower extremity, 11 cyclists; vertebral, 11 cyclists; pelvis, 6 cyclists; skull, 6 cyclists.
  - 45% reported a head injury; 34% reported a concussion, with 9% reporting more than one.
  - 75% reported breaking one or more helmets from a crash.
  - 90% reported having road rash.
  - 37% of crashes involved motor vehicles, 9% were due to road surface hazards, 12% were due to skill errors, and 10% were due to mechanical problems.
  - 17% occurred in a paceline, 12% in racing, often criteriums.

## Equipment and Safety Issues

- Bicycles should be inspected regularly. Tire pressure should be set at the proper amount; lower in wet road conditions.
- Protective gear:
  - **Helmets** manufactured after 1999 must meet the Consumer Product Safety Commission (CPSC) standard by law to be sold in the US. There is no federal law in the US requiring bicycle helmet use. Presently, 22 states, including the District of Columbia, have mandatory helmet laws. Helmets are designed for one crash only. Cyclists should write their name, contact information, and medical information in the helmet for emergencies.
  - Protective clothing: gloves, snug-fitting cycling wear, chamois padding in shorts, and sunglasses

## Biomechanical Principles

### Bicycle Anatomy

ROAD BICYCLE

- **Key frame measurements** are seat tube length, seat tube angle, and top tube length.
- **Key component measurements**
  - **Crank length:** Based on height of rider or inseam length (Table 94.1). Too long may predispose rider to fatigue and knee ailments. "Spinning" is easier with a shorter crank.
  - **Crankset:**
    - Triple (3 chain rings): Large chain ring usually has 52 teeth, medium 39 teeth, and small 30 teeth.
    - Standard (2 chain rings): Large chain ring usually has 53 teeth and small 39 teeth.
    - Compact (2 chain rings): Large chain ring usually has 50 teeth and small 34 or 36 teeth.
  - **Stem length and angle**
  - **Handlebar width** should be close to the width of the shoulders.
  - **Handlebar tilt:** Bars can slip and rotate into downward tilt, which can cause excessive reach, leading to hand, neck, and back symptoms.
  - Gearing: The number of gears on a bike is equal to the number of sprockets on the rear wheel multiplied by the number of chainrings. A double chainring on the front with an 11-speed cassette has 22 speeds. A high or big gear is achieved by riding the larger chainring on the front and a smaller cog on the rear, such as 53 × 11. The smallest chainring combined with the largest cog produces a small gear, used for climbing and spinning. Professionals are efficient in choosing the right gear to maintain a high cadence of 90 rpm or greater while maintaining a high speed for an extended period of time.
- **Key bicycle measurements**
  - Saddle height: center of the bottom bracket to the height of saddle where rider sits
  - Difference between saddle height and handlebar height
  - Saddle tilt
  - Saddle fore-aft
  - Plumb line from nose of the saddle, measure the distance behind bottom bracket
  - Distance from nose of saddle to handlebars

TRACK BICYCLE

- No brakes
- Fixed gear
- Major trauma risk with crashes

TIME TRIAL BICYCLE

- Steeper seat tube angle (78 to 84 degrees), aero bars, aero deep dish wheels or rear disc
- Designed to go fast and straight
- Goal is the reduction of frontal surface area, a flat back, narrow arm position, and elbow flexion of 90 to 110 degrees with the ear directly over elbow. The "Praying Mantis" and "Superman" positions are aero set ups, both banned by the UCI.
- The narrow positioning of the arms does not restrict oxygen consumption and lung function.

**TABLE 94.1 CRANK ARM**

| Height (in) | Crank Length (mm) | Inseam (in) | Crank Length (mm) |
|---|---|---|---|
| <60 | 160 | <29 | 165 |
| 60–64 | 165–167.5 | 29–32 | 170 |
| 65–72 | 170 | 32–34 | 172.5 |
| 72–74 | 172.5 | >34 | 175 |
| 74–76 | 175 | | |
| >76 | 180 | | |

Modified from Burke ER. *High-Tech Cycling,* 2nd ed. Champaign, IL: Human Kinetics, 2003.

### Bike Fit

- A proper fit is essential for rider comfort, safety, injury prevention, and peak performance.
- The goal is the optimization of power and aerobic efficiency while avoiding injury (Table 94.2).
- Fit can be tested using static (at rest) or dynamic (while riding) measurements. Dynamic fit testing may involve video analysis; heart rate, wattage, pedal torque readings; wind tunnel testing.
- Changes should be made gradually.
- There are **three contact areas** where a rider interfaces with the bicycle: shoe–cleat–pedal, pelvis–saddle, and hands–handlebar (Fig. 94.1).

SHOE–CLEAT–PEDAL INTERFACE

- The first metatarsal head lies directly over the pedal axle (see Fig. 94.1).
- Leg length discrepancy: shims can be inserted under the shorter leg, or the cleat may be moved forward (and the foot back). One-third to half of the difference should be corrected.

**TABLE 94.2 OVERUSE INJURIES: CONTRIBUTING BICYCLE POSTURE AND BICYCLE ADJUSTMENTS**

| Ailment | Contributing Position | Bicycle Adjustment |
|---|---|---|
| Posterior neck pain, may extend to head | Too great of a reach, handlebars too low, too stretched out | Ride more upright to shorten reach<br>Raise stem height<br>Shorten stem length<br>Ride with hands on hoods or tops of bars |
| Scapular pain | Too great of a reach, handlebars too low, too stretched out | Ride more upright shorten reach<br>Raise stem height<br>Shorten stem length<br>Ride with hands on hoods or tops of bars |
| Hand neuropathy (cyclist's palsy) | Too much pressure on bars, handlebars too low, saddle too far forward, excessive downward saddle tilt | Increase padding on bars and gloves<br>Avoid prolonged pressure, change hand position often<br>Raise stem height<br>Move saddle back if too far forward<br>If saddle is tilted down, position it level |
| Low back pain | Too stretched out | Ride more upright to shorten reach<br>Raise stem height<br>Shorten stem length |
| Tibialis anterior tendinopathy | Saddle height too high | Lower saddle height |
| Achilles tendinopathy | Saddle height too high (excessive stretch)<br>Saddle height too low (with concomitant dropping of heel to generate more power) | Lower saddle height<br>Raise saddle height |
| Morton's neuroma/foot pain/ numbness | Cleat position<br>Irregular sole<br>Shoes too tight | Usually, move cleat back, but may be forward<br>Check sole for inner wear or cleat bolts pressing inward<br>Wider shoes, loosen Velcro straps/shoe buckle |
| Perineal numbness | Saddle too high<br>Tilt angle excessively up or down | Lower saddle height<br>Adjust angle closer to level with the ground |

From Baker A. Medical problems in road cycling. In: Gregor RJ, Conconi F, eds. *Road Cycling*. Oxford: Blackwell Sciences, 2000, pp 18-45; and Silberman M, Webner D, Collina S, et al. Road bicycle fit: practical management. *Clin J Sport Med*. 2005;15(4):271-276.

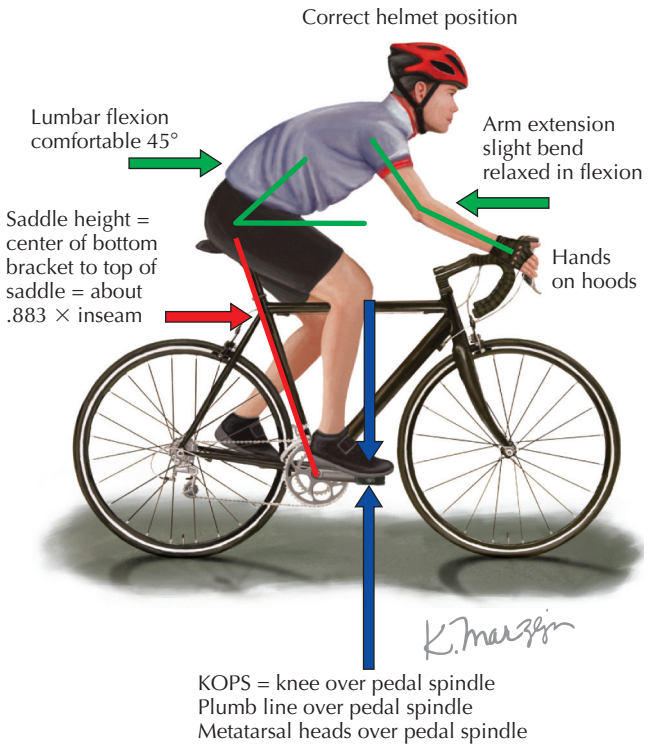


**Figure 94.1.** Road bike fit.

- Heel lifts and orthotics are not sufficient for cycling because the driving force is primarily through the first and second metatarsal heads. Varus forefoot wedges may be used.

PELVIS–SADDLE INTERFACE

- **Saddle height**
  - Traditional existing formulas are designed to fit a rider for the most power at minimal aerobic cost.
  - Greg LeMond and Cyrille Guimard formula: rider's inseam length in centimeters 0.883 × saddle height (see Fig. 94.1).
  - Knee angle method: The knee should be flexed 25 to 30 degrees from full extension, with the pedal in the 6-o'clock position (also known as DBC, dead bottom center) (see Fig. 94.1).
  - **Saddle fore-aft**
    - With the pedal positioned at 3 o'clock (KNOPS, knee over pedal spindle), a plumb line dropped from the inferior pole of the patella should hang directly over the pedal axle (see Fig. 94.1).
    - Time trialists and triathletes prefer a more forward position so that the plumb line falls in front of axle.
    - Moving the saddle forward lowers the saddle height, whereas moving it backward raises the saddle.
    - To compete in a time trial with aerobars, a rider with one bike may move the saddle slightly forward and higher.
    - **Saddle tilt**
      - Saddle tilt should be close to level.
      - About 60% of body weight should be centered on the narrow saddle.
    - Time trialists riding on aerobars may prefer a slight downward tilt of the saddle or a saddle with a split nose to relieve pressure on the perineum.

HANDS–HANDLEBAR INTERFACE

- **Stem and handlebar height**
  - Stem height is a subjective measurement; is important in terms of aerodynamics, power production, comfort, and injury prevention (see Fig. 94.1).
  - With the hands positioned on the brake hoods and the arms slightly flexed, the torso should flex to about 45 degrees in relation to a nonsloping top tube.
  - When the hands are in the drops, the torso should flex to about 60 degrees.
  - The vertical distance, or drop, between the top of the saddle to the bars should be about 5 to 8 cm.
  - A recreational rider may prefer to sit more upright with a shorter reach and higher-raised handlebars for comfort.
  - An average-sized male cyclist may decrease his frontal area by 30 degrees, moving from the upright touring position to a racing position.
  - If forward-flexed excessively, the maximal sustainable power may be reduced because of diminished blood flow and/or changes in muscle lengths.
  - **Handlebar tilt** is a personal preference, but most cyclists prefer the lower curve and brake hoods to be slightly elevated. Bar shape and size also play an integral role in proper fit.
- **Stem length or extension**
  - A rider's reach is determined by the top tube length, stem length, and stem angle or rise (see Fig. 94.1).
  - Too short a top tube or stem length, and the rider will be bunched up. Too long, and the rider will be stretched out.
  - A good starting point is: when the rider looks down with the arms slightly bent and the hands in the drops, the front hub should be obscured by the handlebars.
  - If the frame was properly fitted, the top tube length will allow an optimum position to be achieved with the use of a 10- to 12-cm stem.

## TRAINING AND PHYSIOLOGY

### Performance Testing

**Conconi test:** One of the first tests to determine lactate threshold (LT) without directly measuring lactate; the **pace at which the linear correlation between heart rate and velocity is lost is called the *deflection velocity* or *deflection point*,** and is said to occur at the LT; a ramp protocol of increasing watts is performed in the laboratory with measurement of HR; numerous authors have found the Conconi test invalid.

**Lactate threshold (LT) or anaerobic threshold (AT):** There is no consensus definition. The original *incorrect* hypothesis: the point of exertion where body goes **anaerobic or into "oxygen debt"** and rapidly produces **lactic acid**, causing **fatigue and leg burn.** The onset of blood lactate accumulation **(OBLA)** was originally referred to as the effort level that corresponded to the point at which lactate began to rise exponentially—a blood lactate level of 4 mmol. Most commonly, **LT** refers to the effort (watts) that an athlete can maintain without a rise in lactate. The USOC Sport Science and Technology Division identifies the lactate threshold (LT) as **the point at which a minimum increase of 1.0 mmol/L above baseline values is followed by another increase greater than 1.0 mmol/L.** Maximum lactate steady state **(MLSS)**: the effort level at which there is an equilibrium between lactate production and clearance, such that prolonged exercise does not result in rising serum lactate. Critical lactate measurements are power output at 2 mmol and 4 mmol. Lactate is a useable fuel, and training helps the body become more efficient at shuttling lactate for utilization to other parts of the body.

$\dot{V}O_2$ max test: Ramp protocol consisting of increasing load by 25 watts at 1-minute increments until athlete failure to maintain set cadence. Measure oxygen consumption and heart rate (HR) at different workloads.

**Maximum aerobic power test with lactate:** Ramp protocol with increasing load of 30 to 40 watts at 3- to 4-minute intervals until failure to maintain set cadence. Measure $\dot{V}O_2$, HR, watts, lactate. Maximal aerobic power output (MAP) is defined as the highest power output maintained during the test. Higher power outputs are achieved during shorter ramp protocols of 1-minute stages of 25 watt increments versus 4-minute stages of 35 watt increments. $\dot{V}O_2$ max values of greater than 70 mL/kg/min are found in elite and professional cyclists. Professional cyclists appear to have a decrease in the magnitude of the $\dot{V}O_2$ slow component (oxygen uptake slowly rises during prolonged exercise at submaximal intensity, attributed to recruitment of type II fibers due to fatigue of type I fibers).

**Cycling economy (CE):** Power output generated in watts at a cost of 1 L of oxygen per minute of exercise (Coyle). For a constant load test of 20 minutes at 80% of $\dot{V}O_2$ max, the economy of world class cyclists averages 85 W/L/min.

**Gross mechanical efficiency (GE):** Ratio of work accomplished to energy expended; $GE = 60 \times W \div 20{,}934 \times \dot{V}O_2$ (Jeukendrup). For world class cyclists, the GE is 25%. Both CE and GE are positively related to the percentage distribution of type 1 fibers in the knee extensors. Once a high level of fitness has been obtained, such as in the elite athlete, CE and GE performed at submaximal intensities of 70% to 90% of maximum heart rate are more important determinants of cycling performance than $\dot{V}O_2$ max (Lucia).

**Wingate anaerobic test (WANT):** Developed in Israel during the 1970s and measures **peak anaerobic power** (highest mechanical power generated during any 3- to 5-second interval of the test), **anaerobic fatigue** (the percentage decline in power compared with peak power output), and **total anaerobic capacity** (total amount of work accomplished over 30 seconds); the athlete pedals on a mechanically braked ergometer for 30 seconds all out against a fixed resistance.

### Training Periods of Professional Road Cyclists

- "Rest" (November to December)
  - Weekly average 200 km/week, gentle, easy
  - 90% of the time spent in Zone 1, less than 70% of maximum heart rate (HRmax)
  - 10% of the time spent in Zone 2, 70% to 90% HRmax
  - 0% of the time spent in Zone 3, greater than 90% HRmax
- Precompetition (December to mid-February)
  - Weekly average 700 km/week; building base mileage, long steady rides, no intervals prior to 1,000 miles
  - 80% of the time spent in Zone 1, less than 70% of HRmax
  - 15% of the time spent in Zone 2, 70% to 90% HRmax
  - 5% of the time spent in Zone 3, greater than 90% HRmax
- Competition (mid-February to October)
  - Weekly average 800 km/week
  - 75% of the time spent in Zone 1, less than 70% of HRmax
  - 15% of the time spent in Zone 2, 70% to 90% HRmax
  - 10% of the time spent in Zone 3, greater than 90% HRmax
- Most riders plan for two peaks during the season.
- Training prescription is traditionally not as precise as in other endurance sports; most build long steady low-intensity mileage in winter months; attend one or two 8 day training camps with higher intensity and mileage in the late winter; "race into shape" with early season races. Traditional programs are followed to prepare riders for grand tours, with generally 30 days of racing prior to Tour de France.
- With power measurements on the road, cyclists can follow stricter training programs.

## Training Measurements

**Perceived level of exertion:** Simply monitoring how one feels. Borg 10- or 20-point scale

**Time:** Measure ride duration only, not intensity. Hours per ride for recreational rider: 1 to 1.5 hours. Amateur: 2 to 3 hours per ride, 8 to 12 hours per week. Professional: 15 to 30 hours per week.

**Speed:** Training based on average speed. Generally a poor indicator of training intensity because of the effects of altitude, wind, terrain, road surface, and drafting. Using average speed as indicator of intensity will lead to overtraining.

**Distance:** Training based on weekly or daily mileage. Does not measure intensity

**Heart rate:** Closely correlates with exercise intensity, power output, or rate of oxygen consumption in the laboratory, correlation not as close out on the roads. Influenced by altitude, heat, hydration, illness, sleep, overreaching, and overtraining. Heart rate responds relatively slowly to changes in exercise intensity and cannot be used to regulate the intensity of shorter efforts **(heart rate lag).** Heart rate is not a direct determinant of performance but is a reflection of the strain imposed on the cardiovascular system for the level of exertion.

**Power output:** Provides a direct and immediate answer to exercise intensity. Measured on the road with bike-mounted power meter systems. Power at LT, or 4 mmol of lactate, is one of the most important physiologic determinants of cycling performance. Functional threshold power equals average power during a 40-km (50- to 70-minute) time trial. Correlates very highly, slightly greater than, power at LT (defined as 1 mmol/L increase in blood lactate over exercise baseline). Estimates athlete's threshold power by measuring power athlete can routinely produce in training during long interval repeats of 2 × 20 minutes. Mean power for five mass start stages: 220 ± 22 W (range 190 W to 310 W), average HR: 142 ± 5 beats per minute (bpm). Mean power for uphill 13 km TT: 392 ± 60 W (5.5 ± 0.4 W/kg), average HR: 169 ± 3 bpm. Indirect measurement for 3-week stage races: 246 ± 44 W for high mountain stages, 234 ± 43 W for semimountainous stages, 192 ± 45 W for flat stages (Table 94.3)

## Technique

### Pedaling

- The driving force of forward motion is the downward push on the pedals.
- The muscles involved in the power phase drive the crank downward in an effort to rotate the crank, whereas the muscles active in the recovery phase are firing primarily to reduce resistance versus the contralateral propulsive limb.
- Most athletes who are clipped into pedal systems believe they are supposed to pull up on the pedals; **pulling up on the pedals is inefficient and may lead to overuse injury.**
- In elite cyclists, even on the upstroke, the vector of forces is downward in the opposite direction of the pedal motion (the leg in the recovery phase is not lifted as fast as the crank is rotating). The elite cyclist exhibits reduced negative force and time during the upstroke.
- **Maximal torque during the down stroke is what differentiates elite athletes from the recreational rider.**

### Cadence (Revolutions Per Minute, or RPM)

- To date, no optimal cadence has been determined. Experienced cyclists will spontaneously pick the cadence that appears to work best for them. The freely chosen cadence of elite cyclists is 90 rpm for group stages and time trials, 70 to 100 rpm for mountain ascents.
- Although low cadences (50 to 60 rpm) have been found to be more economical/efficient (lower $\dot{V}O_2$), most cyclists prefer to pedal at high cadences. Improved blood flow and reduced muscular stress are possible advantages at higher cadences.
- Blood flow oxygenation to the vastus lateralis is significantly reduced during the first third of the crank cycle, with a compensatory transient increase in blood supply during the relaxation phase of the upstroke.
- For a given power output, at faster cadences, the force on the pedals and of muscle contractions is reduced.
- Faster cadences may be beneficial because less intramuscular pressure causes less blood vessel constriction.

# COMMON INJURIES AND MEDICAL PROBLEMS

## General Overview

- Injuries can be classified into bicycle contact, traumatic, and overuse categories.
- There are few scientific studies regarding injuries.
- Two-thirds of traumatic injuries occur in the upper extremity, with the most common site being the shoulder.
- Two-thirds of overuse injuries occur in the lower extremity, with the most common site being the knee.
- Cycling is a nonimpact activity, *except when crashing*; overuse injury in cycling rarely prevents an athlete from continuing to ride. Stress fractures do not occur in cycling.
- The primary muscle action is quadriceps concentric activity; acute muscle tears rarely occur.
- Overtraining or staleness is common.

## Contact Area Injuries

There are three areas at which a rider makes contact with the bicycle, and each of these areas lends itself to specific injuries.

### Shoe–Pedal Interface

- Burning feet, numbness, or pain is common. Riders may unclip their cleats to shake their feet to relieve symptoms.
- Plantar radiculopathy, or a **Morton's neuroma,** may result from the impingement of interdigital nerves, classically between the third and fourth metatarsal heads, from tight, rigid cycling shoes and pressure on the pedals.
- Bike fit remedies: adjust cleat, usually by moving farther back; wearing a shoe with a wider toe box; loosening straps on cleats; inspecting the shoe for compression points; using a wider platform pedal or a small metatarsal pad
- Treatment: Massage, manual therapy, nonsteroidal anti-inflammatory drugs (NSAIDs), shoe inserts, injection therapies such as cortisone, nerve block, prolotherapy, platelet rich plasma (PRP), or sclerotherapy injections, surgical excision considered as last resort if a mass is present.

### Pelvis–Saddle Interface

#### PERINEAL VASCULOPATHY/NEUROPATHY

- The most common urogenital problem encountered is genital or perineal numbness; reported in as high as 91% in a study of 17 cycling policemen. Symptoms transient for most individuals, but the long-term risks are unknown
- Ischemic neuropathy may result from the compression of the neurovasculature bundle in the perineum or in Alcock's canal.
- Compression, friction, and/or stretching hypothesized
- "Cyclists syndrome": pudendal nerve entrapment; results in pain, burning, numbness, and sometimes sexual dysfunction, impotence, and urinary incontinence
- Bicycle factors: excessive saddle tilt upward, saddle height too high, handlebars too low or too far forward; too much or too

**TABLE 94.3 POWER-BASED TRAINING LEVELS**

| Level | Name | % of Threshold Power | % of Threshold HR | Perceived Exertion |
|---|---|---|---|---|
| 1 | Active recovery | <56 | <69 | <2 |
| 2 | Endurance | 56–75 | 69–83 | 2–3 |
| 3 | Tempo | 76–90 | 84–94 | 3–4 |
| 4 | LT | 91–105 | 95–105* | 4–5 |
| 5 | $\dot{V}O_2$ max | 106–120 | >106† | 6–7 |
| 6 | Anaerobic capacity | >120 | N/A | >7 |
| 7 | Neuromuscular power | N/A | N/A | Maximal |

***Sample Workouts for the Athlete Who Time Trials at an Average Power of 300 W and HR 160***

| Level | Avg. Wattage | Avg. HR | Workout |
|---|---|---|---|
| 1 | <166 | <111 | 1-hour ride |
| 2 | 166–225 | 111–134 | 3-hour ride |
| 3 | 226–270 | 135–152 | Warmup 30 min at L1-2, then 1.5 hour at L3, 30 min cooldown |
| 4 | 271–315 | 153–170* | Warmup 30 min as if for race, perform 2 × 20 minutes at level 4 with 5 minutes at level 1 between efforts, warmdown |
| 5 | 316–360 | >171† | Warm-up as above, then 6 × 5 min at L5, with 5 min at L1 between efforts, warmdown |
| 6 | >360 | N/A | Warmup as above, then 10 × 1 min at L6 with 3 min at L1 between, warmdown |
| 7 | N/A | N/A | Warm-up thoroughly, then do 6–10 all out 10 s sprints with complete recovery between efforts |

| Level | Description of Level |
|---|---|
| 1 | Easy spinning, light pedal pressure, minimal sensation of leg effort, active recovery after hard training or races |
| 2 | "All day" pace or classic long slow distance training, sensation of leg effort low but may rise when climbing, frequent days in a row possible but complete recovery may take more than 24 hours for very long rides |
| 3 | "Spirited group ride" or brisk paceline, requires concentration to maintain alone, consecutive days still possible if duration is not excessive and carbohydrate intake is adequate |
| 4 | Just below to just above TT effort, mentally very taxing, usually done in multiple repeats of 10-30 min duration, consecutive days possible if completely recovered from prior training |
| 5 | Intensity of long 3- to 8-min intervals designed to increase $\dot{V}O_2$ max, completion of 30-40 min total training difficult, consecutive days not necessary |
| 6 | Short 30 s to 3 min intervals designed to increase anaerobic capacity, HR not useful due to nonsteady state nature of effort, consecutive days not necessary |
| 7 | Very short, high-intensity efforts (jumps, standing starts, sprints) stressing musculoskeletal system versus metabolic systems, power useful as guide to compare versus prior efforts |

*May not be achieved during initial phases of effort.
†May not be achieved due to slowness of the heart rate response and/or ceiling imposed by maximum heart rate.
Data from Coggan AR. *Training and Racing with a Power Meter.* Boulder, CO: Velopress, 2006.

little saddle padding, narrow saddle, prolonged seated riding in one position; riding on rollers is worse than a stationary bicycle, which is worse than riding on the road.

- Effectiveness of ergonomic wide saddles, with a split nose, chopped nose, and central cut out designed to decrease compression have not been scientifically proven.
- Treatment: Relative rest, physical therapy, adjustment of bike fit or riding technique, change of saddle type, nerve blocks, cortisone injection, botox injection, surgical decompression for severe recalcitrant cases

ERECTILE DYSFUNCTION (ED)

- There is anecdotal evidence of cycling-related erectile dysfunction (ED).
- Reported to affect 13% to 24% of male riders. True incidence unknown
- Vascular and neurologic mechanisms are implicated.
- Compression of the perineal region during cycling may cause decreased penile perfusion.
- Prolonged hypoxemia has been associated with penile fibrosis, which is known to cause ED.

- Blunt trauma to the corpora cavernosa has been considered to be a risk factor for the subsequent development of ED.
- Penile blood flow decreases significantly while cycling in a seated position; as high as an 82% drop in transcutaneous penile oxygen pressure has been recorded.
- The elimination of the nose of the saddle results in a reduction in perineal pressure.
- An important factor in safeguarding penile perfusion is a saddle wide enough to support the ischial tuberosities without compression of the perineal region—*not* saddle padding.
- Scientific studies have been limited; most conducted in laboratory settings on stationary bicycles, without attention to technique and factors involved in road cycling.
- Recumbent cycling causes no compression of perineum.
- Perfusion is increased with standing; frequent position changes are beneficial.
- The time trial position in the aero bars increases compression of the perineum; may be reduced with adjustment of saddle tilt and use of a split nose saddle.
- Millions of cyclists are asymptomatic. Some appear at more risk than others. Obesity is a significant risk factor. Bike fit and technique play a role in prevention.

#### SADDLE SORES

- Moisture, friction, and pressure lead to skin ailments in the genital region.
- **Chafing** is its most common mild form. Relieved with 1 to 2 days off the bike.
- **Ulceration** or more severe friction injury may require local wound care.
- **Furuncles and folliculitis** (saddle sores). Very painful and may limit riding for long periods
- **Perineal nodular induration (third testicle)** is the most severe form. Also called *ischiatic hygroma*, *accessory testicles*, and *biker's nodule.*
- Friction and pressure from the saddle induces collagen degeneration, myxoid changes, and pseudocyst formation.
- Histologically: presentation of a nodule with a dense fibrous capsule surrounding a central pseudocyst
- Clinically: elastic 2- to 3-cm perineal nodules fixed to the underlying soft tissue
- Time off from riding and cortisone injections are nonsurgical options. Surgical excision is definitive.
- Bike fit remedies: change or cut a hole in the saddle; check saddle height and tilt, raise handlebar height or shorten reach.
- Medical treatment: prevention, wear a dry, clean chamois; emollients; avoid shaving the area; warm soaks; topical cortisone, antifungal, antibacterials; oral antibiotics; surgical incision and drainage; excision

#### PROSTATE DISORDERS

- No evidence exists linking cycling to prostatitis (PSA)
- A systemic review and meta-analysis in 2015 suggests that there is no effect of cycling on PSA; however, there are only a few, small studies, and none are randomized controlled.
- Hormonal effects from strenuous exercise and local mechanical stress may increase PSA.
- Avoiding cycling prior to PSA sampling may be advised but is without any scientific basis.

#### FEMALE ISSUES

**Description:** The anatomic course of the pudendal artery and nerve is the same as in males; sexual and genitourinary complaints are similar.

**Saddle discomfort:** Women may find the saddle uncomfortable and experience sexual and urinary dysfunction. Study on 282 females found that 33% had perineal trauma, 19% associated with hematuria or dysuria, and 34% with numbness. Bike fit: women's specific frame with shorter top tube for decreased reach, women's saddles, shorts with padded chamois, saddle tilt down slightly

**Vulvar swelling:** Mostly unilateral and caused by prolonged riding; no long-term sequelae

**Urethritis:** May be caused from pressure, friction, or infection. Rule out genitourinary infection with a pelvic exam for vaginitis (yeast due to moisture), and urinalysis and culture. Hematuria and dysuria may be caused by local trauma alone. Treatment: improved bike fit and routine management

### Hands–Handlebar Interface

- **"Cyclist's palsy":** Prolonged compression of ulnar nerve in Guyon's canal. Median nerve may also be compressed. Complaints of numbness, pain, and weakness; resolves with rest. Damage is rarely permanent.
- The distal motor latencies of the deep branch of the ulnar nerve to the first dorsal interosseous were significantly prolonged in a study of 28 riders in a 420-mile tour.
- Twenty-three out of 25 cyclists participating in a 600-km ride experienced compressive palmar symptoms, the majority in the ulnar distribution.
- Thirty-two of 89 touring cyclists on an 80-day tour of 4,500 miles experienced hand numbness, 10 in the median nerve, with one unable to adduct his thumb, one unable to adduct his little finger, and one unable to abduct his little finger.
- Mononueropathy of the deep palmar branch of the ulnar nerve in Guyon's canal may cause clawing. In a Type 1 lesion (mixed sensory and motor), compression is proximal (outside of Guyon's canal) and clawing is not usual. In isolated lesions of the deep terminal motor branch with the distal sensory branches intact, the athlete is unaware of compression until severe motor lesion develops.
- Cycling factors: infrequent change of hand position, downhill cycling, stationary riding, prolonged saddle time, rough terrain, handlebars too low or forward, poor padding in gloves or bars
- Bike fit remedies: frequent change of hand position, reduction of training volume, change ride terrain, increase padding, shorten reach or raise bars, introduction of aero bars. Gel padding may worsen problem.
- Medical treatment: rest, massage, injection therapies, night splint, hand therapy; surgery in severe cases

## Acute Traumatic Crash Injuries

### Skin Abrasion ("Road Rash")

- Most common traumatic injury
- Treatment: Rapid cleansing with scrubbing to prevent infection and staining ("tattooing"). Soap and water is best. Peroxide can inhibit healing. Use local anesthetic.
- The wound should not be left open to the air to heal because large scabs may form.
- Traditional method: Cover wound with a nonadherent dressing with antibiotic ointment or silver sulfadiazine, pad with gauze, wrap with stretch gauze, and cover with tube stretch gauze. Daily dressing changes with cleansing of exudates, until pink healthy tissue emerges.
- Alternative: Semipermeable films, hydrocolloid dressings, or bioclusive bandages may be used and left in place for extended period of time until healing.

### Handlebar Trauma

- More common in children with straight handlebars
- Caused by a direct blow of the bars turning sideways into the abdomen
- Injuries: **splenic, liver, pancreatic, renal, bowel,** and **urethral**
- Presentation is often delayed.

- Most cases of organ contusion are handled with observation and serial computed tomography (CT); occasionally surgery
- **Handlebar hernia** is a rare traumatic hernia involving disruption of the abdominal wall muscles with bowel loop herniation and potential volvulus. Treatment is primary repair.
- **Lacerations** result from the sharp metallic ends of the handlebar cutting through soft rubber handles, or bars with no end plug.

### Concussion

- From direct blow, fall over front of handlebars; most get up and ride concussed.
- May be overlooked due to concomitant injuries
- If helmet has any surface scratch, look for interior damage.
- Discard helmet after one blow.
- Sample return to competition protocol:
  1. Physical and cognitive rest until symptom free; traditionally recommended but now questioned, may start with stage 2 at a level that won't worsen symptoms
  2. Stationary cycling on a trainer for 30 to 45 minutes at HR < 70% of max
  3. Nongroup road riding of low intensity and duration once symptom free
  4. Nongroup road riding with bigger gears, hills, and/or intervals
  5. Group riding

### Upper Extremity Injuries

#### CLAVICLE FRACTURES

- The most common cycling fracture, followed by the wrist, ribs, and elbow
- Caused by a direct blow to the shoulder
- Of clavicle fractures, 72% to 80% occur in the middle third of the clavicle, 25% to 30% in distal clavicle, and about 2% at the proximal clavicle.
- May be associated with **rib/scapula fractures and concussion.** Also rarely associated with **pulmonary contusion, pneumothorax, hemothorax,** and **neurovascular injury.**
- Most cyclists can ride on a trainer within 3 days to 1 week, and outdoors within 2 weeks; resume racing in 4 to 6 weeks.
- Historically, clavicle fractures have been considered to be best treated nonoperatively with a simple sling.
- A prospective randomized trial found that a simple sling caused less discomfort and fewer complications than a figure-eight brace.
- Slings are commonly used for 1 to 2 weeks until pain free, with early range of motion as pain allows.
- Current management of medial clavicle fractures remains nonoperative because significant displacement is rare due to ligament stability. If displacement is present or suspect sternoclavicular injury, image with CT.
- The ideal treatment of distal clavicle fractures remains controversial because of the existence of multiple subtypes of associated ligamentous injury; most cases treated nonoperatively.
- Treatment of middle third fractures is nonoperative, except when severe displacement, communition, or shortening is present.
  - Initial shortening of 20 mm or more significantly associated with nonunion and an unsatisfactory result
  - Displacement of more than one bone width on an X-ray is a risk factor for nonunion
- Late repair of painful nonunion displaced midshaft fractures has results similar to immediate fixation.
- Surgery: Intramedullary fixation, with risk of wire migration, or plate and screws with or without bone grafting, with risk of prominent painful hardware
- Asymptomatic nonunion is not associated with significant disability.

#### OTHER UPPER EXTREMITY INJURIES

**Acromioclavicular separation:** Direct blow from fall on adducted arm. Requires brief use of sling for comfort. Stationary trainer workouts can begin within days, road riding in 2 to 3 weeks. Treatment is same as for noncyclists; Grade I and II always nonsurgical. Even Grade III, grossly visible in thin cyclists, is rarely surgical. For Grade IV, V, and VI, internal fixation with a hook plate, sometimes temporary, may be considered.

**Shoulder dislocation:** Usually involves transient dislocation and relocation with bone bruising, **tear of ligaments and/or labrum, Bankart or Hill-Sachs lesions.** Cyclists can generally return quickly; instability in everyday activity is a considering factor for operative repair later.

**Radial head fractures:** Type I presents with hemarthrosis, decreased extension, and fat pad sign on X-ray. A short-term sling is employed with rapid commencement of range of motion exercises to achieve full extension. Aspiration of hemarthrosis with infiltration of anesthesia may check for mechanical block and aid in range of motion and pain relief. With nondisplaced fractures, the athlete can return to riding within 1 to 2 weeks.

**Distal radius, scaphoid, or hook of hamate fracture:** Falling on an outstretched hand. MRI should be conducted if there is any suspicion of scaphoid fracture. In nonsurgical cases, a rapid return to training within 1 to 2 weeks is possible with casting.

### Lower Extremity Injuries

**Hip pointer and/or greater trochanteric bursitis:** Common from a direct blow to the lateral aspect of pelvis/femur; large **hematoma** may result; have a high clinical suspicion for pelvic fracture—most are stable and treated with crutches and time; MRI and/or CT when in doubt; most are able to ride on a trainer before walking normally. Aspirations of greater than 60 cc of blood are common with hematomas.

**Knee contusion:** If there is a clinical suspicion for a patella fracture, take sunrise or merchant X-ray. MSK ultrasound is reliable in diagnosing cortical fracture. **Prepatellar bursitis** is common and can be treated with aspiration. Caution for infection. Wrapping the patella should not be done when cycling because restriction of patella movement may lead to patella–femoral pain.

**Fractures of the femur and hip** are less common; usually seen in high speed collisions, simple falls at slow speed, and in those with osteoporosis. Common if motor vehicles are involved in the incident.

## Overuse

### Knee Injuries

- The knee is the most common site of overuse injury in the cyclist.
- Simple bicycle adjustments may be made based on the location of knee pain (Table 94.4).

#### ANTERIOR KNEE PAIN

- Patella tendon strain ("tendonitis"):
  - Anterior knee pain in the tendon
  - May become chronic (tendinosis). MSK ultrasound is excellent for imaging the patella tendon.
  - Causes: Pushing big gears; cranks too long; steep, long hills, excessive head wind, rapidly increasing mileage or intensity; saddle too low or forward; acute forceful motion
  - Medical treatment: Physical therapy, eccentric one-legged squats, manual therapy, massage, assisted stretching, strapping; prolotherapy, PRP, tenotomy, or sclerosis of neovessels for tendonosis/partial tears; use of surgery is rare
  - Cycling treatment: Decrease mileage and intensity, avoid hills, and spin 80 to 90 rpm. Check saddle height and fore-aft position.

**TABLE 94.4 BICYCLE ADJUSTMENT BASED ON THE LOCATION OF KNEE PAIN**

| Location | Causes | Bicycle Adjustment |
|---|---|---|
| Anterior | Seat too low<br>Seat too far forward<br>Climbing too much<br>Big gears, low rpm<br>Cranks too long | Raise seat<br>Move seat back<br>Reduce climbing<br>Spin more<br>Shorten cranks |
| Medial | Cleats: toes point out<br>Floating pedals<br>Exiting clipless pedals<br>Feet too far apart | Modify cleat position: toe in, consider floating pedals<br>Limit float to 5 degrees<br>Lower tension<br>Modify cleat position: move closer<br>Shorten bottom bracket axle; use cranks with less offset |
| Lateral | Cleats: toes point in<br>Floating pedals<br>Feet too close | Modify cleat position: toe out; consider floating pedals<br>Limit float to 5 degrees<br>Modify cleat position: apart; longer bottom bracket axle; use cranks with more offset; shim pedal on crank 2 mm |
| Posterior | Saddle too high<br>Saddle too far back<br>Floating pedals | Lower saddle<br>Move saddle forward<br>Limit float to 5 degrees |

From Baker A. *Bicycling Medicine: Cycling Nutrition, Physiology, and Injury Prevention and Treatment for Riders of All Levels.* New York: Fireside, Simon and Schuster, 1998.

- **Patella femoral pain syndrome:** Presents as pain in the patella–femoral groove or patella facets. Similar to "tendonitis" in etiology and cycling treatment. Limit the use of cortisone injections.
- **Prepatellar bursitis:** Swelling overlying the patella. Caused by direct trauma or repetitive overuse. Treatment as above; occasional aspiration, injection, and/or culture
- **Pes anserine bursitis/enthesopathy:** Swelling and/or pain at the pes anserine insertion. Use cortisone, prolotherapy, or PRP for recalcitrant cases, otherwise treat as above.

LATERAL KNEE PAIN

- **Iliotibial band (ITB) syndrome:** Lateral knee pain with or without snapping; one of the most common complaints. Ultrasound may show anechoic echotexture beneath ITB along femoral condyle.
- Causes: High mileage and intensity, big gears, hills, prolonged steady state riding, windy conditions, toes pointing inward, or a narrow bottom bracket
- Cycling treatment: Adjust cleats; check bike fit; change pedaling technique.
- Medical treatment: Massage and assisted stretching form an effective first-line treatment. Physical therapy, foam rolling. Osteopathic manipulation of sacrum and pelvis. Leg length evaluation and correction. Orthotics. Use of cortisone, prolotherapy, and/or PRP a last resort.

MEDIAL KNEE PAIN

- **Medial collateral ligament (MCL) bursitis:** Often overlooked; responds well to relative rest, bike fit adjustment, massage therapy, and injection therapies
- **Medial meniscus tear:** Not known to be primarily caused by pedal systems; if tear is present, take caution when unclipping. Often seen on MRI, but not often the cause of the cyclist's pain
- **Medial plica** syndrome may cause anteromedial knee pain, as well as snapping or clicking.
- Repeated irritation from cycling may lead to fibrosis with decreased elasticity and condylar cartilage damage.
- Presence of a plica alone on the MRI is not diagnostic.
- Symptoms similar to meniscal tear
- If treatment with therapy, rest and/or cortisone fails, arthroscopic resection can be used as it has a quick recovery.

POSTERIOR KNEE PAIN

- Not as common; hamstring tendonopathy, bakers cyst, or ganglion cyst.
- Contributing factors: saddle too high or far back, pedals with excessive float, improper technique of pulling up on the pedals, tight hamstrings.
- Treatment: Proper bike fit, relative rest, physical therapy, massage, injection therapies and/or aspirations

## Hip Injuries

FLOW LIMITATIONS IN ILIAC ARTERIES

- Prevalence as high as 20% in top cyclists; underreported and unrecognized problem; have a high clinical suspicion
- Presents as a sensation of dead leg, powerlessness, pain in thigh; disappears upon rest. Worsens with increased hip flexion while riding, time trialing or climbing (steady state riding)
- Physical exam is usually normal, may hear bruit in the inguinal region at rest or postexercise.
- Flow limitation may be caused by kinking (functional iliac artery obstruction) or endofibrosis (external iliac artery endofibrosis, EIAE).
- Mechanical, anatomic, and hereditary factors
  - Anatomically, the external iliac artery (EIA) may be anchored to the psoas muscle and lengthened with multiple collateral vessels.
  - Mechanically, the middle part of the EIA kinks during flexion–extension of the hip, with stress lesions in the vessel wall from shearing forces created by constant cycling.
  - Hereditary and metabolic factors are implicated because 75% of surgical cases also presented with increases in homocystinemia and homocystinuria after a load test with methionine.
  - Histopathology: A nonatheromatous vascular lesion with intimal subendothelial fibrosis, leading to wall thickening and reduction of lumen caliber. May be caused by mechanical loading in susceptible individuals
  - Functional kinking may lead to intravascular damage and poor perfusion.
  - Chevalier treated 334 lesions of endofibrosis surgically from 1991 to 2003. The first method of surgery, deemed conservative, involved endofibrosectomy with the shortening of excessive length and closing with a venous or arterial closing angioplasty. The second method involves saphenous bypass surgery. Mean return to competition timeframe is 3 months.
  - Schep studied 80 suspected cases (92 symptomatic legs) and demonstrated a flow limitation in 58 legs (63%). In 40 of the legs (69%), the primary cause was kinking of the EIA due to a psoas muscle side branch or fibrous fixation of the iliac bifurcation. 23 legs (40%) underwent surgical release, with 87% able to return to high level of competition.
  - Testing
    - The provocative cycling test ankle–brachial index (ABI) is the most important clinical test, and has a specificity and sensitivity in detecting moderate lesions of 90% and 87% respectively. 20 watt per minute ramp protocol, ABI measured immediately postexercise while supine with

90-degree hip and knee flexion. The readings of the ankle pressures must be corrected for the height difference of the ankle from the arm (1 cm = 0.76 mmHg). Positive test: ABI less than .54, ankle difference greater than 23, ankle pressure less than 107; suspicious if ABI is .54–.70.
  - Echo Doppler at rest and immediately post provocative exercise cycling test
  - Magnetic resonance angiography with hip flexed
  - Arteriography is the gold standard.
  - CT angiography and MR angiography do not appear as sensitive.
- Treatment: Surgical, one or more of the following: 1) release of artery (mobilization) for treatment of primary kinking when there is no structural narrowing or lengthening, 2) treatment of excessive length with shortening, 3) treatment of endofibrosis with endofibrosectomy with venous or arterial closing angioplasty, polyester patch angioplasty, or saphenous bypass surgery, 4) inguinal ligament release. Angioplasty and stenting are NOT recommended.
- Medical treatment: Vitamin B1, B6, and folates when minor hyperhomocysteinemia is detected. Less hip flexion on bike. Retirement (unknown natural history, both untreated and treated)

GREATER TROCHANTERIC BURSITIS/GLUTEUS MEDIUS TENDONOPATHY, "PROXIMAL ITBS"
- **Proximal lateral thigh pain with or without snapping**
- **Distal hip external rotator tendon involvement not uncommon**
- Treatment: Relative rest, easy riding, physical therapy, stretching, massage, osteopathic manipulation, injection therapies
- Iliopsoas bursitis rare compared to running; treatment as above for greater trochanteric bursitis

LABRAL TEARS AND OSTEOARTHRITIS
- High incidence of asymptomatic labral pathology found on MRI. May not be underlying cause of the athlete's pain. May be found incidentally after a crash. If associated osteoarthritis is present, then pain is more likely from arthritis. First-line treatment involves physical therapy and/or injection therapies. Risks and benefits of surgery for labral tears should be discussed.
- Femoroacetabular impingement may also occur (see Chapter 54: Pelvis, Hip, and Thigh Injuries).

### Ankle Strains
- **Including Achilles, tibialis anterior, posterior tibialis**
- Rare; usually the result of a cyclist running, an improper bike fit, or poor pedaling technique (excessive pushing down of heel in downstroke, or pulling with foot in upstroke), leg length inequality
- Treatment: Physical therapy, massage, relative rest, cycling technique modification, bike fit adjustment, injection therapies

### Spine Injuries
- **Cervical strain with or without radiculopathy**
  - More common in older athletes with underlying spondylosis or disc disease
  - Worsens with long rides, rough terrain, and handlebars too far forward or too low with hyperextension of the neck
  - Cycling treatment: assume more upright cycling posture. Medical treatment: same as for noncyclists
- **Thoracic strain,** may be associated with **scapula dysfunction and muscle spasm at the levator scapula,** worse with aero bars
  - Treatment: Trigger point injections, manipulation, massage, physical therapy, bike fit adjustment, and strengthening exercises
  - **Coronary artery disease** may present as scapula or upper back pain.
- **Lumbar spine and low back pain**
  - May be caused by strain, spasm, spondylosis, or disc disease
  - Imaging should be performed more quickly for patients with risk factors of other medical conditions.
  - Cycling treatment: bike fit adjustment, avoiding hills and big gears, and shorten rides
  - Medical treatment: same as for noncyclists

### Ischial Tuberosity Pain
- Discomfort in the "sits bones" where the rider makes contact with the saddle
- Area can be tight and sore with or without bursitis.
- Risks: pushing big gears and time trialing, especially early in the season and/or on colder rides
- Treatment: Relative rest, massage, extra padding with new chamois, a new saddle. Injection therapy, cortisone, prolotherapy, or PRP for severe cases

## MEDICAL CONDITIONS
### Osteoporosis
- Masters cyclists with a long history of training exclusively in cycling had low bone mass density (BMD) compared to age-matched peers.
- Cyclists were seven times more likely to have osteopenia of the spine than runners, controlling for age, body weight, and bone-loading history.
- Several factors may play a role:
  - Bicycling is a nonimpact sport; cyclists avoid weight bearing when they are not training.
  - Calcium lost in sweat may play a role. Most cyclists live on a low-fat, high-carbohydrate diet with little extra calcium in the foods they eat.
  - Overtraining can lead to a sex hormone imbalance, increasing bone turnover and decreasing resorption.
- Performance-enhancing substances can accelerate bone loss.
- Unlike in running, where stress fractures are common, cyclists with osteoporosis are at risk of major fractures from crashes.

### Asthma
- 40% to 80% of Tour de France riders reported to "have a diagnosis" or use inhalers during the race.
- The diagnosis of asthma requires supporting medical history with recurrent symptoms of bronchial obstruction (chest tightness, wheeze, or cough provoked by hyperventilation, exercise, or other stimuli), physical examination, and lab or field testing, especially if initially recommended treatment fails or if indicated by governing agencies.
- WADA recommends all athletes considering taking asthma medications seek a clear diagnosis from a specialist and undergo testing.
- Since 2010, salbutamol (not to exceed 1,000 ng/mL in the urine) and salmeterol by inhalation were removed from the Prohibited List and no longer require a Therapeutic Use Exemption (TUE). Inhaled Formoterol to a maximum dose of 54 mcg/24 hours is no longer prohibited. All other β2 agonists are prohibited and require a TUE.
- A positive response to any one of the provocation tests below is required to confirm airway hyperresponsiveness:
  - Exercise Challenge Tests (field or laboratory) ≥ 10% fall of FEV1
  - The Eucapnic Voluntary Hyperpnea (EVH) test ≥ 10% fall of FEV1
  - Methacholine Challenge ≥ 20% fall of FEV; PC2O < 4 mg/mL if steroid naïve. If taking inhaled glucocorticosteroids longer

than 1 month, then PD2O should be ≤ 1600 mcg or PC2O ≤ 16.0 mg/mL
    - Mannitolinhalation ≥ 15% fall in FEV1 after challenge
    - Histamine Challenge ≥ 20% fall of FEV1 at a histamine concentration of 8 mg/mL or less during a graded test of 2 minutes
- A 12% or higher increase in FEV1 following the use of an inhaled β2 agonist is considered the standard diagnostic test for reversibility of bronchospasm.
- Medication inquiries should be directed to the athlete's National Anti-Doping Organization. USADA's Drug Reference Line 719-785-2000, option 2, provides an expert available Monday through Friday 8 am to 4 pm MST. The Global Drug Reference Online (Global DRO, www.globaldro.org) provides support regarding the prohibited status of medications, but not supplements, based on the current WADA Prohibited List.

## Overtraining Syndrome

- Accumulation of training and/or nontraining stressors resulting in long-term performance decrement, with or without related physiologic and psychological signs and symptoms; restoration of performance capacity may take weeks to months; exclude organic disease.
- Disparity of load and load tolerance
- Cyclists are prone to overtraining because long hours may be spent cycling without a musculoskeletal injury.
- Staleness and lack of improvement more common than frank overtraining syndrome.
- Pathophysiology: Damage in muscle fibers and mitochondria, impaired recruitment of muscle fibers, impaired hypothalamic–pituitary axis, and reduced sympathetic nervous system activity.
- Risks: High training load, nontraining stressors, sticking to a training program when sick or injured, making up lost time (to illness, injury, or missed training), increase in training because of poor performance, monotonous training, and **not enough rest**
- Early signs: Performance decrement, progressive weight loss, poor sleep, increased resting heart rate, increased fluid intake in the evening
- Symptoms: Loss of vigor, fatigue, depression, agitation, insomnia, irritability, restlessness, loss of motivation, poor concentration, heavy legs
- Prevention: Individualize training, build base mileage before speed work, periodization, avoid monotony, rest, carbohydrate rich diet, avoid overcompensating
- Lab tests: **No single identifiable parameter;** exclude organic disease; complete blood count to rule out anemia, infection, cancer; complete metabolic profile to rule out renal or hepatic disease, electrolyte disturbance as clue to eating disorder; thyroid-stimulating hormones, Epstein-Barr virus titer, Lyme disease if in endemic region, vitamin D, magnesium levels. Also consider mycoplasma, cytomegalovirus, parvovirus B19, hepatitis, and chronic sinus or tooth infections. Consider hormonal workup. Glutamine/glutamate decreased. Salivary immunoglobulin A decreased. Consider echocardiogram, EKG, $\dot{V}O_2$ max, stress test, pulmonary function test.
- Two best markers of overtraining: (1) decreased performance on standard exercise tests with HR, lactate, and $\dot{V}O_2$ max; (2) self-analysis of well-being by the athlete such as with Profile of Mood State (POMS)
- Best log book markers: mood, muscle soreness, stress, quality of sleep, and performance decrement
- Overtraining should be discussed not only under the clinical aspect, but more under the aspect of training content.
- Late diagnosis may result in loss of months of racing.
- Cycling treatment: easy riding or complete time off the bike until fresh, return slowly with no hills or hard efforts for 1 month for severe cases. Three days off and 7 days easy spinning on a small gear is sufficient for mild cases of overreaching.

## Gastrointestinal Distress

- Not as common as with running
- Blood flow may be reduced to the GI system by 40% to 50% during exercise with shift toward working muscles, skin, and vital organs.
- In triathlon, majority of issues in the run section are related to the lower GI tract with diarrhea, and in the bike section are related to the upper GI tract with reflux and vomiting. Swallowing water and air during swimming contributes to this. Increased pressure on abdomen from cycling position may also contribute. Dehydration and nutrition contribute.

## Nutrition

- A cyclist with average efficiency and aerodynamics requires about 21 kcal per minute to ride at 40 kph.
- Carbohydrate stores in the body (2,000 to 3,000 kcal) can fuel about 90 minutes of riding at 40 kph.
- During the Tour de France, cyclist energy expenditure may be as high as 9,000 kcal per day.
- The training diet for a cyclist:
    - 60% to 70% carbohydrates = 3.1 g per pound of body weight daily
    - 15% to 20% protein = 0.5 to 0.9 g per pound of body weight daily
    - 20% to 30% fat = *not* less than 20 g per day
- Pre-exercise:
    - Intake of 150 to 300 g of carbohydrate 3 hours prior to exercise will increase muscle glycogen and performance.
- During exercise:
    - 60 to 70 g carbohydrate per hour of training
    - Use of carbohydrate levels off at 1 g per minute
    - Glucose, maltodextrins, sucrose, maltose, and soluble starches oxidized at high rates versus fructose and galactose
    - 300 to 400 calories per hour of training
    - 400 to 600 mL of fluid per hour
    - Drinking ad libitum (according to the dictates of thirst) has been shown to have no detrimental effect on performance
    - Sodium intake of 0.15 to 0.45 g per L of fluid
- Postexercise:
    - Maximum rate of muscle glycogen resynthesis is reached at intake of 1.2 to 1.4 g per minute (75 to 90 g of carbohydrate per hour).
    - Carbohydrate intake of 1.2 g per kg body weight per hour for 4 hours post exercise, preferably within 90 minutes
    - Carbohydrate intake of more than 1.4 g per minute provides no additional benefits in glycogen storage and can increase gastrointestinal distress.
    - Total carbohydrate intake of 8 to 10 g per kg of body weight within 24 hours

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 95 MOUNTAIN BIKING

Christopher C. Madden • Steven J. Collina

## GENERAL PRINCIPLES

### Definitions

- The term "mountain biking" broadly refers to riding bikes with specific design characteristics in various off-road settings.
- Mountain bikes generally differ from road bikes in several ways: a smaller frame, stronger wheels, larger range of gears, a wider flat or upright handlebar, hydraulic brakes, suspension, and wider, knobby tires.
- There are many riding and bike types, with some overlap between bikes and riding styles.
- The five main mountain biking categories are cross-country (XC), downhill (DH), freeride (FR), all-mountain (AM), Enduro, and trials and urban riding (TR) (Fig. 95.1).

### Demographics

- Participation is open to all age groups, with the average age dependent on the type of biking; 22 to 36 years is the gross average, with most competitors aged between 19 and 44 years. There are more males than females, but female participation is increasing; the sport is especially popular among young males.
- Mountain bikes are the largest category of bikes sold in United States (US) bike shops.
- Racing is now common in XC, DH, FR, and other styles.
- The number of noncompetitive mountain bikers is increasing; less is known about their injury epidemiology.
- The sport attracts risk-tolerant personalities.

### Research

- Injuries are reported inconsistently in the literature, ranging from specific injuries to general types of injury (e.g., laceration, fracture) to injured body areas (e.g., joint, upper or lower extremity, head).
- Early data are from competitive XC; now more data are from all areas: XC, DH, and FR. Some recreational data are available; data from other areas are minimal.
- Available studies are primarily descriptive and focus mainly on injuries themselves (generally defined by body or joint area); there are fewer details available regarding mechanisms of injury.
- Most injury studies have a preponderance of male participants (75% to 80%). Many studies depend on victim recall.

### Competition

- XC still popular, but a shift to Enduro, DH, and other events is being seen.
- Categories are established based on age, gender, and rider skill.
- Rider skill categories were changed in 2009 (applicable to riders 15 years of age and above): Pro, Category 1 (previously Expert), Category 2 (previously Sport), and Category 3 (previously Beginner); previous riders classified as "Semi-Pro" must choose between the new Pro or Category 1 tiers.
- Age categories: Youth (<10 years old), Junior (10–18 years old), Under 23 (19–22 years old), Senior (23–29 years old), and Master (>30 years old)

### Protective Equipment

**Protective torso armor and extremity padding:** Primarily for FR and DH; use of lighter, flexible pads increasing in XC

**Helmet designs:** Full face (mainly DH, FR) and standard (mainly XC, Recreational); helmets should be Snell-, CPSC-, or ANSI-approved and tested. Recommend replacement after any significant crash.

**Shorts:** Multiple types of synthetic moisture-wicking chamois; some have built-in hip padding, loose-fitting shorts with inner chamois are becoming more popular

**Gloves:** Varying thickness, with shell protection (dorsal fingers and hand) and padding

**Eyewear:** Ultraviolet protection, shatterproof, changeable light-reducing and colored lenses for varying conditions; clear lenses for night riding; goggles in DH and extreme cold

### Mountain Bike Fit

- Start with a professional fit (see Chapter 94: Road Biking).
- **Mountain bike fit not as straightforward as road fit;** use the initial road fit to help achieve a mountain bike fit window (an ideal, individualized fit that may deviate slightly from a virtual perfect fit).
- For more information, see eAppendix 95-3.

## PHYSIOLOGY AND TRAINING

### Physiology

- High intensity sport—probably higher (especially XC) than road stage races
- XC circuits average about 2 hours, performed at a heart rate (HR) of approximately 90% (± 3%) of HRmax, corresponding to a $\dot{V}O_2$ max of approximately 84%; more than 80% of race time is spent above the lactate threshold (LT).
- Intensity is related to a fast start, several climbs, rolling resistance, and isometric and eccentric arm and leg muscle contractions that are required for shock absorption, bike handling, and stability over rough terrains (increases HR response to submaximal cycling).
- Start has fundamental importance to the entire race as XC riders race to the narrows (where the trail becomes singletrack) to achieve a good position; fast starts and early steep climbs lead to high intensity and HRmax early in the race.
- Anaerobic energy systems taxed, especially during steep climbs (require high power output: up to 250 to 500 watts); anaerobic power and ability to sustain high work rates for prolonged periods are prerequisites for competing in high-level off-road cycling events.
- Various factors may affect off-road XC performance, especially in elite cyclists: $\dot{V}O_2$ max, peak power output (PPO), power output (PO), and $\dot{V}O_2$ at the ventilatory threshold (VT) and at the respiratory compression threshold (RCT); studies are conflicting regarding which is most important:
  - PO and $\dot{V}O_2$ at the RCT normalized to body mass are predictors of off-road performance times.
  - $\dot{V}O_2$ max, PPO, and LT normalized to body mass correlate with XC performance in some studies.
  - PO at the VT may correlate with time trial performance.
- Body mass is a factor: power to weight characteristics are important for success in off-road events; high power-to-weight ratio is good for strong hill climbing ability; higher mass may assist with rapid descents.
- Factors other than aerobic power and capacity may affect off-road cycling performance: cycling experience and economy

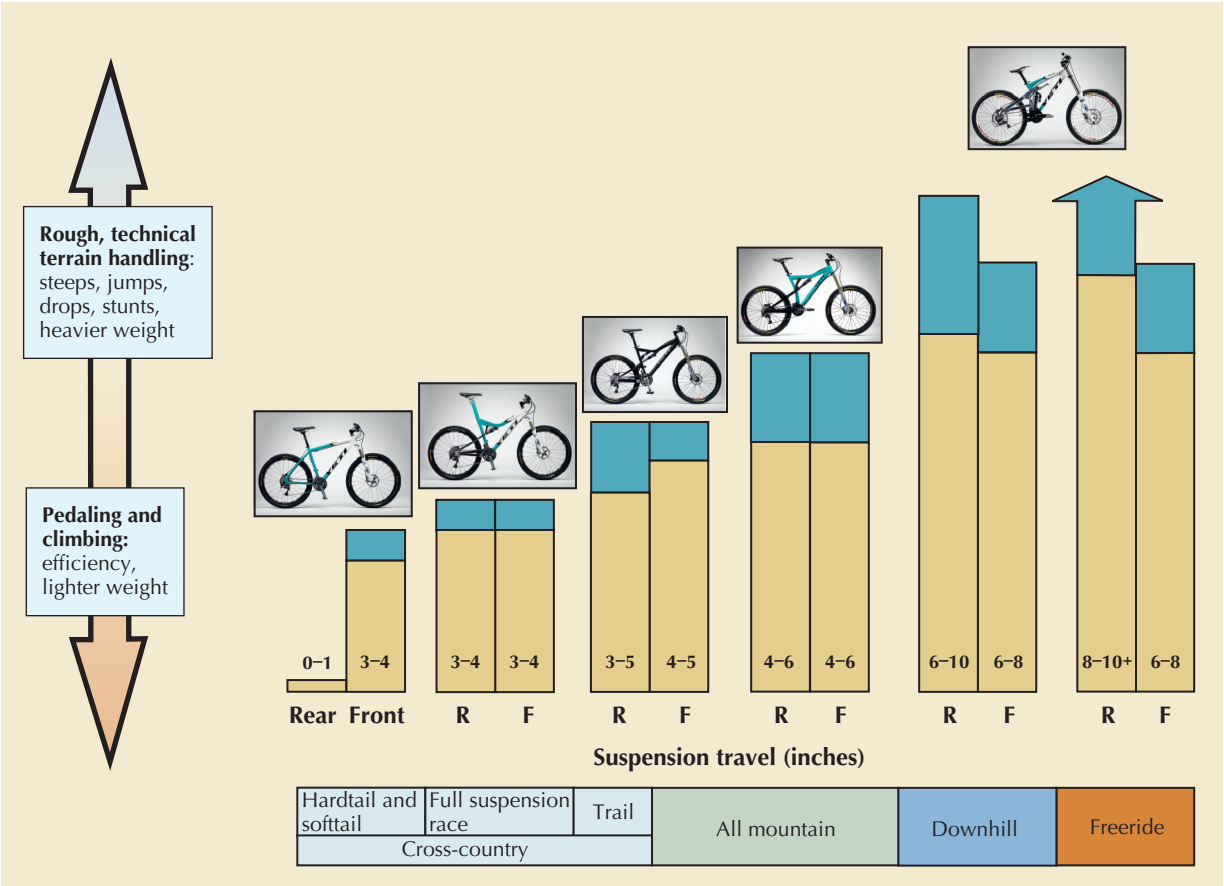


**Figure 95.1.** Mountain bike styles and characteristics. (Photographs from Yeti Cycles with permission. Data from Lopes B, McCormack L: *Mastering Mountain Bike Skills: Techniques to Excel in all Riding Styles.* Champaign, IL: Human Kinetics, 2005.)

(specificity of principle), technical ability, and pre-, during, and postcompetition nutrition

## Training

- Need to develop good aerobic endurance, anaerobic capacity, overall muscle strength, good coordination, and bike-handling skills
- Significant upper body and core muscular strength is necessary for repeated isometric and/or eccentric muscle contractions required to absorb shocks and constantly adjust to changing terrain; accomplished using weight training and riding off-road
- Many competitive XC riders train for 10 to 14 hours weekly; some XC racers train systematically in a similar fashion to road racers. Others train with much less structure.
- Athletes ride at varying aerobic and anaerobic intensities (zones); it is helpful to use a HR monitor.
- Mountain bikers train both on- and off-road.
  - Use different types of training in a similar manner to other endurance events (listed in order of decreasing intensity): race pace, intervals and hills, speed and tempo, endurance, strength, recovery, or overdistance
  - Training cycle (depends on peaking goals): base (4 months), intensity (4 months), peak (4 to 6 weeks), racing (8 to 12 weeks), or recovery (4 to 6 weeks)
  - Off-road terrain incorporates various training types and is less flexible than road riding under controlled intensity.
  - Long off-road rides over rough, technical terrain, especially if at altitude, require longer recovery compared to equidistant road rides.
  - Use periodization and monitor overtraining, especially if training at high altitude and/or frequently on rough terrain.

# INJURY OVERVIEW

## Epidemiology

- Injury types and numbers likely grossly under-reported secondary to varying and limited study design, difference in injury definitions, understudy of a large population of recreational and noncompetitive aggressive riders in all styles.
- Peak incidence: June to August
- Mixed competition and noncompetition data indicate that 50% to 90% riders have been injured in the previous year; one study showed 20% had a significant injury requiring medical attention and were prevented from cycling for at least 1 day; competitive cyclists were injured more than recreational cyclists in earlier studies, but unclear if this still reflects the current trend.
- Reported injury rates (multiyear data) is as low as 0.45% to 0.6% per year in XC, DH, and DS competitions; 0.30% injury rate for recreational cyclists, but some reports are higher; definition of injury rate may vary (e.g., number of injured cyclists per number of starts per 100; number of injured cyclists per 100 hours of race time).
- Injury rates are greater for DH versus XC relative to time spent on bike (0.37 injuries per 100 hours on bike for XC vs. 4.34 injuries per 100 hours on bike for DH).
- There is a possible association between increased hours on bike and injury severity, but some data indicate the presence of fewer injuries in competitive cyclists who spent 1 hour per week more on the bike during the competition season and 3.5 hours per

week more on bike during the off-season compared to those who experienced major injuries.
- Cannot extrapolate injury data from road bikers because road bike crashes are often specific to riding on pavement; many more collisions with motor vehicles, which is rare in off-road cycling (a few case reports of serious injury resulting from mountain bike–motor vehicle collision do exist)
- Experienced cyclists injure bones and joints more frequently than beginners.
- Professional DH racers are more likely to sustain injuries than amateurs.
- Young males are the most frequently injured population, because of the popularity of the sport in this group and the likelihood to engage in aggressive and technically demanding riding styles.
- Injury risk in competing females may be greater than in males.
  - Loss of bike control, less upper extremity strength, fewer riding years
- The number of injuries occurring during racing and training are about equal, but traumatic injuries are more common in races, while overuse injuries are more common in training.
- Injuries are divided into **overuse, acute traumatic,** and **environmental.**

## Mechanisms of Injury and Risk Factors

- Most commonly reported: **excessive speed**, **unfamiliar terrain**, **loss of control** (encompasses multiple factors), **inattentiveness**, **riding beyond ability**, and **riding DH**, **slippery terrain** (approximately 90% may be viewed as errors in judgment) (Table 95.1)
- **Most crashes occur while riding DH.**
- Injuries from loss of control, loss of traction, and mechanical failure result in similar injury patterns.
- Special attention is applied to the cyclist–bike–terrain interface and how it relates to injuries.

**TABLE 95.1 SPECIFIC REPORTED CAUSES AND RISK FACTORS**

| | |
|---|---|
| Rider-related | Errors in judgment and riding technique<br>Excessive speed<br>Inattentiveness<br>Riding beyond ability and loss of control<br>Incorrect braking<br>Improper training and overtraining<br>Female or young male<br>Intoxication<br>No helmet; especially children |
| Bike/equipment-related | Mechanical failures (more common in DH): flat tires, brakes, chains, forks, handlebars, pedals, suspension parts, seatposts, frames<br>Improper fit |
| Terrain-related | Surface: mud, gravel, loose dirt, wet<br>Unfamiliarity<br>Downhill<br>Obstacles and jumps |
| Environment-related | Competition<br>Heat<br>Cold<br>Sun<br>Lightning<br>Orienteering mishaps<br>Animals and reptiles (attacks and collisions) |

### Falls

- Forward fall over handlebar (endo)
  - **Most common direction of fall** and **mechanism of acute traumatic injury**
  - Common causes are rapid deceleration during downhill descent (most common cause of severe injury), hitting an object, improper jump landing, improper braking.
  - Reported injuries are soft tissue injuries and trauma to head, torso, shoulder, upper extremity; head, neck, and face injury.
- Side falls
  - Common causes are sliding out around corners (sideslip), misjudged handling resulting in tip over, and dabbing of hand after losing balance in technical terrain.
  - Reported injuries are mainly soft tissue injuries; leg injuries, especially to knee and ankle; some upper extremity (reaching out or a fall on lateral shoulder).
- Rear falls
  - Common causes are forceful wheelie, preloading (compressing suspension) to adjust for change in terrain, or a jump too early.
  - Reported injuries are soft tissue, upper extremity (especially hand and wrist), head, spine and torso, tailbone.

### Collisions

- Collisions with other cyclists are common in XC.
- Sometimes with stationary object (trees or rocks)
- Collisions with bike parts are common, especially bar, stem, and pedals; frame, brakes, and seat less common
- Animals (prairie dogs and other)
- Injuries from collision and noncollision similar in severity and anatomic location
- One reported fatality in 2015 from blunt force trauma to the chest in an Enduro event

## Evaluation

### History

- Review athlete **training history** to detect common errors (e.g., volume, intensity, hill work, periodization).
- Acute traumatic versus overuse injury
- **Bike fit** history (professional vs. self)
- Experience, type of bike and riding, type of terrain and challenges
- Helmet and other protective equipment use

### Examination

- Use the stationary trainer in the office for overuse and fit issues (dynamic evaluation); may use digital video.
- Evaluate on and off the bike. Identify anatomic variations or malalignments (e.g., excessive knee valgus, leg length discrepancy (LLD) and errors in bike fit; adjust rider; evaluate shoes and orthotics if used.
- Helpful tools: plumb line, long carpenter's level, laser level, goniometer, suspension pump, zip ties (for sag settings), Allen wrenches and screwdrivers (for quick office adjustments)

## General Treatment

- General treatment approaches for traumatic and overuse injuries in off-road cyclists are similar to approaches used with other athletes (e.g., physical therapy, cryotherapy, anti-inflammatory medication).
- Specific cycling injury diagnosis and treatment is a highly individualized area; affected by multiple variables including training, experience, riding style/type, and bike fit.
- Relative rest and activity modification: temporarily decrease mileage, intensity, hill work; spin using low-resistance and high-cadence pedaling; correct training errors.

- Consider placing rider back to neutral position, especially if bike was never fit; adjust from there
- Medication: nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia and inflammation, bacitracin ointment for soft tissue trauma
- Ice appropriate overuse injuries after rides and intermittently throughout day; keep the affected joint warm during ride (e.g., knee warmers)
- Physical therapy (see specific injuries)
  - Lower extremity (especially hamstring, iliotibial band) flexibility
  - Back and neck flexibility
  - Strengthening: lower extremity (eccentric programs for Achilles, patellar, and hamstring tendinopathy), dynamic core stabilization, upper extremity
  - Plyometrics, proprioceptive, and other cycling-specific coordination training
  - Deep tissue massage and release techniques
  - Neural stretching maneuvers
- Consider ionto- or phonophoresis in the appropriate settings.
- Bracing, strapping, or taping where appropriate
  - Knee: soft patellofemoral brace, infrapatellar strap, McConnell taping
  - Hand and wrist: use an off-the-shelf wrist or thumb spica split (can bend steel stay to handlebar, most cycling gloves fit over), custom-molded orthosis (e.g., Orthoplast)
- Corticosteroid injection
  - Upper extremity: carpal tunnel syndrome, de Quervain's tenosynovitis, intersection syndrome
  - Lower extremity: pes anserine bursitis, trochanteric bursitis, iliotibial band syndrome, Morton's neuroma

## SPECIFIC INJURIES

### Overuse Injuries

#### Epidemiology

- Retrospective questionnaire surveys indicate 45% to 90% of mountain bikers have been affected.
- Body regions most commonly involved: **knee** and **low back** most common, then hand, wrist, neck, and buttocks/saddle region
- Likely grossly under-reported: poorly studied, probably common
- Many studies evaluate injuries resulting in lost time on bike; excludes many overuse injuries
- **Bike fit is closely linked**; interaction between cyclist, bike, and terrain
- Anatomic variations and fit errors (even by a few mm) magnified by many hours of training and cumulative repetition; especially affects lower extremity
- Upper extremity injuries related to weight distribution over the front of the bike and are affected by bars, bar ends, grips, and stem height relative to saddle; also related to shifter type and front suspension (travel, preload, rebound)
- Causes and risk factors: **too much training** and **improper training progression** (e.g., sudden increases in mileage or riding intensity, climbing too many hills during the early season, inadequate recovery), too many hours of training/racing, too many different types of riding, too much rough terrain, improper fit, anatomic variations and faults, incorrect saddle position (dynamic), insufficient stretching, incorrect gear ratio (pushing large gears too much, especially during the early season), not enough warm-up, wrong shoes, not enough training (inadequate preseason conditioning), cold weather
- **Treatment and prevention focuses on training and fit.**

#### Knee

May be the most common joint affected by overuse; affects 30% to 40% of mountain bikers

PATELLOFEMORAL PAIN (BIKER'S KNEE, PATELLAR TENDONITIS, QUADRICEPS TENDONITIS)

**Causes:** Saddle set too low, aggressive sprinting, pushing big gears and aggressive climbing during the early season, pes planus and overpronation, excessive genu valgum, LLD, crank arm too long

**Treatment:** Pedal spacers, medial wedge, or a cycling orthotic for excessive knee valgus, raise seat, move cleats rearward, move seat back, set a higher cadence (90 to 100 rpm), easier gears, avoid hills, shorten crank arm, correct LLD (shim), correct training errors

PES ANSERINE BURSITIS

**Causes:** Saddle set too high, pedals with too much float, stance too wide on pedals, LLD, overpronation, tight hamstrings, cleats inappropriately neutral or internally rotated, external tibial torsion

**Treatment:** Lower saddle, correct LLD (short leg) and overpronation with an orthotic, shim, wedge, or by adjusting cleat position (move anterior); adjust cleats to match foot alignment (e.g., toes point out slightly)

ILIOTIBIAL BAND FRICTION SYNDROME

**Causes:** Stance too narrow on pedals, cleats inappropriately neutral or internally rotated, saddle set too high, pedals with too little or too much float, genu varum

**Treatment:** Add a threaded spacer or washer between the crank arm and pedal (correct varus), adjust cleats to reflect foot alignment (toe out), lower the saddle, use pedals with appropriate float, introduce cycling orthotics to control excessive lower leg rotation

PLICA SYNDROMES (ESPECIALLY MEDIAL)

**Causes:** Saddle set too low, genu valgum, overpronation, internal tibial torsion, excessive pedal float

**Treatment:** Raise saddle, introduce cycling orthotics, adjust cleats to reflect foot alignment (toe in), decrease pedal float.

HAMSTRING, POPLITEUS TENDONITIS, AND POSTERIOR CAPSULE STRAIN

**Causes:** Saddle set too high and/or posterior, riding a fixed gear bike (hamstrings used to decelerate), genu varum, pedals with too much float, LLD

**Treatment:** Lower and/or slide the saddle forward, slide forward on seat during steep climbs in granny gear (increases quad workload), pedals with minimal float, use of a threaded spacer or washer between crank arm and pedal, correct LLD, eccentric strengthening program

#### Hip

GLUTEUS MEDIUS PAIN SYNDROME, TROCHANTERIC BURSITIS, ILIPSOAS TENDONITIS, AND HIP EXTERNAL ROTATOR TENDONITIS

**Causes:** Weak and/or inhibited hip abductors and external rotators, genu valgus, overpronation, pedals with too much float, LLD, saddle set too high

**Treatment:** Strengthen hip abductors and external rotators, decrease pedal float, introduce a cycling orthotic, medial wedge or shim, lower the saddle, consider trochanteric bursa corticosteroid injection if there is evidence for bursal fluid on ultrasound (avoid multiple injections due to concerns for gluteus medius tendon rupture)

#### Leg and Ankle

ACHILLES TENDONITIS AND RETROCALCANEAL BURSITIS

**Causes:** Improper pedal form (drop heel, toe pedaling, or combination of both called ankling—excessive dorsiflexion and plantar flexion during pedaling), cleat too far forward, overpronation,

tight Achilles, shoe rubbing (with Haglund's deformity), saddle set too high or too low
**Treatment:** Correct pedal form, move cleat rearward, stretch Achilles, raise or lower saddle depending on problem

TIBIALIS ANTERIOR TENDONITIS
**Causes:** Saddle set too high, ankling
**Treatment:** Lower saddle, anterior ankle stretching, tibialis anterior eccentric strengthening, establish proper pedal stroke

POSTERIOR TIBIALIS TENDONITIS
**Causes:** Overpronation, ankling
**Treatment:** Cycling orthotic or medial wedge, posterior tibialis eccentric strengthening, establish proper pedal stroke

## Foot

HOT FOOT (FOREFOOT/TOE NUMBNESS AND PAIN, METATARSALGIA, PARESTHESIAS, AND MORTON'S NEUROMA)
**Causes:** Improper cleat position, irregular sole (cleat bolt causing localized plantar pressure), tight or narrow shoes, small pedal platform, toe clips, improper rotational cleat adjustment
**Treatment:** Move cleats back (lower saddle same amount) and adjust rotation to individual mechanics, loosen toe clips or convert to clipless pedal, install thinner insoles and/or add metatarsal or neuroma pad, larger pedaling platform, wider shoe or shoe with anatomic footbed, occasional corticosteroid injection

PLANTAR FASCITIS
**Causes:** Tight plantar fascia, overpronation, excessive pedal float, saddle set too low
**Treatment:** Stretch plantar fascia, introduce night splint or sock, cycling orthotic or anatomical footbed, decrease pedal float, raise saddle, neutral position or dorsiflexion night splint or sock

## Hand and Wrist

Wrist pain in 19%, hand numbness in 19%, and finger tingling in 35% of all mountain bikers.

ULNAR (CYCLIST'S PALSY) AND MEDIAN NEUROPATHY (CARPAL TUNNEL SYNDROME)
**Description:** Incidence of compression of nerves at Guyon's canal and carpal tunnel syndrome is probably similar in road and mountain cyclists; presents with motor, sensory symptoms or both; ulnar nerve presents with the most sensory symptoms; experienced and inexperienced cyclists equally affected
**Causes:** Improper grips (size, shape, firmness), infrequent change in hand position, bumpy terrain, improper suspension settings and tire pressure relative to terrain, lack of suspension, "death grip," lack of glove padding
**Treatment:** Proper grip size and comfort fit, frequent changes in hand position (bar ends can be helpful), proper weight distribution on bars and seat (affected by fit and riding style), experimentation with bar angles, padded gloves, instructing the athlete to loosely grip bars, decrease suspension preload and tire pressure, consider front suspension if rigid, stretching while on the bike.

DE QUERVAIN'S TENOSYNOVITIS, EXTENSOR CARPI ULNARIS TENDONITIS, AND INTERSECTION SYNDROME
**Causes:** Occurs especially with grip shift, sometimes with integrated shifters
**Treatment:** Wrist and/or spica splint (dynamic) to minimize provocative motion, change shifters to trigger, consider a local steroid injection

## Muscle Cramps

**Description:** Especially affects quads, hamstrings, and calves, some upper extremity (especially triceps)
**Treatment:** Hydrate with appropriate electrolyte drinks (especially when hot), decrease riding intensity slightly, soften suspension, ensure appropriate training volume, stretching

## Low Back Pain

**Description:** 37% of cyclists are affected; one of the most common overuse complaints, **may be as common as knee pain**
**Causes:** Most frequently by improper fit, riding position, or inappropriate progression of training volume and intensity. Specific causes are over-reaching (stem too long or seat too posterior), too much drop, rough terrain, incorrect suspension preload and tire pressure settings relative to terrain, and training errors (especially too many hills and long rides early in season).
**Treatment:** Proper fit, raise handlebars (especially during early season), shorten stem, correct LLD, core strengthening (also addressing abdominals and iliopsoas), adjust suspension setting to terrain, lower tire pressure slightly and consider changing to wider tires, hamstring flexibility, proper riding form, avoid excessive bumpy terrain and intense hill climbs early season, change positions on the saddle during a ride and stand intermittently, sometimes may need to increase reach if pain is related to crowded position (lengthen stem). Disc injuries and radiculopathy frequently worsen with hills and rough terrain. Certain styles of yoga may be helpful.

## Neck Pain

**Description:** Common in mountain cyclists, similar rider–bike causes and treatments to low back pain
**Treatment:** Ride with elbows appropriately flexed, change neck positions frequently, avoid tensing. Hydration packs worn by most mountain cyclists can augment neck pain. Make sure pack straps are adjusted appropriately. Consider decreasing weight of or eliminating the pack by using water bottles and/or an under-the-seat storage pack.

## Scapulothoracic Pain

**Causes:** Common in mountain cyclists, similar causes to neck pain. Scheuermann's kyphosis (classic or atypical) can worsen with mountain cycling, especially over more aggressive terrain. Hydration packs especially contribute to levator scapula, trapezius, and rhomboid myofascial pain and trigger points.
**Treatment:** Stretching, needling or acupuncture, fascial release, establishing proper rider form, stretching on the bike (especially during long rides)

## Genitourinary

PUDENDAL NEUROPATHY
**Description:** Numbness in saddle region reported in 19% of cyclists. Males affected more than females; females report sensory symptoms.
**Causes:** Compression of the pudendal nerve (and possibly the artery), especially after repeated long hill climbs and many hours on the bike.
**Presentation:** Perineal, penile shaft, and scrotal numbness; impotence/erectile dysfunction and priapism (rare)
**Treatment:** Ensure proper bike fit (especially seat height, fore-aft position, and tilt), proper weight distribution between bars and seat, make sure saddle not tilting up, lower seat, consider change to a softer with a central cutout, use quality cycling shorts with adequate padding, increase handlebar height (which distributes weight more posteriorly over ischial tuberosities), recommend standing during long climbs

SKIN PROBLEMS RELATED TO SADDLE
**Description:** Chafing, callus (ischial tuberosities), maceration and ulceration, and painful nodules. Painful nodules (saddle sores) likely inflammatory (repetitive friction of hair follicle with resultant inflammation and scarring), but can be infectious;

considerations: folliculitis, furuncles, carbuncles, pseudocysts, hidradenitis suppurativa

**Treatment:** Mostly aimed at prevention. Proper seat height and fore-aft, level seat position; individualize saddle fit (consider softer saddle with center cutout and/or broader rear); proper reach and bar height; proper hygiene (skin and shorts, shower immediately after ride, and wash cycling shorts between every ride); intermittent standing; avoid wearing low-quality underwear; use well-padded cycling shorts; lubricants or chamois creams are frequently used (petroleum-based probably better than water-based); antibiotic ointments or gels (bacitracin, clindamycin) can be used for infection and for prevention if recurrent folliculitis or hidradenitis suppurativa; occasional corticosteroid injection of nonresolving painful nodule; sometimes requires surgical resection

URETHRITIS

**Description:** Traumatic irritation of urethra from rough terrain and/or improper rider position and fit

**Symptoms:** Variable; urethral paresthesia, dysuria, hematuria, sometimes pyuria

**Treatment:** Similar to pudendal neuropathy

PROSTATE

**Description:** In healthy men, the measurement of total prostate specific antigen (PSA), free PSA, and complexed PSA is not disturbed by long distance mountain biking or endurance exercise.

**Treatment:** Based on the present data, there is no evidence for a recommendation to limit bicycle riding or physical activity before any measurement of PSA.

SCROTAL ULTRASOUND

**Description:** One study compared mountain bikers to healthy males and found 94% of mountain bikers had abnormal findings compared to 16% of the control group. Another study compared mountain bikers to road cyclists, and found that 94% of the mountain bikers had abnormalities versus 48% of the road cyclists. Most common abnormalities include scrotoliths, spermatoceles, and epididymal calcifications.

**Treatment:** Clinical significance is unclear.

### Overtraining

**Description:** Aggressive terrain, often with repeated hill climbing, may contribute to overtraining in mountain bikers who are not careful to incorporate adequate recovery between rides. May especially be a factor during the early season, after a "melt out" in colder regions, when mountain bikers tend to get spring fever and increase their training volumes rapidly because of a desire for trail time. Altitude training may increase need for recovery.

**Treatment:** See Chapter 23: High Altitude Training and Competition and Chapter 28: Overtraining. Mountain cyclists often complain of "heavy" legs on the bike, especially during hill climbs (a loss of power is also noted) and with stairs.

## Acute Traumatic Injuries

### Epidemiology

- Most injuries are minor (e.g., skin and soft tissue wounds); second most common are orthopedic injuries such as fractures, sprains, and dislocations; most commonly affecting the upper extremity in XC and DH studies (Table 95.2).
- Reported more than overuse injuries in literature
- Injury severity measures vary: level of treatment required, lost riding time, different injury registries
- General trend of frequency for body area (not specific to riding style): upper extremity (fingers and wrist are most common) injuries are more common than lower extremity (knee and ankle are most common), which are more common than head and trunk injuries.
- **Extremity injuries** are more common than any other body area.
- General trend of injury type, in first-to-last order of greatest incidence: skin lacerations, wounds, and contusions; joint injuries (ligament sprains and dislocations); fractures; muscle injuries; neck/spinal and brain injuries (concussions are most common); facial and dental injuries, abdominal injuries, and genitourinary injuries
- Ligament injury trend by location, in first-to-last order of greatest incidence: acromioclavicular (AC) joint, knee, ankle, and fingers
- Off-road cyclists sustain more fractures, dislocations, and concussions than road cyclists.
- Causes and risk factors (see the Mechanisms of Injury and Risk Factors section): Often multifactorial with some connection to bike fit (related to bike control), falls/unscheduled dismounts, collisions, problems releasing from pedals (inexperience and release adjustments for some pedals are similar to ski bindings; avoid pulling up and off pedal), contact with rocky terrain.

**TABLE 95.2 BICYCLING-RELATED INJURIES**

| Injury | % |
|---|---|
| **Orthopedic** | 46.5 |
| Fractures and dislocations | 68 |
| Lower extremity | 29 |
| Upper extremity | 25 |
| Soft tissue | 29 |
| Other (including nerves and tendons) | 3 |
| **Head** | 12.2 |
| **Fatality** | Rare, most commonly due to intracranial hemorrhage |
| **Other** | |
| Spine | 12 |
| Chest | 10.3 |
| Facial | 10.2 |
| Abdominal | 5.4 |
| Genitourinary | 2.2 |
| Neck | 1 |

Most riding likely FR (North Shore style riding); from 1992 to 2002, 399 patients sustained 1,092 injuries while mountain biking; 1,037 total patients identified with bicycling-related injuries. 67% patients required surgery.

Data from Kim PT, Jangra D, Ritchie AH, et al. Mountain biking injuries requiring trauma center admission: a 10-year regional trauma system experience. *J Trauma*. 2006;60(2):312-318.

### Skin and Soft Tissue

- **Contusions, lacerations, and abrasions are the most frequently reported injuries** (up to 65% to 75% of reported injuries): contact with rocky, varied terrain, sometimes pedals and chainrings
- Road or "rock" rash: abrasions affecting small to large surface areas, usually the lower extremities (with some incidences of upper extremity and torso injury), caused by contact with rocks and other rough terrain. Appropriately irrigate and cleanse, cover with a hydrocolloid, protective dressing (e.g., Duoderm) and antibacterial ointment (bacitracin), oral antibiotics should be administered if infection is present.
- Prepatellar bursa laceration, scalping, olecranon bursa laceration, ragged lower leg lacerations (problems releasing from clipless pedals, injury on chainrings, grease in wound, skin loss, debridement, and delayed primary closure concerns)

### Upper Extremity

- Upper extremities (57%) injured more frequently than lower (21%) (not the case in FR)
- **Shoulder: most frequently injured joint in mountain biking**
  - Dislocation and rotator cuff injuries (arm raised during forward fall, anterior or posterior depends on arm position relative to body with fall)
  - **Clavicle fracture** and acromioclavicular separation: falls on lateral shoulder are common; sometimes, force is transmitted through the arm axially.
  - **Acromioclavicular separation is the most common joint injury.**
  - Prevention: learning how to fall and roll is key, especially over the bars: cyclist should snap the bike under the body like a center snaps a football, tuck and roll (can turn endo into two-footed landing or tuck and roll); fall to side: avoid locking elbow while reaching, absorb impact with whole body.
- **Finger** dislocations and fractures, metacarpal fractures
  - Caused mainly by falls or catching a finger on an object (e.g., sapling) while riding
  - Buddy taping, clamshell type or custom-molded Orthoplast brace for protection (mold using handlebars)
- **Wrist injuries:** falls resulting in wrist hyperextension, bracing and collisions
  - Sprains, including of the distal radioulnar joint and intercarpal ligaments
  - Triangular fibrocartilage complex tears
  - Extensor carpi ulnaris subluxation
  - Fractures: radius most common, also ulna, scaphoid, hook of hamate
  - Prefabricated wrist splints and custom orthoses (Orthoplast) that limit provocative repetitive motion may allow an earlier return to riding.
- **Elbow:** Falls resulting in direct impact or axial load
  - **Radial head fracture (most common elbow fracture), radial neck fracture**
  - Subluxation/dislocation
  - Olecranon fracture
  - Medial epicondyle fracture

### Lower Extremity

- Hip and pelvis: side fall with lateral impact
  - Trochanteric bursitis (traumatic)
  - Intra-articular injuries: labral tears, bone bruise, hip fractures
  - Pelvic fractures
- Knee: More frequently injured with falls in DH and FR than in XC
  - Ligament injury common (especially FR and DH); injury to collaterals (especially medial) probably more than to the anterior cruciate
  - Meniscal injuries occur, other intra-articular injures less common
- Ankle and foot: forward momentum can be augmented by bike rider plus bike weight, combined plantar flexion and inversion on contact foot/ankle
  - **Inversion sprains most common**, syndesmosis and medial sprains sometimes occur
  - Other intra-articular injuries (e.g., talar dome lesions)
  - Can sustain midfoot sprains, but are less common, probably due to relative protection from torsional rigidity of cycling shoes
  - Prevention: Use of stable, torsionally rigid shoes with a strong heel counter, practice dismounts to enhance proprioception and reaction time, bracing, strengthening

### Fractures

- **The second most common injury behind skin and soft tissue injuries**
- Upper extremity incidence greater than lower extremity overall
- Women affected more than men (bone mineral density issues have been suggested as a cause but has not been proven). Bone mineral density for male mountain cyclists higher than comparable road cyclists
- **Clavicle is the most common fracture,** fingers may be second, radial head, distal radius (also common), olecranon, medial epicondyle, scaphoid, metacarpals, phalanges, and other forearm fractures.
- Other reported fractures:
  - Pelvis (few details available), proximal femur, open and closed tibia and patellar
  - Tarsometatarsal fracture–dislocation
  - Trunk: ribs and scapula
  - Cervical > thoracic, and lumbar spine
  - Various others less common

### Muscle Strains

- Back, thigh, and calf common

### Spine

- Cervical and lumbar fractures reported
- **Cervical** most common site of injury, followed by thoracic, then lumbar
  - Most common cervical spine injury caused by fall over bars and landing on head
  - Spinal cord injuries reported in FR (24% total spine injuries). Some paraplegics, quadriplegics, central cord syndromes, some nerve root injuries
  - About half of reported FR spine injuries required surgery.
    - Vertebrae C2 to C3 subluxation with cord compression (halo traction treatment, result: incomplete C3 tetraplegia)
    - Vertebrae C6 to C7 bifacetal dislocation with cord transaction and cord edema to C2 (halo traction treatment, result: C3 complete tetraplegia necessitating tracheostomy and ventilatory support)
    - Vertebra C4 burst fracture body, C3 lamina fracture, nondisplaced C1 arch fracture (C4 vertebrectomy with grafting and plating, result: C4 complete motor and incomplete sensory tetraplegia)
  - All of the above listed injuries involved falls on head; mechanism flexion–compression and axial compression; helmet showed significant damage in all three cases.
  - Assume cervical spine injury if altered mental status and damaged helmet; have a low threshold to obtain CT and/or MRI scans if plain radiographs are negative and suspicion high.
  - The cervical spine is the least protected and most mobile region of vertebral column.
- T5 to T6 vertebral body fracture and lumbar fractures reported

### Abdomen and Chest

- Uncommon in mountain biking; blunt trauma from bike bars and bar ends (exposed bar ends are uncommon now)
- Chest wall injuries most common: rib fracture, intercostal injury, sternum fracture
- Liver–subcapsular hematomas (straight bar ends)
- Pancreatic transaction
- Small bowel evisceration
- Renal laceration and hemorrhage
- Hemothorax, hemopneumothorax, pneumothorax
- Blunt cardiac injury and cardiac contusion
- Solid organ injuries more common than hollow viscus or abdominal wall injuries
- **Spleen most frequent solid organ injured** (49%), then liver (15%); injury to adrenals and pancreas have been reported.
- Small bowel most frequent viscus site of injury; then the mesentery, colon, and omentum

- Spleen injuries required surgery in 25% of cases; 17% of liver injuries and most bowel injuries also required surgery.

### Other

- Genitourinary
  - Straddling most common mechanism of injury
  - Cavernosal artery laceration
  - Perineal and vulva laceration, contusion, hematoma
  - Scrotal and testicular injuries more common than penile shaft
  - Ureter injuries
- Brain
  - Concussion incidence is common; a fall over the bars is the most common mechanism of injury
  - Intracranial hemorrhage rare, but occurs with helmet use, and is the most common cause of death; may be more common in FR than DH due to higher velocities and forces compared to XC
  - Bleeding or contusion to the cerebral cortex more common than that to the cerebellum, followed by the brainstem
  - Most mountain bikers wear helmets (80% to 90%), but they are less commonly used in children and during recreational riding.
- Eye
  - Foreign objects: insects, gravel, tree branches, sleet
  - Shatterproof lenses and goggles (DH) help prevent eye injuries; should be breathable to avoid fogging
- Facial and dental
  - Usually caused by fall over handle bars
  - Facial and dental fractures, lacerations, and abrasions
  - Most common facial fracture: maxilla (FR)
- Fatal
  - Brain injury, ruptured diaphragm, transected coronary vessel, pulmonary contusion, and others are generally not reported.

## INJURY PREVENTION

- Appropriate bike fit (helps prevent overuse injury more than acute traumatic injury)
- Mountain biking skills development (more effective in preventing acute traumatic injuries than overuse): workshops or clinics, skills manuals, home skills drills, and riding time and practice can assist in developing mountain biking-specific skills that will improve riding and reduce injuries (Box 95.1).

**BOX 95.1 BASIC MOUNTAIN BIKING SKILLS**

- Mounting and dismounting in various situations
- Braking
- Cornering
- Wheelies and hops
- Drops (slow and fast)
- Jumping
- Line selection and flow
- Pedaling efficiency
- Climbing
- Switchbacks

- **Riders, sponsoring organizations, manufacturers:** improvements in rider training, race course design, and safety equipment
- Rider-related: technical skill and neuromuscular coordination drills; strength and endurance training, especially the upper body and core; awareness of limitations (riding near "the edge"), especially when riding with more experienced cyclists or on unfamiliar terrain; avoid intoxicants.
- Bike/equipment-related: use of padded and rigid protection (e.g., bulky chest and shoulder protectors, shin guards), full face helmets for DH and FR, appropriate shoes (clipless, stiff sole, flat bottom in DH and FR), padded shorts, padded gloves, regular bike inspection and maintenance, appropriate tire setup (tubeless conversion, appropriated width, tread, and compound for setting), ensuring wheel build strength matches riding type (rim strength, number and size of spokes, type of hubs), grips (cover ends, no-slip lock, curved bar ends [if used])
- Terrain-related: responsible race organizers; avoid exciting "spectator spots," adjust course and number of laps to skill and competition level; note risky surfaces (gravel, mud, wet)

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eAppendix 95-1 Types of Mountain Biking
- eAppendix 95-2 Bicycling Modernization and Specialization
- eAppendix 95-3 Mountain Bike Fit
- eAppendix 95-4 Environmental Injuries

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

# eBOOK SUPPLEMENTS

## eAppendix 95-1 Types of Mountain Biking

### Cross-Country (XS)

- Most common form; most aerobic form
- Point-to-point or circuit riding over varied terrain, including climbs and descents, but generally less aggressive and extreme than FR or DH
- Races are mass start over a circuit course. Lengths vary, but lengths up to 30 miles are common, and ultra-endurance (over many hundreds of miles), 24-hour, and multistage events (over multiple days) are popular.
- Lightest bikes (21 to 28 pounds); lightest parts; more aggressive frame angles; short travel suspension common (3 to 5 inches), front or both front and rear; clipless (higher level or competitive) or toe clip pedals (recreational)

### Downhill and Freeride (DH)

- DH gravity time trial competition consisting of riders racing one at a time, against the clock, down a trail, jeep road, fire road, or a combination; FR similar to DH riding against the clock and over extreme terrain.
- DH courses and FR terrain are technically challenging and feature high speeds, steeps, jumps, drop-offs (ranging from 10 to 40 feet) and other natural and man-made technical features.
- FR terrain may include rocks and cliffs, man-made structures such as interconnecting wooden bridges, drops, logs, bridges, gap jumps, and other stunts.
- DH trials typically last for 5 to 10 minutes; speeds exceed 50 mph; ultimate test of nerve and bike control, much skill required
- DH bikes usually shuttled to top, not ridden; FR can be ridden, sometimes pushed between destinations
- FR also referred to as "North Shore," black diamond, and big hit riding
- Requires significant total body, aerobic, and anaerobic strength
- Body armor and full face helmet used
- Heaviest bikes (32 to 50 pounds); considerable overlap between FR and DH; frames with slack angles and large tubing; DH bottom bracket may be lower than FR; powerful, large disc brakes; longest travel suspension (6 to 10 inches), always front and rear; platform (more popular in DH) or clipless (more popular in FR than DH) pedals are used (sometimes a hybrid variant with clipless on large platform).
- "Slopestyle" riding is a variation of FR that combines big air freeride stunts with BMX style tricks; events held at mountain bike parks.

### All-Mountain (AM)

- Increasingly popular style; broadest range of riding
- Riding over aggressive XC to challenging AM natural terrain, may approach light FR at the extreme end of spectrum
- More aerobic than anaerobic
- Bikes often referred to as "trail bikes," are slightly heavier than XC (28 to 35 pounds, broad range depending on need); frames with slacker angles than XC and steeper than DH and FR, but the heaviest bikes of the class are designed to climb well; bottom bracket is higher than XC and DH; longer travel suspension (AM: 5 to 6.5 inches), always front and rear; heavier and stronger parts than XC; usually clipless pedals

### Enduro Racing

- A form of racing that typically has a greater proportion of downhill sections
- There are usually at least four timed sections; the combined time determines the overall winner.
- Combines downhill technical expertise with cross country fitness
- All-mountain-type bike is used.
- Less protective equipment is typically used compared to downhill racing because the rider must ride from one timed section to the next, which requires climbing

### Trials and Urban Riding (TR)

- Riding that involves hopping and jumping onto and/or over obstacles, and incorporates a variety of tricks and stunt maneuvers
- Can be performed almost anywhere, ranging from competition (observed trials) to "urban assault" (trials type riding using urban or city obstacles)
- Riding involves highly developed balance; emphasis on style, originality, and technique.
- A broad range of trials bikes, ranging from XC mountain bike conversions to smaller, specialized trials bikes; wheel size ranges from 20 to 26 inches; frames are smaller and lower; seats are low or absent; usually have front or no suspension; platform pedals.

## eAppendix 95-2 Bicycling Modernization and Specialization

### Bike

- Modern mountain bikes have evolved into sophisticated, expensive machines, and many have complex suspension systems.
- Designs are tailored to specific events, but considerable overlap between styles still exists.
- Complex front suspension and dual suspension systems:
  - Increases rider comfort, control, and performance
  - Reduces physical trauma from excessive vibration, and makes more risky maneuvers possible
  - May be significant differences in power output between front suspension and dual suspension bikes during uphill climbing, but differences may not translate into increased oxygen cost or time to complete an event; newer valved or platform technology reduces inappropriate suspension engagement, as well as pedal "bob," and probably decreases metabolic cost further
  - Dual suspension now mainstream on climbing bikes: can help preserve power and contribute to forward momentum with acquired usage skill
  - The dropper seat post is a relatively new innovation which allows on-the-fly seat height adjustment. Dropped for steep downhills and raised for flats and climbing
- The frame tubes angles vary relative to each other and determine handling characteristics.
  - Common angles: head tube, seat tube
  - Steeper angles generally create more responsiveness regarding turning the front wheel; slacker angles create more stability in high-speed cornering, but handle more clumsily or less responsively over "tight" terrain
  - Some manufacturers offer female- and youth-specific geometries.
- Multiple shifting options: grip, trigger, integrated (same brake and shifter handle)
- Broad gearing options: one to three rings up front (size varies), nine to 10 gear clusters/cogs in rear; lose "in-between" gear range with single front ring, but the setup is lighter and does not require a front shifter; many variations available
- Powerful brakes: composed of a hydraulic disc and mechanical disc primarily; strong stopping power; preserves forearm and hand strength and endurance; "one-finger" braking and excellent modulation allow for better bike control
- Multiple clipless (like mini ski binding) pedal systems: vary in rotational release angle and tension, rotational and lateral float, adjustability, ease of engagement and release, and ability to shed mud

- Platform or "flat" pedals mainly used in DH, sometimes FR, AM, and Trials bikes; allow for immediate dismount; have small, sometimes sharp pins so that shoes do not shift on pedals, which can cause shin injuries
- Toe clips mostly used by recreational cyclists; rare on aggressive or competition bikes
- Handlebars are wider and more upright
- Tires available in broad range of widths (1.95–2.4 inches most common, larger in DH), tread patterns (semi-slick to knobby), and compounds (soft, sticky rubber to firm rubber, or mixed); matched to riding conditions; can be a factor in crashes
- Newer tire sizes include "plus"-sized tires (2.8–3.0 inches) for an improved ride with less mechanical suspension, and "fat bike" tires (3.8–5.0 inches) allowing snow riding and racing.
- Tubeless tires gaining popularity over tubes (tubeless specific rims and tires vs. tubeless conversions using a non-tubeless rim and a tire with rubber liner and sealant)

## eAppendix 95-3 Mountain Bike Fit

- Directly applicable to overuse injuries, indirectly to acute traumatic injuries
- A static bike fit (using a standardized bike fit system) is a good place to start in order to identify a neutral position as a starting point for individualized adjustments.
- Dynamic fit is better than static due to better individualization and customization; digital video and anatomic markers are sometimes used.
- Experienced physician's office or a high-end bike shop will offer help with fit customization.
- Important to note that riding position and repetitive forces remain relatively constant in road cycling, but mountain bikers frequently change rider position while riding over varied terrain; leads to broader, less repetitive forces
- Fit adjustments for a mountain bike follow the same order as a road bike: frame size, saddle height, saddle fore-aft (to establish neutral knee), saddle tilt, reach and stem/handlebar height. Keep in mind that changing one usually affects the others. Various fit guide tips are available online.
- Frame size for a mountain bike is likely smaller than that for a comparable road bike, depending on riding style.
  - Top tube should have 3 to 6 inches clearance with standover (applicable with non- or minimally sloping tube) (shoes on).
  - Mountain bike inseam method: (____cm inseam × 0.67) × 10–12 cm = ___–___cm frame size (shoes off).
  - Mountain bike frame designs vary broadly, so check specific seat tube and top tube lengths; top tube length is more important than seat tube length (the latter is more adjustable) with mountain bike fit; frame angles and suspension type and design must be factored in because they will affect resultant ride.
  - Take measurements and frame angles from a previous mountain bike that fit well; sometimes difficult to transfer over due to variations in frame, suspension designs, and geometry

### Saddle Height

- Many mountain bikers lower seat at least 1 cm from a road riding position to increase control, assist with steep climbing, and to allow easier dismounts; can measure crank arms and lower the seat by an amount equal to the difference in crank arm length between the mountain and road bike (e.g., mountain: 175 mm, road: 170 mm; therefore, lower mountain saddle by 5 mm).
- Quick-adjust drop seatposts are now popular (riders often drop post before the downhill section after a long climb): a lower saddle is more maneuverable and controllable (DH and FR); higher achieves more aerodynamic position (XC racing).

### Saddle Fore-Aft

- A plumb line or level dropped from the inferior pole of patella with the pedal at 3 o'clock (forward, parallel to ground) should bisect the pedal axle (some perform the drop behind the crank arm, in which case it should bisect the center); make sure suspension sag set appropriately.
- Long climbs: some mountain riders prefer to set seat back from this position to increase rear wheel traction and leverage (especially if pushing large gears)
- Sprinters: some prefer forward cleat position; neutral is safest
- Seatpost: standard (clamp on top of post) or setback (clamp rearward from top of post) available
- Do not use fore-aft setting to compensate for improper reach.

### Reach and Stem/Handlebar Height

- Most individualized part of mountain bike fit; style of riding will affect adjustment/setup significantly
- Initial reach should be set so that torso is 45 degrees to ground; reach typically more upright than in road cycling; influenced by riding style, flexibility, and comfort.
- Longer reach and lower drop for competitive XC riders; shorter and higher to achieve a more upright posture for novices and for certain riding styles (e.g., DH, FR, DJ)
- Reach is affected by stem length and angle, flat or upright handlebars, top tube length, saddle fore-aft position.
- Handlebar and stem heights are adjustable: initial steerer tube length (determined by cutting initially long tube; take care not to cut too short), spacers between headset and stem, stem angle, and handlebar type (flat or upright, width, sweep).
- Handlebar width: grips initially set at or wider than shoulder width; a narrower setup increases steering responsiveness; a wider setup increases stability and leverage.

### Suspension

- Various suspension settings combined with a broad range of frame designs can make the initial setup challenging; settings may include sag, preload, rebound and/or compression damping, as well as pedal platform adjustments.
- Sag: amount that the travel suspension compresses with static body weight over it. Preload: the amount of initial spring compression, which controls when the spring begins to move
- Less preload = more sag; more preload = less sag
- Sag is set by placing the bike on a trainer, starting with no preload or the easiest valve setting and recommended air pressure, having the rider gently climb on bike to weight suspension, placing a zip tie around shock stanchion, and measuring the amount it slides (percentage of total suspension travel in mm) with rider weight; add or remove air pressure or change preload setting to achieve ideal.
- General sag recommendations: XC racers: 12% to 20%; DH racers: 30% to 40%; recreational cyclists: 20–30% (see manufacturer's manual or discuss with a bike shop professional for variations)

### Shoe–Cleat–Pedal Interface

- FR, TR, and DH use platform pedals with studs or "pins" that dig into flat shoes, this setup can cause significant pretibial and other soft-tissue injuries if feet slip off pedals; DH (especially during competition) cyclists often uses clipless pedals, but some use platform or hybrids.
- XC uses clipless (mini ski binding) with adjustable release spring; clipless mechanism and platform size varies.
- Cleat placement is adjustable fore/aft, medial/lateral, and rotationally; usually with two Allen screws attaching to shoe.
- Foot moves opposite of cleat adjustment.
- Initial cleat placement so that ball of foot (metatarsal heads) directly over pedal axle

- Rotational "float" (amount of allowable rotation on pedal before release) varies with clipless pedal designs: Shimano and most other SPD-type (≤4 degrees) have less float than Time ATAC (approximately 5 degrees), Crank Brothers Candy, Eggbeater (approximately 6 degrees), or Speedplay Frog (20 degrees "free float").
  - The ideal amount of float varies with terrain and the biomechanics of the rider–bike interface and must be individualized; less float results in more power, whereas more float results in increased muscle work to maintain foot stability on the pedal, but more versatility for small adjustments over rough terrain.
  - Set the initial rotational cleat position (center of float; toe-in, neutral, or out) to individual foot mechanics: may estimate by observing foot rotation of the rider sitting on a table with legs dangling (hip and knees at 90 degrees, ankles at neutral, have rider bend forward to see if affects foot position), or may determine more exactly using rotational adjustment device (RAD).

### Other

- Mountain bike shoes: less stiff than road, more traction on sole (can interfere with clipless mechanism), hybrid hiking-clipless shoes available (some ultraendurance and epic riders use due to significant time spent hiking off bike)
- Crank arm length: mountain bikers use long levers, 170–175 mm is typical
- Cycling orthosis and anatomic footbeds:
  - Extend through metatarsal head where force transfer occurs
  - More rigid than running orthotics to provide better control and force transfer (with varying foot orientation on pedal)
  - Anatomic footbeds often incorporate a varus wedge to accommodate a canted forefoot position, longitudinal arch support to optimize force transfer, and a metatarsal button to minimize nerve and vessel compression at metatarsal heads with pedaling.
- Shims, medial (varus) wedges, and spacers:
  - **Make adjustments to longest leg first.**
  - Shims: if leg length discrepancy (LLD) is more than 6 mm, use shim (especially with back and knee pain); between 3 to 6 mm, it is possible to simply move the cleat on the long leg back and/or the cleat on the short leg forward 1 to 2 mm; also possible to combine shims and cleat position adjustments (especially for femoral-based LLD)
  - Commercial wedges, biowedges, or custom wedges available (use material with minimally compressibility and high torsional rigidity)
  - Threaded spacers better than washers to achieve ideal stance and accommodate varus knee malalignment; placed between pedal and crank arm
  - May correct femoral LLD using combination of shim on shorter leg and cleat position
- **Rider–bike–terrain interface:** See Fig. 95.2 for factors and components to consider with bike fit and terrain goals.

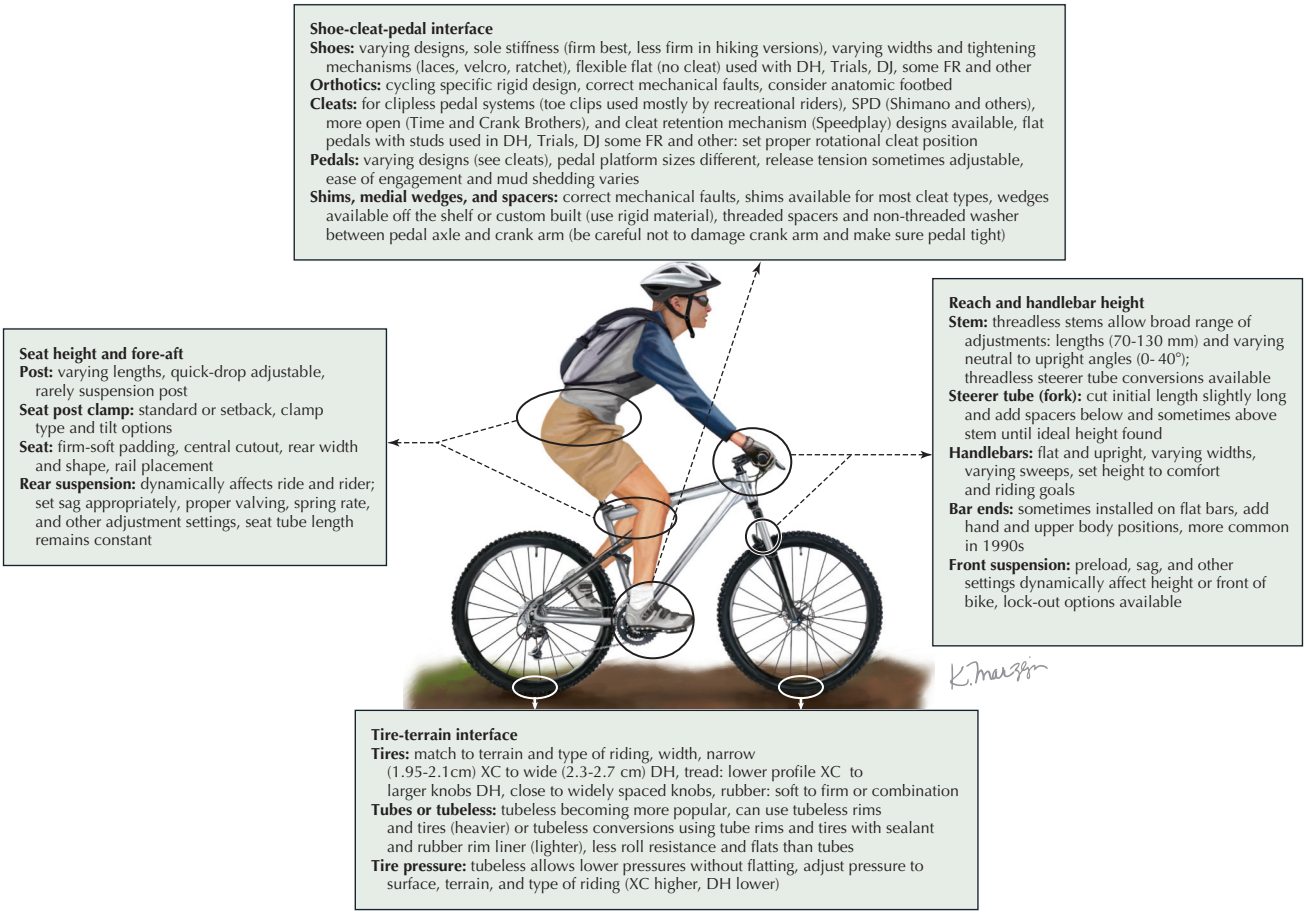


**Figure 95.2.** Rider–bike–terrain interface.

## eAppendix 95-4 Environmental Injuries

- These injuries are reported rarely or not reported at all in the literature but remain a concern, especially for mountain bikers who venture far into the backcountry on epic rides, which is becoming more common.
- Pulmonary: exercise-induced bronchospasm (especially in cold weather), dust bronchitis (mass start endurance and ultraendurance events), high-altitude pulmonary edema (especially for events at higher than 10,000 feet)
  - Pharmacologic pretreatment (salmeterol in longer events for exercise-induced bronchospasm), wear mask or cover mouth and nose with a handkerchief in dusty conditions
- Altitude illness (acute mountain sickness most common)
  - Adequate acclimatization techniques, pharmacologic prevention if a history is present
- Exposure: heat illness, cold injury, sunburn
  - Adequate hydration pack and planning, light-colored and breathable jerseys (heat), adequate helmet ventilation, sunscreen, proper layering and coverage (gloves, head cover, shoe covers, windproof jacket and tights) for cold
- Dehydration: road cyclists traditionally use water bottles held in cages attached to the frame, but mountain bikers typically use hydration packs that carry much larger volumes of replacement fluids and other necessities
  - Match fluid reservoir size to needs of event, timed drinking (using slight weight loss < 2% and urine color and frequency to monitor needs in long events), carry water purification tablets or a small filter in backcountry.
- Weather: sunburn, lightning, sudden rain or snow, changing temperatures
- Wildlife: attacks are rare (cougars, rattle snakes), collisions can cause a crash (prairie dogs and other small animals)
- Collisions: trees, rocks most common; cliffs and drops less common; motor vehicle involvement less common than in road cycling
- Orienteering mishaps and getting lost
  - Carry detailed topographic maps, compass, GPS.
  - Use a buddy system, carry personal identifier, notify others of planned starts and stops.
  - "Adventure pack" with appropriate tools, extra calories, head lamp, fire-starting material, extra clothing layers, appropriate first aid, cell phone if access is available

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# 96 INLINE SKATING, SKATEBOARDING, AND BICYCLE MOTOCROSS

Erica L. Kroncke • Craig C. Young

## INLINE SKATING

### History

- Inline skates date back to 1849; Louis Legrange crafted a pair with wooden wheels to simulate ice skates for a scene in Giacomo Meyerbeer's opera *La Prophète*.
- In the 1970s, inline skating was primarily limited to hockey players looking for a way to practice during summers.
- Its popularity exploded in the 1990s; it became the fastest-growing recreational activity of the decade.
- In 2014, there were an estimated 4.7 million inline skaters in the United States (US) according to the National Sporting Goods Association (NSGA).

### Equipment

**Boots:** Higher and firmer boots provide more ankle support and better control and are used by beginners and trick skaters. Lower boots allow more ankle flexion and are used by speed skaters.

**Frame:** Shorter frames are more maneuverable; longer frames are faster and more stable. Most recreational skaters use four-wheeled frames. Speed skaters use five-wheeled frames. Artistic skaters use two- or three-wheeled frames.

**Wheels:** Wheel sizes vary depending on skating style. An average recreational skate has wheels that are 72–90 mm in diameter; smaller wheels (48–72 mm) are used for tricks and dancing; larger wheels (80–100 mm) are used for speed skating. Harder wheels are faster and more durable, but soft wheels may have better grip. There are elliptical and rounded profiles.

**Bearings:** Higher-rated Annular Bearing Engineering Committee (ABEC) scale bearings are better in overall quality and theoretically translate into faster and smoother rides with less friction.

**Brakes:** Most inline skates have a hard rubber brake built into the heel of one of the skates, creating friction against the pavement. The standard rear brake pressure does not allow quick or efficient stopping. Older styles require the skater to lift the toe, causing loss of contact between the front wheels and the ground, reducing stability. Expert skaters, trick skaters, figure skaters, and speed skaters often use skates without brakes. Other braking techniques include spin stops, dragging the skate behind the body, or skating into grass.

### Competitions and Skating Styles

- Competitive inline skating events:
  - Speed skating (sprint and long-distance events)
  - Team sports (roller hockey and roller derby)
  - Group skating as a social activity—one of the largest groups to skate city streets is the Pari Roller in France, involving up to 35,000 skaters in one night
  - Figure skating and dancing
  - Half-pipe or "vert" competitions, wherein the tricks are performed at or above the rim of the pipe
  - Slalom racing thorough cones
- Recent growth has been observed in increasingly aggressive skating events such as "big air competitions" that rate competitors on height of the jump, execution of tricks, and artistic performance.
  - Street competition—tricks, grinds, and jumps on railings, curbs, stairs, ramps, and other obstacles found in typical urban settings
  - Free skating (also known as urban skating or free "riking")—The objective is to reach from one point to another by the fastest possible route, negotiating obstacles with jumps and slides as needed.
  - Off-road skating on dirt trails while on skates using special "all-terrain" wheels
  - These more extreme forms of inline skating increase potential for number and severity of injuries.
- There was a strong statistically significant downward trend in emergency department (ED)-treated inline skating injuries from 2005 to 2014 according to the Consumer Product Safety Commission (CPSC)'s National Electronic Injury Surveillance System (NEISS). Medically attended injuries (which includes ED, doctor's offices, clinics, and school nurses) in 2014 were estimated at 29,857 according to the CPSC.

### Injury Patterns

#### Risk Factors

- Male: ratio of male-to-female injured skaters is 1.5–2:1
- Age: 10–14-year-olds account for a majority of injuries
- Inexperienced skaters are at an increased risk of injury; 25% of skaters in a small study were injured their first time out
- Experienced skaters are also at an increased risk of injury related to performing skating tricks and dose-response skating (the more hours per week spent skating, the higher the number of injuries, specifically >10 hours/week).
- Skitching, a dangerous practice of skaters hanging on to the back of a moving vehicle, can lead to speeds of >70 mph.
- Deaths from inline skating usually involve collisions with motor vehicles.

#### Injuries

- Falls lead to numerous injuries. Most frequent site of initial impact after an unintentional fall was the hands and wrists (44.6%).
- Approximately 50% of head injuries in youth and adolescent sports/recreational activities occur while skateboarding, skating, and bicycle riding.
- 10%–25% of inline skaters have reported at least one injury.
- Severity: In a previous study, injury severity score (ISS) was 10.6 for inline skating compared with 10.5 for skateboarding and 12.7 for cycling.

#### Location

- Approximately two-thirds of injuries occur in the upper extremity. Wrist and forearm are the most frequent sites of injury, particularly more severe injuries (fractures and dislocations).
- Compared with other sports, skating sports have one of the highest frequencies of elbow dislocations.
- Knee and ankle are the most common sites for lower extremity injuries.
- Head injuries account for up to 5% of serious injuries. Approximately half of the inline fatalities in the Centers for Disease Control (CDC) database are from head injuries.

## Prevention

### Protective Equipment (Personal Protective equipment [PPE])

- American Academy of Pediatrics (AAP) and the American Academy of Orthopaedic Surgeons (AAOS) recommend helmets, wrist guards, elbow pads, and knee pads. American Dental Association (ADA) recommends mouth guards.
- Estimated that wrist guards and elbow pads can decrease upper extremity inline skating injuries by 90%
- Failure to wear wrist guards may increase the relative risk of injury by 10.4; nonuse of elbow pads may increase the relative risk of injury by 9.5.
- Cadaveric studies using high-speed impact loading suggest that wrist guards may reduce injuries.
- Helmets have been shown to reduce the risk of injury in bicyclists; this may be even more important for skaters who can reach speeds >40 mph.
- Fatally injured skaters universally seem to be nonhelmet wearers.
- Helmets
  - Should be certified by one of these groups: American National Standards Institute (ANSI), American Society for Testing & Materials (ASTM), CPSC, or Snell Memorial Foundation
  - Recreational skaters can use bicycle helmets. Aggressive or trick skaters should use skateboard helmets. For more information, visit the CPSC website.
  - Should be professionally inspected or replaced after any hard helmet impact

### Use of Protective Equipment

- Actual use: unknown; wrist guards are the most commonly worn personal protective equipment (PPE).
- Peer effect seemed to increase the use of PPE in observational studies.
- Adolescent skaters were much less likely to use PPE.
- Most influential factors for PPE use in adolescents: parents (40%), requirement of skating location (33%), friends (15%), and coaches (14%)
- Common reasons for not wearing protective equipment: lack of perceived need (47.3%) and discomfort (37.5%); appearance was also a factor (cited by 25% of adolescent skaters)
- Formal instruction in school-based program has been shown to have a small but significant positive effect in attitude toward and use of protective equipment.
- PPE has the potential to transmit forces to areas away from the impact site, placing other areas of the body at a risk of injury (e.g., wrist guards may place skaters at a risk of "splint-top" fractures).

OTHER

- Safe age for inline skating is not known. AAOS does not recommend skating (inline or skateboarding) under the age of 5, and 6–10-year-olds should be closely supervised.
- Skate parks: advantages are a smoother surface separate from traffic compared with roads; however, some have concern for increased injuries.
- Beginners should consider lessons; learning how to fall can help decrease injury.
- A small-scale study showed increased balance and strength in preteens in a 4-week instructional inline skating program.

# SKATEBOARDING

## History

- Modern-day skateboard is thought to be an adaptation of the scooterboard (wooden crate connected to a board and attached to rollerskate or other wheels) or soapbox. No definitive inventor is known.
- Skateboarding has experienced intermittent periods of popularity since the 1960s.
- There were an estimated 5.4 million skateboarders in 2014 (NSGA) and approximately 3,100 skate parks nationwide (according to Skate Park Association USA).

## Equipment

- The skateboard has three parts:
  - Deck: 7–9 inches wide; 28–33 inches in length; made of aluminum, bamboo, carbon fiber, fiberglass, Kevlar, plastic, or wood. Longer, inflexible decks are safer for beginners; shorter, more flexible decks lead to increased maneuverability. Wider decks are used for vert (vertical) skating. The longboard ("sidewalk surfer") is a variant of the skateboard with a longer deck, larger wheels, and a more stable truck. Boards in the past were often constructed in the shape of a surfboard without concavity.
  - Wheels: Usually polyurethane or plastic; wide wheels ("stokers") are 6–10 cm and provide more stability; narrower wheels ("slicks") are 3–4 cm and increase maneuverability
  - Truck: Made up of axles, frames, and hardware; they connect the wheels to the deck
- Modern skateboards usually have nose and tail kicks (upward curves) with a concavity between them, allowing cupping of the foot for more control.
- Stance
  - Regular (left foot forward) is most common
  - Goofy (right foot forward)
- Improved technology (polyurethane wheels) has led to increased speed and maneuverability as well as increasing the number of skating tricks.

## Competitions and Skating Styles

- Vert uses 8–10-foot high ramps (usually half-pipes); involves "big air" flare
- Slalom skating (weaving between cones) uses a technique called *pumping* that allows skaters to accelerate with every turn.
- Downhill: coasting or racing downhill on a longboard
- Cruising involves longboards but denotes riding from one place to another without focusing on tricks or speed.
- Freestyle emphasizes technical flat-ground skating, essentially "dancing" with a skateboard.
- Street skating is boarding on sidewalks, streets, and parking lots; may include tricks or skating on handrails, stairs, and lower ramps
- Pool skating (in a drained swimming pool) or park (as in skate park) is named by location.
- Off-road or dirt-boarding uses a large board and wheels with the skater's feet strapped in like snowboarding.
- New types of skateboarding continue to be developed.
- X Games have popularized competitive skateboarding.

## Injury Patterns

### Risk Factors

**Male:** CPSC reviewed 11 years of injury data and found approximately 87% of injured skaters were men.

**Age**: 10–14-year-olds are most often injured. Among CPSC age groups, 5–14-year-olds have the most injuries. Most severe head or neck injuries typically occur in skaters aged <5 years. However, recent literature questions increased injury severity in older skaters (aged >16 years).

**Experience and skate parks:** One in three injuries occurs in the first week (see inline section).

### Injuries

- Skateboard injuries increase with periods of popularity.
- Skateboard injuries were somewhat cyclical over the last decade according to NEISS data; there was an increase from 2005 to 2008, a decrease from 2008 to 2011, and an increase from 2011 to 2013. There were an estimated 299,286 medically attended skateboard injuries in 2014 (CPSC).
- Upper extremity injuries are most common.
- Approximately half of the injuries presenting to ED are fractures: forearm, wrist, and ankle most common.
- Ankle sprains are common.
- Head injuries account for almost 7% of injuries. 18% of hospitalizations from skateboarding are due to head injuries.
- Two-third to three-fourth of injuries are left sided owing to regular stance.
- A majority of deaths in skateboarding involve motor vehicle collisions.
- Mixed results on severity: certain studies suggest more severe injuries in skateboarding compared with other types of skating; see ISS data in the inline section.
- Skateboard elbow: olecranon process fracture caused by directly falling on one or both elbows. Elbow dislocations: see the inline section

## Prevention

- Use of PPE decreases the risk of injury (similar to inline section).
  - Injury less likely to require hospital admission if wearing PPE
  - Helmet use would have prevented all head injury admissions over a 30-month period.
- Recommended PPE
  - AAP and AAOS recommend helmets, wrist guards, elbow pads, and knee pads. ADA recommends mouth guards.
  - Helmet certification/type: see the inline section
- Actual use of PPE: unknown; earlier studies suggested 13%–33% of skateboarders of all ages wear PPE. Certain skate parks require PPE, hence, use may be increasing
  - Requirement of skating location, parents, and peers are the most common reasons adolescent skateboarders wear PPE; discomfort, lack of perceived need, and appearance were most commonly cited reasons by adolescents for nonuse of PPE.
  - Age: see the inline section
  - Formal instruction and PPE: see the inline section

# BICYCLE MOTOCROSS (BMX)

## History

- Started in California in the 1960s; around the time that motocross (motorcycles) became popular. For riders who wanted to participate in motocross but did not have the means, bicycle motocross (BMX) was an option. Riders dressed in full motocross gear and raced on tracks they made themselves.
- Scot Breithaupt, considered the founder of BMX, organized the first race in 1971; manufacturers began constructing bikes with 20-inch wheels specifically for this growing sport.
- Union Cycliste Internationale (UCI, or International Cycling Association) is the governing body for cycling. It also manages the classification for cycling disciplines, which includes BMX.
- Two sanctioning bodies of BMX racing in the US:
  - National Bicycle League (NBL), which is certified under the UCI
  - American Bicycle Association (ABA)
- International BMX Federation founded in 1981. In 1982, the first World Championships were held.
- BMX was introduced in the 2008 Olympic Games in Beijing.
- BMX is among the top 10 most popular extreme sports.
- The Outdoor Foundation considers biking (including BMX) a "gateway activity" to other outdoor activities; it is among the most popular outdoor activities.
- Approximately 70%, or 4 million, of the BMX bikers in the US are aged between 17 and 43 years.

## Competitions and Styles

### BMX Racing

- Track
  - Closed loop of earthen material 300–400 meters in length and 5–10 meters wide
  - Includes jumps, banked corners, and other obstacles
  - Electronically controlled starting gate
- Head-to-head competition of up to eight riders per heat (qualifying rounds, quarter finals, semifinals, and finals); the top four qualify for the next round
- Competition classes are based on age, gender, bike style, and level.
- Junior and elite levels
- Racers may reach speeds >30 mph.
- A majority of racers are men.
- ABA estimates over 370 BMX tracks currently in the US.

### Freestyle BMX

- Evolved from BMX racing when riders focused on aerial maneuvers and began to merge skateboard park riding with stunts and tricks.
- Events are timed and judged based on difficulty and originality; most well-known is the X-Games
- Riders usually participate in more than one discipline.
- The disciplines or styles may overlap to a certain extent, and as new styles of riding are developed, the current may change as with skateboarding.
- Disciplines:
  - Street: Riding on streets or public property; allows for creativity because almost anything can be used as an obstacle.
  - Park (or skate park) riding can differ based on whether the skate park is predominantly wood or concrete (latter lends itself to a faster, smoother style).
  - Vert: Analogous to vert in skateboarding, this discipline involves riders performing tricks on a vert ramp (half-pipe).
  - Dirt (or dirt jumping) involves lines of jumps built from compact dirt. Airborne riders often perform tricks before landing.
  - Flat (or flatland) riding terrain is smooth and flat as the name suggests. A majority of tricks involve spinning as well as balancing the rider and the bike in various positions.
- Competitions
  - May or may not be timed
  - Judged (based on discipline) on combinations of difficulty, originality, creativity, style, flow, numbers of maneuvers, and height

## Equipment

- BMX bikes usually have 20-inch wheels.
- Certain bikes are free-wheeling (wheels operate independent of pedal motion).
- Racing bikes
  - Standard: 20–22.5-inch wheels
  - Cruiser: 22.5–26-inch wheels
  - Typically, with a single brake in the rear
- Freestyle bikes
  - Street bikes are often the strongest and heaviest.
  - Dirt-jumping bikes are heavier than racing bikes but lighter than other freestyle bikes; may only have one rear brake

  - Flatland bikes have a shorter wheelbase with frames that are more reinforced.
  - Most freestyle bikes have front and rear brakes; the handlebar is designed to completely spin around without twisting the front brake cable; has axle pegs for tricks
- Pedals
  - Platform (flat) pedals without a cage are most often used in BMX; they offer grip when using short metal studs and do less damage if the rider is in an accident.
  - Toe clips generally not used

## Injuries

### Risk Factors

- Stunts/tricks
- Poor technique or mishandling of the bicycle
- Inexperience
- Male: >93% of injuries treated in EDs are male cyclists

### Types

- Most injuries are acute.
- In studies reporting BMX injuries seen at EDs, mild injuries were most common (e.g., abrasions and contusions), although 7% of injuries in one study were head injuries.
- Abdominal, scrotal/genitourinary, perineal, and spinal injuries are reported.
- Extremity injuries are most common, but facial injuries are also common (21%–22%).
- Comparison of BMX with conventional bicycle injuries shows conflicting results; overall, still limited data on BMX injuries.
- Racing injuries at the 1989 European BMX Championships: 61 injuries/976 participants; injury breakdown: 42% abrasions, 29% contusions, 8% sprains, 4% fractures (majority upper extremity), 2% concussion, and 2% other; injury rate: 1190 injuries per 1000 competition hours; higher rate of injury in females.
- 6.2% of athletes were injured at the 2007 World Championships.
- BMX was among the sports with the highest injury risk at the 2012 London Summer Olympic Games.

## Personal Protective Equipment

- Helmets
  - Full face or open face (latter must be used with mouth guards)
  - CPSC set standards for helmets after 1999 (see the CPSC website for BMX standards).
- In addition, long-sleeved shirts and pants with gloves and elbow pads required at UCI-sanctioned races
- Knee pads
- Closed-toe shoes
- Chest protectors
- Shin guards

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## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 97 MASS PARTICIPATION ENDURANCE EVENTS

William O. Roberts

## GENERAL PRINCIPLES

- This chapter shows the development of an algorithm for the management of mass participation endurance events.
- The medical director is the safety and health advocate for athletes who participate in the race.
- The safety of athletes is the primary purpose of race medical operations.
- A central medical command structure can improve the efficiency of the medical team, integrate community resources into the medical plan, and reduce response times.

### Events

- Road running
- Cycling
- Cross-country skiing
- Triathlon
- Wheelchair
- Swimming

### Approach as a "Planned Disaster"

- Mass participation events should be approached as a "planned disaster" (potential mass casualty incident), which has potential to adversely affect the community medical delivery system.
- Mass gatherings always have potential for medical illness or injury.
- Potential casualties can occur in two groups of people: participants (a literature review allows estimation of injury type and incidence; individual race experience allows for more accurate estimates) and spectators.
- Endurance events share common injury and illness risks that must be addressed by medical management teams, but each event will also have a unique injury and illness profile.
- Participant safety is the primary goal of the race committee and race medical committee.
- A comprehensive medical plan utilizing a central command structure will decrease the community medical burden and reduce the potential for emergency room overload.
- A central command structure can respond to unexpected race-related or race course incidents by drawing on community emergency medical, public safety, and law enforcement assets.

### Incidence and Risk

- Estimating medical encounters is best done with race data.
  - Anticipated number of starters multiplied by encounter incidence; a race with several years of start history will have an average "no show" rate for race registrants.
  - Project needs: staff, supplies, equipment
- Risk ranges
  - Running (56 km): 13% risk of injury over 4 years
  - Running (42 km): 0.5% to 20% risk of injury (as defined as requiring a medical encounter)
    - Twin Cities Marathon (Minnesota): 0.5% to 3% risk of injury (average 1.89% for entrants from 1983 to 1994)
    - Boston Marathon: 1.6% to 10% risk of injury (data from recent races only as the start time was moved from noon to 10 AM)
  - Running (≤21 km): 1% to 5% for the 11.5 km Falmouth Road Race (Massachusetts), less than 1% severe injury incidence rate; 0.54% for 21 km Two Ocean race (South Africa), 0.05% serious injury rate
  - Triathlon (225 km): 15% to 35% injury rate, 13%–21% injury rate among Kona Ironman participants from 1995 to 2014
  - Cross-county skiing (55 km): 5%
  - Triathlon (51 km): 2% to 5%
  - Cycling (variable): 5%
- Variables and unknowns: race day weather, event distance, event type, and condition (health and fitness) and acclimatization of participants
  - Influence of increasing heat and humidity on marathons:
    - Medical encounters increase and race times slow.
    - Race dropouts increase.
    - Increases in exertional heat stroke and exercise associated hyponatremia
  - Heat limits
    - Cancellation at 82°F wet bulb globe temperature (WBGT) is recommended by the American College of Sports Medicine (ACSM) guideline, but this is based on fit and acclimatized participants.
    - Twin Cities Marathon data imply cancelling at WBGT near 70°F may be better for nonelite runner safety and community emergency response load, especially for unacclimatized participants.
    - The cancellation level is likely event specific, but the number of medical encounters and nonfinishers seems to accelerate with WBGTs above 60°F. Elite runners seem to tolerate hotter conditions, and races may elect to run the elite race while cancelling the nonelite race.
    - A WBGT measurement on site is the best course of action, but if not available, WBGT can be calculated using a formula (available at http://www.zunis.org/hsa.htm) based on ambient temperature, relative humidity, time of day, and sky cover. Conversely, enter the ZIP code of the event, the time of day, the cloud conditions, and the sport at http://www.zunis2.org/.

### Anticipating Casualty Types

- Exercise-associated collapse (EAC) is most common: hyperthermic, normothermic, and hypothermic with normothermic exercise-associated postural hypotension most frequent
- Low-frequency but potentially fatal medical emergencies can occur, including cardiac arrest, exertional heat stroke, exercise-associated hyponatremia, asthma, insulin shock, anaphylaxis (exercise associated or "bee" sting), and high-velocity or impact trauma.
- Macrotrauma: musculoskeletal (fracture, dislocation, sprains and strains, contusions), vascular (closed, open), head and neck (concussion, intracerebral bleed, fracture–dislocation), and visceral organs (contusions, laceration, rupture)
- Microtrauma: tendinitis, stress fracture, fasciitis
- Dermatologic trauma: blisters, abrasions, lacerations
- Drowning, near drowning, and swimming-induced pulmonary edema can occur in water-based events.

### Race Medical Operations Purpose

Prerace: Develop strategies to improve competitor safety and reduce race-related injuries and illnesses.

Race day: **Primary:** stop progression of injury or illness; evaluate casualties (triage, treatment, transfer); reduce community medical burden. **Secondary:** prevent overloading of emergency medical services and emergency departments.

### Role in Race Operations

- Event and runner safety
- Medical decisions
- Medical spokesperson
- Executive committee administrative functions
- Coordinate medical aspects of transfer to "unified central command"

## PREVENTION STRATEGIES

### Primary

**Definition:** Prevention or reduction in the occurrence of casualties, reducing the severity of casualties

**Passive:** Cooperation of participants not required. Examples: start times, course modifications, traffic control

**Active:** Cooperation or self-initiated behavior change required. Examples: education, safety advisories

**Enforced active:** Helmets, wetsuits (required)

### Secondary

**Definition:** Early detection of injury or illness; intervention protocols to stop progression

**Examples:** Impaired runner policy; advanced cardiac life support (ACLS), advanced trauma life support (ATLS), or EAC protocol; on-course ambulance; finish line triage

### Tertiary

**Definition:** Treatment and rehabilitation of illness or injury

**Examples:** Emergency department transfer, hospital admission, rehabilitation center

## PREPARATION

### Race Scheduling

- Location (latitude, longitude, and altitude)
- Season of year
- Safest start and finish times (if average high temperature is >60°F, schedule race start for sunrise)
- Maximum time limits for competitors to remain on course

### Competitor Safety

- Consider the safety of the athlete first and foremost in all race-related decisions.
- Use the safest start and finish times for both elite and nonelite competitors.
- Determine hazardous conditions and develop a written race administration plan to simplify decisions on race day.
  - Ensure volunteer and competitor safety.
  - Define heat, cold, traction, wind, wind chill, lightning, and torrential rain race limits.
  - Alternatives: alter, postpone, cancellation
  - Publish protocol in advance.
  - Announce risks at start.
- Some local incidents, such as residential or commercial building fires, gas line explosions, train derailments, etc., may require cancellation of the race if the local public safety personnel are called upon to respond, leaving the race "unattended"
- Natural disasters and terrorist activities can also shut down a race (bomb detonation–Boston, Hurricane Sandy–New York).
- Impaired competitor policy:
  - Define an approach regarding an athlete who appears ill or injured during the competition, especially concerning fluid balance abnormalities and heat or cold stress.
  - No disqualification for medical evaluation. Most event rules allow medical assessment of athletes who appear ill without automatic disqualification and allow athletes deemed fit to continue participation as long as they leave and enter the course in the same spot and receive no intravenous fluid. This is especially important for citizen-class (nonelite) runners.
  - Criteria for continuation of event participation: oriented to person, place, and time; straight line progress toward the finish; good competitive posture; clinically fit appearance
  - Publish policy in advance.
- Emergency department (ED) notification: notify local EDs of date, time, and duration of event; also estimate numbers and types of possible race casualties.
- Preparticipation screening
  - Decide whether event should require pre-event medical screening: will it improve safety of participants? Will it be cost effective? Will it protect event and volunteer staff from liability?
  - Generally not recommended beyond usual health screening, as well as interventions by the participant's personal physician based on risk factors and symptoms.
- New data from of South African distance running races suggest that an online, automated, and individually targeted medical screening and educational intervention program for runners reduces the incidence of medical complications, specifically serious life-threatening cardiovascular complications, during a race. This screening and intervention program also included an acute prerace illness check with an educational intervention in symptomatic runners.
- Competitor education: safety measures, risks of participation, fitness level recommended for participation, hydration and overhydration (drink to thirst, knowledge of sweat rate, ingestion of adequate fluid to nearly replace sweat losses without excessive intake), volunteer identification (standard colors, visibility), nutrition.
  - Medical information should be registered with smart phone or computer apps designed for race medical care or placed on the back of race bibs, and should include training weight, prerace weight, allergies, medications, chronic medical problems, and emergency contact phone number.
  - Medical alert tags should be worn during the race.
- Child and adolescent participation in endurance events: there are no data to support the banning of participation of individuals under the age of 18 years for medical reasons, and children as young as 7 years have completed marathons without reported adverse effects. A motivated child (not parent), who is growing physically, physiologically, psychologically, and socially during training, should be allowed to participate if the race or event does not ban participation for administrative reasons.

### Course

- Course survey: hills; turns; immovable objects; traffic control; altitude changes; open water (participant safety and environment influences)
- Start: downhill starts increase risk for wheelchair competitors; wave starts should be employed to relieve congestion and risks associated with falls in mass starts

**Figure 97.1.** Finish area of mass participation events.

- Aid stations
  - Major: full medical care; equipped and staffed for most anticipated problems
  - Minor: comfort care, fluids, first aid, shelter
  - Location: start; every 15 to 20 minutes along course; finish line
  - Rolling aid: vehicle (bus or van) equipped and staffed to deliver medical care for expected injuries along the course; requires an open lane on the race course for the vehicle(s)
  - First-response teams: motorcycles, bikes, golf carts, or gators; 2–3 person teams; automatic defibrillator and first aid
- Finish area (Fig. 97.1)
  - Triage: chute, postchute, and area triage (sweep teams)
  - Field hospital: major aid station (see Fig. 97.1). Subdivisions may include triage, intensive medical, intensive trauma, minor medical, minor trauma, skin, and medical records.
  - Ambulance support for ED transfer (see Fig. 97.1)
  - Shelter for healthy finishers
  - Dry clothes shuttle; consider clothes dryer for wet or cold conditions.
  - Fan out finish line area to spread out participants in hot races.
  - Elevated spotters to identify downed participants in crowded areas (see Fig. 97.1)

## Transportation

- Healthy competitors who abandon the race
  - Prevent new or exacerbation of previous injury by transporting to shelter or reducing repetitive stress: hypothermia, hyponatremia, stress fracture, strain.
  - Examples: vans, buses, golf carts, gators, snowmobiles, snowcats, public transportation, sled, toboggan, boat
- Ill or injured competitors
  - Prevent progression of illness or injury (both overuse and acute injuries) without increasing individual morbidity or mortality.
  - Access care for more severe acute illness or injury. Minor injury can use transportation for healthy athletes who abandoned. Casualties requiring medical care need transport by ambulance to nearest ED or event medical station.
  - Examples of medically equipped vehicles for ill competitors who abandon: advanced (ALS) or basic (BLS) life support ambulance, life flight helicopter.
  - Finish area
- Access medical care in finish area.
  - Examples: wheelchair, litter, stretcher, manned carriers (Fig. 97.2)
  - Access tertiary care by ambulance: ALS, BLS

## Communications

- Type: phone (cellular/digital, hard wire), ham radio systems, two-way radio systems. Three-tiered system provides overlap and backup if a single system fails. It is common in disaster scenarios for cellular service to be shut down.
- Location: start; course (aid stations, pickup vans, course spotters, ambulance, race volunteers with cell phones forming a "line of sight" spotter network to blanket the course); finish area (field hospital [central dispatch for course], triage teams).
- 911 access: given to any volunteer to summon an ambulance, but must know exact location

**Figure 97.2.** Finish area should be equipped with wheelchairs, litters, stretchers, and manned carriers.

- Give runner number to ensure that the dispatched ambulance responds to the correct person. Notify race incident command central dispatch of pickup and disposition.
- Consider smart phone apps that can be used to access race communications and record participant medical encounters along the course and in the finish area.

## Fluids and Fuel

**Type:** Water, carbohydrate–electrolyte solutions, high carbohydrate foods or gels

**Location:** Start, aid stations, finish area (postchute area, medical area)

**Amount:** 6 to 12 ounces available for each competitor every 15 to 20 minutes. Double for start, finish, and transition areas. Total volume and course distribution can be estimated from similar events or past race needs. Competitors should know their individual needs based on sweat rate and most are safest if drinking to thirst.

**Publish in advance:** Fluid and food types, locations; risks and symptoms of under- and over-hydration; calculation of personal sweat rate (measure nude weight, run at race pace for an hour in expected race conditions, strip down, towel off, and reweigh nude; difference in weight is the maximum fluid volume to be replaced every hour).

## Equipment

- Shelter (tents, vehicles such as school buses, buildings) with heaters for cool weather
- Security fencing
- Cots (chaise lounges work well for leg elevation), chairs, tables
- Heating and cooling equipment (microwave to warm blankets; tubs for immersion cooling; coolers for ice)
- Generator
- Defibrillator (manual or automatic)
- Back boards
- Lights
- Portable sink
- Toilet
- Point of care lab measuring devices for serum sodium, blood urea nitrogen (BUN), potassium, hematocrit, glucose, oxygen saturation (if available)

## Supplies

- Medical
- Trauma
- Intravenous fluids (normal saline [NS] or 5% dextrose in NS)
- Medications (albuterol, epinephrine 1:1000, dextrose 50% in water, oxygen, midazolam, cardiac arrest drug kit, 3% NaCl solution, others)

## Staffing

- Personnel located at start, throughout course, and at finish
  - Physicians
  - Acute care nurses (intensive care unit, coronary care unit, ED)
  - Paramedics
  - Emergency medical technicians (EMT)
  - Physical therapists
  - Athletic trainers
  - First aid personnel
  - Nonmedical assistants
  - Sources for volunteers: hospitals, clinics, the American Red Cross, National Ski Patrol, National Mountain Bike Patrol, National Guard, Armed Forces Reserves, medical personnel training centers

## Medical and Race Records

- Document care
- Calculate incidence of casualties
- Project future needs
- Research injury/illness patterns
- Design a system to easily document care for common problems (eForm 97-1).

# MEDICAL PROTOCOLS

- **First aid:** Do no harm; stay within training level.
- **Basic problems:** Exercise-associated collapse/exercise-associated postural hypotension, low-frequency medical emergencies (cardiac arrest, exertional heat stroke, exercise-associated hyponatremia, anaphylaxis, insulin-related hypoglycemia), trauma, repetitive use, and skin injury
- **Initial assessment of collapsed athlete (ABCDE): A**irway (cervical spine control), **b**reathing, **c**irculation (hemorrhage control), **d**isability (neurologic status), **e**xposure and examination
- **Initial disposition:** Race medical facility, transport to emergency facility. Decide if on-course problems are triaged in race medical facility or moved directly to nearest ED. This will depend on both the medical capabilities and the accessibility of the race facility(s).
- **Treatment and transfer protocols**
  - Decide on level of care (first aid vs. medical treatment) in the finish area and course aid stations.
  - Determine in advance cases warranting automatic transfers (cardiac arrest, respiratory arrest, shock, symptomatic hyponatremia, severe trauma) and delayed transfers.
  - Keep treatment protocols simple.
  - Consider initiating treatment for exertional heat stroke and exercise-associated hyponatremia in the race medical facility for runner safety (avoid delays).
  - Integrate into emergency medical services (EMS) protocol.

- Medical precautions
  - Exposure to body fluids (blood, stool, emesis; *not* sweat) can transmit disease.
  - Modified universal precautions are most frequently used: handwashing, gloves, no food or eating in treatment areas.
  - Risks: hepatitis B, C, D, others; AIDS/HIV
  - Disposal of contaminated waste: Red Bag materials, sharps containers
- Adverse event protocol
  - If a participant has a medical event with an adverse outcome resulting in death or catastrophic injury, a predetermined protocol should be in place to communicate with the family, public, and media.
    - Medical event is first reported to the medical director and head of event administration.
    - Medical event should not be discussed with or by volunteers outside immediate need for medical care.
    - Medical director or a designated alternate should present the incident to media.
  - Event administration and medical director should keep detailed records.

## EXERCISE-ASSOCIATED COLLAPSE (EAC) CLASSIFICATION AND TREATMENT SYSTEM

- **Based on symptoms and signs of collapsed finishers**
  - Simple treatment protocols should be in place.
  - Weather influences injury patterns, and warmer weather increases the number of medical encounters and participant abandons.
  - Clinical classification system: varied presentation of symptoms within each temperature class; symptoms do not reflect body temperature; similar treatment for all classes; rapid recovery for most victims
- **Definition of EAC:** Requiring assistance during or after endurance activity; not orthopedic or dermatologic
- **Etiology:** Undetermined, but most cases are of exercise-related postural hypotension; hypotheses include sudden loss of muscle pump blood flow from legs (secondary heart function of legs during exercise), vasovagal response, depletion of energy stores, and central nervous system failure. Dehydration may be present in more severe cases but is not common.
- **Diagnosis:** Presence of signs or symptoms. Major criteria: body temperature, mental status, ambulation status
- **Clinical picture** (derived from clinical presentations of Twin Cities Marathon casualties)
  - Symptoms: exhaustion, fatigue, hot, cold, nausea, stomach cramps, lightheadedness, headache, leg cramps, and palpitations
  - Signs: abnormal body temperature, altered mental status, central nervous system changes, inability to walk unassisted, leg muscle spasms, tachycardia, vomiting, diarrhea, and unconsciousness
- **Classification scheme**
  - Types: **hyperthermic** (body temperature ≥103°F [39.5°C]), **normothermic** (temperature between 97°F [36°C] and 103°F; **hypothermic** (temperature ≤97°C)
  - Severity ratings: **mild** (presence of any symptom or sign, able to walk with or without assistance, alert, systolic blood pressure greater than 100 mmHg, heart rate less than 100 beats per minute, weight loss <5%), **moderate** (no oral intake, extra fluid loss, unable to walk, severe muscle spasm, weight loss 5% to 10%, temperature ≥105°F [40.5°C] or ≤95°F [36°C]), and **severe** (central nervous system changes, no oral intake, extra fluid loss, unable to walk, severe muscle spasm, weight loss 5% to 10%, temperature ≥106°F [41°C] or ≤90°F [32°C])

## Management Protocol

### Diagnosis and Documentation

- Initiate medical record; record presenting symptoms and medical history.
- Record vital signs.
  - Temperature: rectal measurement required for accurate core temperature estimate; tympanic membrane, temporal artery, oral, and axillary measurements (shell temperatures) are not accurate for core estimates in athletes and not recommended.
  - Blood pressure (BP), pulse, respiration
  - Orthostatic changes
- Record mental status and orientation, walking status, and other physical examination findings.
- Record treatment and log times.

### Fluid Replacement and Redistribution

- Supine position (nonambulatory): elevate legs and buttocks; restore pooled blood to circulation; if ambulatory and able, assist walking (Fig. 97.3)
- Oral fluids (preferred method): all mild cases; all moderate cases, if tolerated
- Intravenous (IV) fluids: all severe cases; moderate cases if no response to oral fluids or unable to tolerate oral fluids
- **Recommended fluids**
  - **Oral:** start with hypertonic fluids like bouillon broth, and then move to simple glucose–electrolyte drinks, fruit juices, or water
  - **IV:** 5% dextrose in NS for first liter if Na is normal or high, or if clinically dehydrated, then NS unless blood glucose is low (remember that lactated Ringer's solution contains potassium; avoid until potassium status known; do not use in hypothermic patient because a cold liver does not metabolize lactate)
- **IV access uses** (invasive procedure that should be used for set criteria)
  - Medication access
  - Measure serum electrolytes, BUN, glucose, and hematocrit.
  - Fluid replacement in participants with normal or high Na (most finishers are minimally to moderately dehydrated and improve rapidly with IV fluids when introduction of oral fluids fail)
  - Hypertonic (3%) NaCl solution for symptomatic hyponatremia (present in marathons usually due to inappropriate antidiuretic hormone levels in the face of excess fluid intake, resulting in fluid overload; in hot condition Ironman triathlons or ultramarathons, may be related to sweat Na loss and

**Figure 97.3.** Fluid replacement and redistribution after exercise-associated collapse. Supine position (nonambulatory)—elevate legs and buttocks; restore pooled blood to circulation.

dehydration (rare); initial treatment for symptomatic cases is the same).
  - Do not give IV fluids to any athlete who appears overhydrated, or has increasing confusion, severe progressive headache, or vomiting, without first checking Na levels.
- **Selection criteria for rapid IV fluid replacement** (based on data from the Twin Cities Marathon)
  - Administration should be considered after leg elevation for at least 10 minutes with no improvement (may take 20 to 30 minutes to resolve).
  - Systolic BP less than 100 mmHg (orthostastic BP drop after leg elevation)
  - Persistent heart rate greater than 100 beats per minute
  - Temperature above 104°F or below 95°F, if not responding to initial therapy
  - Severe muscle spasms
  - Anorexia, nausea, diarrhea
  - Hypoglycemia (<60 mg/dL)
  - High sodium and/or hematocrit and BUN
  - Confusion with normal or high sodium levels
  - Not doing well and hyponatremia ruled out
- Other options: no IV starts for fluid replacement, and send all who do not recover with simple first aid to ED (may potentially overwhelm EMS transport and ED centers)

## Temperature Correction

### HYPERTHERMIC EAC

- Move to cool or shaded area, remove excess clothing.
- Active cooling (rectal temperature >105°F): ice water tub immersion for fastest cooling rates; ice packs in neck, axilla, and groin combined with rapidly rotating ice water-soaked towels to the arms, legs, trunk, and head will give adequate cooling rate (Fig. 97.4)
- Control continued muscle contractions: shivering, muscle cramping, seizure (considerations include cardiac arrest, heat stroke, and hyponatremia). Following medications may be considered: midazolam (1 to 2 mg, slow IV push), repeat as needed (must be prepared to intubate and enforce no driving for 24 hours). Dantrolene may be considered in casualties who are resistant to cooling (ED administration) and is not generally recommended for field use.
- Cool aggressively until athlete "wakes up," then monitor temperature every 10 minutes to assess efficacy of treatment and to reduce overcooling. End active cooling at 100°F to 102°F. (Overcooling to 97°F probably not harmful)
- Precool IV fluids.

### HYPOTHERMIC EAC

- Move to warm area, remove wet clothing (use a clothes dryer in cold conditions).
- Dry skin and insulate with prewarmed blankets (use a clothes dryer or microwave).
- Use BairHugger or warm heater air to blow into blankets (see Fig. 97.4).
- Have patient breathe warmed, humidified air (Bennett or Bird respirator).
- Place warm packs (hot water bottles, warmed IV bags) in neck, axilla, and groin.
- Prewarm IV fluids, and consider IV dextrose 50% in water.
- Monitor temperature at regular intervals.
- Walk to generate intrinsic heat (if body temperature >95°F).

### NORMOTHERMIC EAC

- Maintain temperature; monitor temperature if not improving (postrace hypothermia, delayed hyperthermia [unlikely]).

### LEG CRAMPS

- Neuromuscular inhibition techniques, salty fluid and glucose replacement, assisted walking.

**Hyperthermic EAC.** Rapidly rotate ice water soaked towels to the arms, legs, trunk and head to give adequate cooling rate.

**Hypothermic EAC.** Use BairHugger™ or warm heater air to blow into blankets.

**Figure 97.4.** Temperature correction.

- Consider midazolam 1 to 2 mg IV push (be prepared to intubate and enforce no driving for 24 hours). Avoid massage until patient is well hydrated.
- Can be a sign of hyponatremia

## Fuel Supply

- Oral glucose solutions
- IV glucose solutions: dextrose 5% in stock IV NS solutions (no dextrose in water)
- Indications for usage of IV dextrose 50%: low blood glucose (measure with home glucose meter at toe and/or ear lobe), slow response to IV hydration, slow response to temperature correction, muscle cramping, severe EAC, cardiac arrest

## Transfer or Discharge

- Transfer to an emergency facility if patient does not respond to usual treatment, or if severe cases do not respond rapidly. Remember automatic transfer rules.
- Discharge clinically stable and normothermic patients with good cognitive function from race medical facility.
- Instruct in fluid and food replacement.
- Reevaluate if change in status.
- Recommend follow-up examination for severe cases (eForm 97-2)

## LOW-FREQUENCY MEDICAL EMERGENCIES

### Cardiac Arrest

- Equipment and supplies: automatic or manual defibrillators, intubation equipment and induction medications, ACLS drug kits (epinephrine, atropine, amiodarone, lidocaine, verapamil, sodium bicarbonate, morphine, magnesium sulfate, dextrose 50% in water [check for updated ACLS recommendations]), oxygen and delivery system, and IV kits
- Assume that seizure-like activity is due to cardiac arrest and apply automated external defibrillator (AED); if pulse and breathing present, check temperature; if normal, assume hyponatremia and check serum sodium
- ACLS standard protocol: ABCs (airway, breathing, circulation), defibrillation and cardiopulmonary resuscitation, cardiac monitor, IV access, intubation, medications
- Amiodarone stabilized cardiac rhythm in recent Twin Cities Marathon cardiac arrest case.
- In prolonged resuscitation of marathon or longer distance runners, consider placement of pressure transducers in lower leg compartments to detect elevated compartment pressures.
- Sudden cardiac arrest (SCA) and sudden cardiac death (SCD) in marathon road racing:
  - Twin Cities Marathon and Marine Corps Marathon combined database 1982–2014: 2.45 SCA per 100,000 finishers and 1.14 SCD per 100,000 finishers
    - US marathons from 2000 to 2010: 1.01 SCA per 100,000 finishers and 0.63 SCD per 100,000 finishers
    - Successful resuscitation for race-related SCA: Twin Cities Marathon, 5 of 7; Twin Cities 10 Mile, 2 of 2; London Marathon, 6 of 11 (through 2006); Marine Corps Marathon, 3 of 8. (In major US metropolitan areas, nonrunners with out-of-hospital SCA have a 1 in 20 survival rate.)
- Road racing by trained individuals is safer than a sedentary lifestyle.
- Preventable
  - Can cardiac screening reduce deaths? Cost is a problem.
  - Close attention to symptoms and risk factors with appropriate screening
- Emerging data from South Africa suggest that integrating cardiac risk questions into the registration process with suggestions to be evaluated may reduce race-related SCA cases.

### Cardiac Chest Pain (Acute Coronary Syndrome)

- Activate emergency response system
- Give 325 mg aspirin (chew)
- Start high flow oxygen if available
- Attach AED if available
- Start IV line if available
- Transport to nearest cardiac center

### Exercise-Associated Hyponatremia

**Etiology:** Water excess and dilution due to overhydration combined with inhibition of renal water clearance (inappropriate ADH) is the most common cause; in very long events such as the Ironman distance triathlons, athletes with high sweat sodium concentrations may develop sodium depletion hyponatremia with dehydration (rare).

**Incidence:** Low in events lasting shorter than 4 hours; more common in ultramarathon distances and long-distance triathlons; five deaths in marathons since 1998 and reports are increasing in slower participants (3 symptomatic runners in 210,000 finishers at Twin Cities Marathon, but 12%–13% of Boston and London Marathon cohorts [asymptomatic]).

**Significance:** Potential for fatal outcome is characterized by rapid deterioration, progression to seizure, respiratory distress, and coma because of worsening cerebral and noncardiac pulmonary edema; reason for transfer to ED. May present at home, in hotel, or in transit (subway, train, airplane, automobile) several hours postrace

**History:** High fluid (sports drink or water) intake of 1 to 2 glasses at every aid station or lack of weight loss during event in dilutional hyponatremia; severe pounding headache that progressively worsens over time; feeling of "impending doom" or feeling scared; puffiness in extremities; muscle cramping; sleepiness; nausea and vomiting; confusion; slow times; small body habitus; inconsistent reports of nonsteroidal antiinflammatory drug (NSAID) use

**Physical findings**: Ashen skin (including lips), tight rings, watches, or shoes; vomiting; normal pulse, BP, and respiration (early); intact mental status (early); confusion, check for muscle spasms, tetany, clonus, seizure

**Lab tests (if available):** Sodium (<135 mmol/L); low hematocrit and BUN from fluid overload. Average sodium in fatal cases is between 121 and 122 mmol/L. It is desirable to have point-of-care sodium, BUN, and hematocrit measurements available in the medical area.

**Treatment on site:** If minimal symptoms, may treat on site with observation until urination or administration of hypertonic fluid (four bouillon cubes dissolved in 4 ounces water) and observation until urinating freely. If symptomatic and deteriorating mental or respiratory status, start high flow oxygen supplementation and give 100 mL 3% NaCl IV push (can repeat for two additional boluses or more if not responding) and continue hypertonic saline at 50 to 70 mL per hour during transfer to ED. The initial treatment for either water dilution or hyponatremia is hypertonic saline. Patients with sodium levels of <135, clinical signs of dehydration, and normal to high BUN/hematocrit levels may be treated on site with IV NS. Have midazolam and/or magnesium sulfate ready if clonus or seizure develops.

**Disposition:** Transfer runner with this clinical constellation to an emergency facility if no point-of-care lab tests are available. Start IV line for medication access but do not assume dehydration and do not administer hypotonic or isotonic fluids. Alert ED of transfer and expected treatment and complications (especially CT or MR imaging delays). For athletes with improving clinical status and lab values who begin to urinate freely, onsite observation with discharge in the company of responsible family or friends is permissible.

#### Other

**Anaphylaxis:** Types (atopic, exercise-induced) and treatment (epinephrine, antihistamine)

**Asthma:** Inhalers (albuterol metered-dose inhaler with extender), oxygen high flow, nebulizer (albuterol), subcutaneous terbutaline or epinephrine

**Insulin shock:** Dextrose 50% in water, glucagon

## TRAUMA

- **High-velocity activity collisions and falls:** biking, skiing, wheelchair racing
- **Vehicle on course (pedestrian or bicycle–vehicle collision)**
- **ATLS protocol**
  - Equipment: back boards, neck collars (semirigid), splints, cricothyroidotomy kit, oxygen, IV fluids
  - Primary survey: airway and cervical spine control; breathing and ventilation; circulation and bleeding control; disability or neurologic status; exposure and examination
  - Initial resuscitation: high-flow oxygen, shock management (fluid therapy, position, shock trousers), cardiac monitor
  - Secondary survey: look for other problems
  - Definitive field care: temperature maintenance, pain control, splint

- Transport to emergency medical facility (in cases of major trauma) or race medical facility, if equipped and staffed to care for minor trauma.

## POSTRACE REVIEW

- **What went right** (and can it be improved)?
- **What went wrong** (and why)?
- **Proposed changes** to improve safety and care of athletes

## BUDGET FOR MEDICAL OPERATION COSTS

- Race or event should develop budgets that include medical equipment and supplies.
- Equipment and supplies can be purchased, donated, rented, or borrowed; cost of high-tech equipment makes it difficult to purchase or borrow.
- Volunteer time is usually donated, but the race should provide a T-shirt, poster, pin, or similar reward for personnel.
- Ambulance service should be enlisted to help with transport of athletes off course and from finish area to local hospitals.

## eBOOK SUPPLEMENTS

Visit www.ExpertConsult.com for the following:

- eForm 97-1 Medical Record Form
- eForm 97-2 Medical Discharge/ER Transfer Form

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## eBOOK SUPPLEMENTS

- eForm 97-1 Medical Record Form (used with permission from William O. Roberts, MD, MS, FACSM)
- eForm 27-2 Medical Discharge/ER Transfer Form (used with permission from William O. Roberts, MD, MS, FACSM)

**MEDICAL RECORD – CONFIDENTIAL**

**Arrival time:** _____
**RACE:** (circle) Marathon / 10 Mile / 10K / 5k **Location:** Medical Tent / Aid Station Mile_____ **Finish time:** _____/ **DNF**
**Race #** _____ **Name** _____ **Age** _____ **Sex** M / F
**Pre-Event injury/illness:** Y / N Describe _____ # Previous Marathons _____ Best Time _____
**Medical History:**
**Symptoms:** Exhaustion Fatigue Lightheaded Hot Cold Syncope Weak Headache
**Resp symptoms:** SOB Wheeze Cough
**Cardiac symptoms:** Chest pain Tachycardia Palpitations
**GI status:** Nausea Vomiting Diarrhea Stomach cramps
**Muscle, Skin, Bones, and Joints**
**Complaint:** Muscle cramps Pain Blister Abrasion Bleeding Swelling
**Location:** Toe R / L Foot R / L Ankle R / L Calf R / L Knee R / L Thigh R / L Hip R / L Back R / L (upper/lower)
**Mobility:** Independent With assistance Wheelchair
**Orientation:** Person Place Time
**Mental Status:** Alert Confused Unresponsive
**Skin:** Hot Cold Sweaty Dry
**Admission Pain Level:** 0 1 2 3 4 5 6 7 8 9 10
**Tissue:** Skin Muscle Tendon Ligament Bone
**Details or Other:** _____

| Time | Rectal Temp | BP | Pulse (reg / irr) | $O_2$ Sat (RA) | Glucose Check | Meds/Rx ($D_{50}W$, Zofran, Benzodiapine) |
|---|---|---|---|---|---|---|
| | | | | | | |
| | | | | | | |
| | | | | | | |
| | | | | | | |

**Lab: $O_2$ Sat** (ra) ____% **$Na^+$** ____ **$K^+$**____ **Hct** ____ **BUN** ____ [#2 (if indicated) **$O_2$ Sat** (ra) ____% **$Na^+$** ____ **$K^+$**____ **Hct** ____ **BUN** ____]
**Treatment:**
**Treatment Refused**
**Leg elevation**
**PO Fluids:** _____ **IV Fluids: IV #1** 1L $D_5NS$_____ **IV #2** 1L $D_5NS$ or NS_____ **IV #3** 1L $D_5NS$ or NS_____
time time time
**Musculoskeletal:** Ice pack / Compression / Stretching / Joint Movement Facilitation / Prolonged Stretch / Assisted Walking / Other
**Physical Therapy Rx:** yes / no
**Skin:** Prep / Lance / Bacitracin / Dressing
**Discharge Pain Level:** 0 1 2 3 4 5 6 7 8 9 10

**Discharge status:** Home / ER transfer (ER Follow-up: Admit / Home)
**Discharge Mobility:** Independent With assistance Wheelchair
**D/C instruction sheet:** Blister / Fluids
**Notes: (Continue on opposite side)**

**Diagnosis:** Blister Abrasion Sprain Tendinitis Strain Fracture (suspected)
EAC/EAPH: Hyperthermic:Normothermic:Hypothermic – mild / mod / severe
Exercise Associated Muscle Cramps / Heat stroke / Exertional Hyponatremia
Other _____

**Signature:**_____**MD / DO / DPM / PA / NP / RN / EMT / ATC**
**Discharge time** _____
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## MEDICAL DISCHARGE / ER TRANSFER FORM

**Name:** _____ **Race #:** _____

**Race:** Marathon / 10 Mile **Location:** Finish Line Medical Tent / Aid Station Mile_____

**Diagnosis**

Exertional Heat Stroke — Exercise Associated Collapse – Severe

Exertional Hyponatremia – Dilutional — Fracture (suspected stress fracture)

Exercise Associated Muscle Cramps — Other:

**Discharge Vital signs: BP** _____/_____ **Pulse** _____ **$O_2$ Sat** (ra) _____%

**Labs:** **$Na^+$** _____ **$K^+$**_____

**Hct** _____ **BUN** _____

| | |
|---|---|
| **Treatment:** | |
| **Body cooling:** | Initial rectal temperature: _____ Discharge rectal temperature: _____ |
| **IV Fluids:** | ____ liter(s) $D_5NS$ ____ liter(s) NS Medication Access Only |
| **$D_{50}W$ (50 ml):** | #1_____ #2_____ |
| **Medications:** | Midazolam (Versed): 1mg 2mg 3mg Time _____<br>Ondansetron (Zofran: 4 mg X _____ IV Time _____<br>Other: (i.e. asa, acetomenophin) _____ Time _____ |

**Discharge status:** Home / ER transfer **Discharge time:** _____

**Discharge instructions:**

- ❑ You cannot drive a vehicle for 24 hours.
- ❑ You should eat your normal foods and drink extra fluids to keep your urine pale yellow in color.
- ❑ You should not drink extra fluids until you are urinating regularly and your weight is back to training level. You should eat your normal foods.
- ❑ You should have your liver, kidney, and CK muscle chemistry tests checked by your physician tomorrow to monitor you for changes from exertional heat stroke.
- ❑ You should be evaluated in the emergency room or by your primary physician if you feel worse today or tomorrow, you do not get better, or you have questions about your health.

**Notes:**

**Signature:**_____**MD / DO**
**ER or physician contact:**

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# 98 ULTRAENDURANCE AND ADVENTURE RACING AND EVENTS

Chad Asplund • George Wortley

## INTRODUCTION

- Ultraendurance and adventure races are prolonged events, usually longer than 6 hours (and some may be as long as 10 days) that usually take place in remote, austere, harsh, or extreme environments.
- These events include challenging terrain, extreme elevation changes, weather challenges, and on-course obstacles.
- Adventure races require participants to perform multiple disciplines that may include trail running, hiking, mountaineering, mountain biking, boating/rafting, climbing, caving, and/or orienteering.

## EPIDEMIOLOGY/PARTICIPATION

- More than 100,000 ultramarathoners compete in more than a thousand races held annually worldwide.
- Adventure racing participation has increased by 211% in the last 5 years (1.3–2.2 million participants in 2013).

## GENERAL PRINCIPLES

### Terminology/Event Types

- Adventure races
  - Sprint (2–6-hour races): 4–8 miles on foot, 15–25 miles biking, and 2–4 miles paddling
  - Endurance (12 hour races): 6–12 miles paddling, 8–14 miles trekking, and 25–50 miles biking
  - 24-hour races: 10–25 miles paddling, 10–25 miles trekking, and 50 miles biking
  - Expedition: 400+ miles of varied disciplines
- Ultraendurance races
  - Any running race longer than the standard marathon distance
  - Most common distances are 50 km, 100 km, 50 miles, and 100 miles.
  - 100 km is recognized as an official world record distance by the IAAF.
  - Other distances include double marathons, 24-hour races, and multiday races of 1,000 miles or longer.
  - Many events with challenges such as trails, variable terrain, altitude, weather, and variable aid and support

### Equipment

- **Discipline-specific** (e.g., shoes, clothing, pack and tools, lights, bike, kayak, paddle, rope harnesses, compass)
- Participants must carry/provide their own food, water, protective clothing/footwear.
- Hydration system
- Water purification devices (e.g., iodine tablets, filter system, ultraviolet pen)
- Compass (GPS is usually prohibited), maps, and race directions
- First aid kit and personal medications
- Boxed food, water, clothing, other apparel for resupply points
- Additional information can be found at http://www.usaranationals.com/gearlist.aspx.

### Specific Training

- Need to think about physical, mental, and skills training
- Master the logistics of fluids, nutrition, use of equipment, and rest.
- Plan multidiscipline, multihour sessions so that athletes know how their bodies will react and feel after extended exertions in different disciplines (Table 98.1).

### Nutritional Issues

#### General Guideline

- Aim for a healthy weight; avoid rapid weight loss.
- Choose foods sensibly and consume them during training to ensure body tolerance on race day.
- 60/20/20 split common (aim for 60% of calories from carbohydrates, 20% from protein, and 20% from fat)

#### Pre-Event

- Carbohydrate loading: load muscles and liver with glycogen (stored carbohydrates) during the week before an event
- Consume a normal intake of carbohydrates (5–7 grams per kilogram of body weight) during the first 3 days of the taper week.
- Increase this amount to 10 grams of carbohydrate per kilogram of body weight for the next 3 days.
- Eat the last big, high carbohydrate meal two nights before the race.
- Do not overeat night before race! Avoid items high in fiber or in fat, which may be hard to digest and may lead to GI distress during the event.

#### During Event

- General carbohydrate needs vary but often range between 30 and 60 grams per hour or approximately 1/4 to 1/3 of body weight in pounds per hour
- Some athletes also utilize small amounts of protein during events, up to 5 grams per hour.
- Athletes may need up to 300 calories per hour, and it may be difficult to digest more with the demands of exercise.
- Athletes must realize that the rate of loss will exceed rate of assimilation by approximately 3-fold during endurance events.
- Many athletes use sports bars, drinks, and gels to replace calories on event day. Make sure to test tolerance in advance.

#### Postevent

- Important to replace calories, especially glucose, within the first few hours after training sessions lasting >90–120 minutes to ensure liver and body stores are replenished adequately before future training sessions

#### Train Low, Race High

- Some athletes train with low carbohydrate reserves/supplies to train the body to increase oxidative enzymes and utilize fat stores and to spare glycogen use for longer periods of time (fat adaption).

**TABLE 98.1 GUIDELINES FOR ADVENTURE RACES**

| Race Distance | Total Training Hours | Bicycling | Running | Paddling |
|---|---|---|---|---|
| Sprint | 5–10 | 2–3 | 2–3 | 1–2 |
| Endurance | 10–20 | 4–5 | 4–5 | 2–4 |
| Expedition | 20+ | 5–7 | 5–7 | 4–6 |

- These athletes then race while ingesting carbohydrates so that their bodies will burn the ingested carbohydrates but will be able to rely on fat stores for energy when the carbohydrate supply decreases.

### Fluids

- Drink to thirst
- Train with the fluids you will use during the event.
- Aim for no more than 2% weight loss during training/event; avoid weight gain, as too much fluid may lead to hyponatremia.
- If mixing calories in fluids, make sure to avoid >6% sugar solutions, because they can interfere with fluid absorption and cause GI distress.
- Utilizing different types of sugars (e.g., glucose, fructose, maltodextrin) may be helpful.
- Maltodextrin is a complex carbohydrate that may be mixed at high percentages (e.g. up to 18%) without exceeding system osmolality, and it may cause less GI distress than other sugar types.

### Electrolytes

- Mainly used by athletes for the prevention of exercise-associated muscle cramping
- Larger athletes, faster athletes, and those who have high volumes or high concentrations of sodium in their sweat (white salt deposits on chin straps, headband, clothes, skin, etc.) may need to ingest additional sodium beyond what can be found in sports drinks, but the best method to ingest is in fluid or food form.
- Some athletes use salt tablets but can easily ingest too much; GI upset and cramping are common.
- Need to remember that muscle cramping is also frequently a result of exercise-associated muscle fatigue, excitability, dehydration, and heat illness

### Practical Issues

- Must experiment with a variety of different foods/nutritional items during training; essential to have a basic hydration and nutrition plan before entering competition
- The amount of calories tolerated per hour is inversely related to the intensity of effort (harder the effort, fewer calories tolerated).
- Should assume that calorie and fluid deficits are inevitable even in ideal circumstances; must plan to minimize this as much as possible
- In multiday events, the goal is to return to a state of euhydration prior to resuming competition the next day.
- Urine should be "copious and clear" prior to resuming competition.

## Environmental Issues

- Remember that the variables listed below are often a greater threat in ultraendurance athletes due to sustained exposure.
- Heat (see Chapter 21: Exercise in the Heat and Heat Illness)
  - The most serious temperature-related illnesses are heat stroke and hyperthermia.
  - Heat acclimatization is an important adaptation but is unrealistic for many athletes who may live in variable climates; takes approximately 10–14 days training in heat to acclimate, loss of acclimatization is rapid once heat exposure is removed.
- Cold (see Chapter 22: Exercise in the Cold and Cold Injuries)
  - Hypothermia and frostbite are the most serious cold–related illnesses.
  - Need to factor in wind chill (especially when on a bike) and body heat loss in water, which is constantly displaced from body in swimmer (wetsuit helps).
- Altitude
  - Altitude illness may occur above 2500 m; see Chapter 16: The Wilderness Athlete and Adventurer
  - Acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE) may all occur.
- Lightning
  - 54 running-related lightning deaths per year
  - Strike types:
    - Direct strikes: lightning directly strikes the athlete
    - Ground strike: travels through ground and may be conducted through an object (body) nearby
    - Side flash: lightning strikes a taller object and part of the current jumps to the victim
  - If feasible, suspend event start if thunder or lightning is in the area ("if you can hear it, clear it").
  - Athletes should seek shelter if thunder heard or lightning seen while on course.
  - Avoid ridgelines or summits, as well as tall objects such as ski lifts, cell phone towers, or isolated trees.
  - If lightning strike is imminent, assume lightning position:
    - Sitting or crouching with knees and feet close together to create only one point of contact with the ground; get as low as possible.

## COMMON INJURIES AND MEDICAL PROBLEMS

## Soft Tissue/Dermatologic Issues

- The most commonly encountered complaint in ultramarathon running and one of the top reasons that adventure athletes seek medical care

### Blisters

**Description:** Most common soft tissue injury in ultraendurance or adventure athletes (Fig. 98.1). Caused by repetitive friction between the skin and another surface

**History:** Compose 20% to 40% of injuries occurring during same day ultramarathons; 35% to 75% of injuries occurring during multiday ultramarathons; most likely to occur during the middle of multistage races as this is the timeframe when accumulated friction and skin wetness reach critical levels to create the blister

**Physical examination:** Common locations: distal aspects of toes, balls of the feet, and posterior heel. Areas of redness and chafing are precursors of blister formation and invite prevention.

**Risk factors:** Heat, moisture, ill-fitting shoes, anatomic abnormalities (e.g., calcaneal exostosis, accessory navicular, bunion), and increased running distance

**Treatment:** Painful nonbloody blisters can be drained in a sterile environment, taking care to preserve the overlying skin. A blister with murky or hazy fluid may be infected and should be opened (deroofed) and irrigated with the appropriate cleanser (e.g., saline with iodine, hibliclens, etc.); the cavity should be then treated with bacitracin or another non-neosporin antibiotic ointment before covering.

**Prevention:**

- Decreasing shear force of friction on the skin
- Concept of a prevention layer at high friction spot – may use tape, second skin, tegaderm, Compeed products

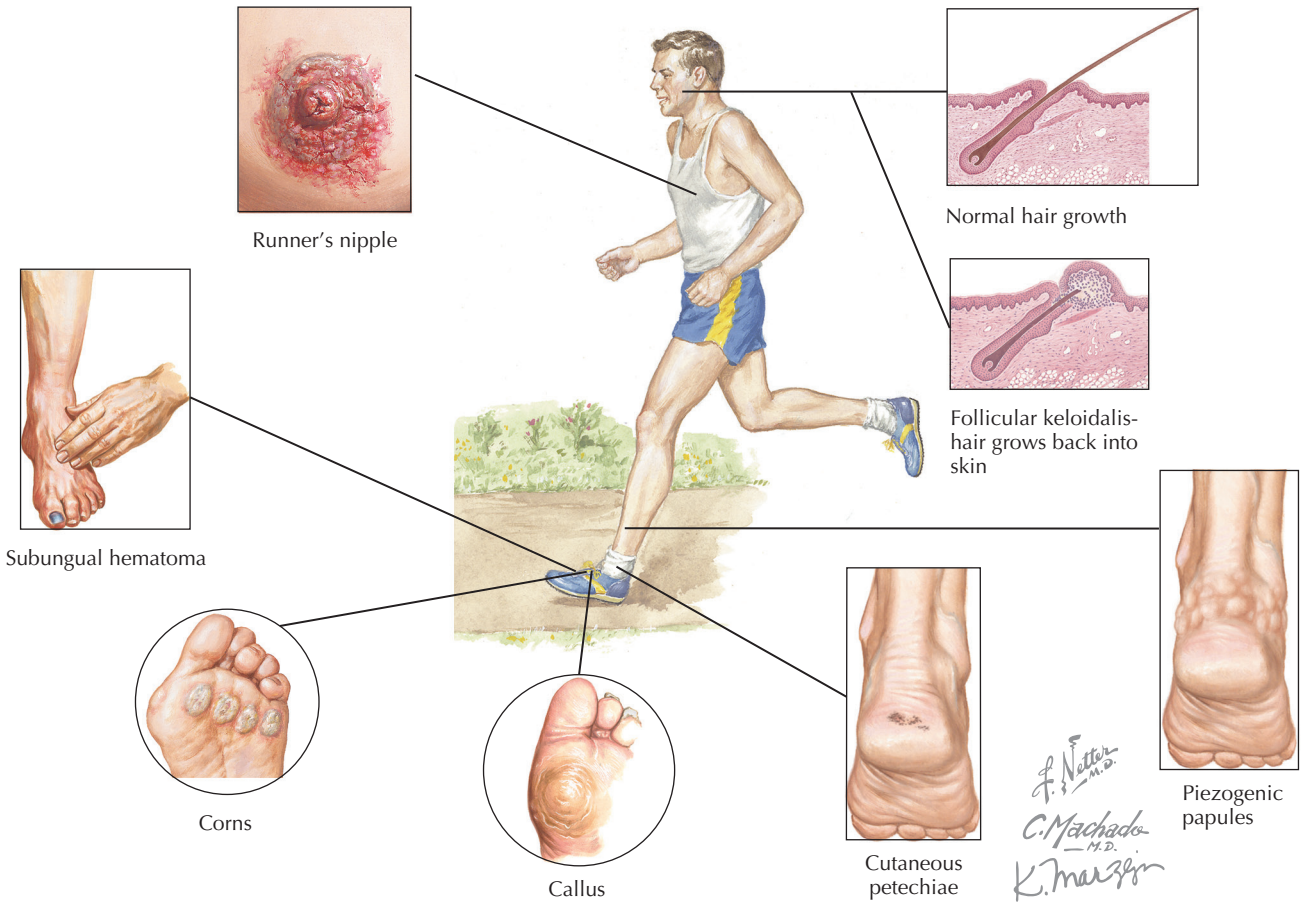


**Figure 98.1.** Common problems caused by friction and pressure.

- Moisture-wicking synthetic socks; ensure feet are dry
- Lubricants, while theoretically helpful, may paradoxically increase the rate of friction for long duration events by trapping moisture and grit.
- *Educating participants and encouraging them to carry their own blister care kit will greatly minimize the burden to the medical staff.*

### Subungual Hematoma

- Accumulation of blood underneath the nail bed
- Caused by repetitive contact between the longest toe and the toebox of the shoe, especially during predominantly downhill courses
- Occurs in 3% to 10% of participants in ultraendurance events
- If painful, may be drained by piercing the nail with a clean wide-bore hypodermic needle or heated paper clip under sterile conditions
- Monitor closely for infection
- May be prevented by trimming toenails short prior to event

### Chafing

- Superficial inflammatory dermatitis due to skin rubbing on skin or clothing
- Affects 9% of runners
- Mainly involving thighs, groin, back, axilla, and nipple areas
- Can be prevented by lubrication (e.g., Aquaphor), chamois cream, or taping
- May be treated with topical corticosteroid cream to reduce inflammation
- May be prevented by wearing dry, breathable, well-fitting clothing

### Abrasions

- Damage to superficial layers of the skin usually caused by a fall
- Large abrasions may cause pain secondary to exposed nerve endings.
- Clean with soap and water (or just water if that is all that is available).
- If available, a semipermeable and/or hydrocolloid dressing such as Tegaderm, Duoderm, Bioclusive, or second skin may be used.

## Respiratory Issues

- Respiratory illnesses, including upper respiratory infection (URI), infection, bronchitis, and reactive airway disease, are a common reason for athlete withdrawal from adventure races.
- Heavy exercise, especially with inadequate recovery, may induce immune suppression and can increase susceptibility to URIs and other respiratory infections. J-shaped curve: average risk of infection with moderate physical activity, increased risk of infection as intensity of exercise increases (Fig. 98.2).
- Endurance athletes are known to have a higher incidence of asthma and allergies than the general community.

### Upper/Lower Respiratory Infection

- Majority are caused by viral pathogens and lead to inflammation of the respiratory tract.

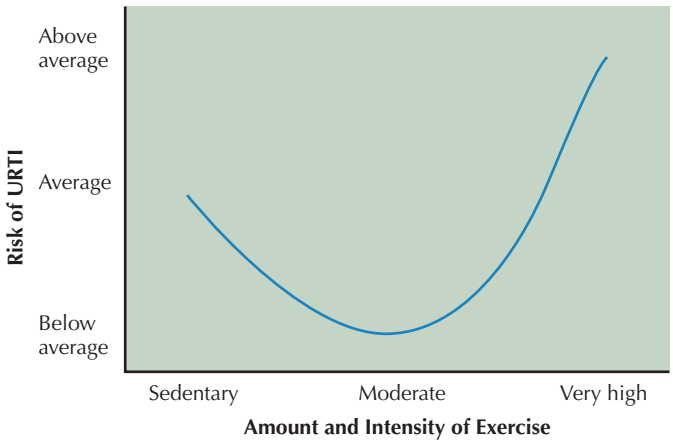


**Figure 98.2.** "J"-shaped model of relationship between varying amounts of exercise and risk of URTI. This model suggests that moderate exercise may lower risk of respiratory infection while excessive amounts may increase the risk. *URTI,* Upper respiratory tract infection. (From Nieman DC. Exercise, infection, and immunity. *Int J Sports Med*. 1994;15[Suppl 3]:S131-S141.)

- Hold febrile athletes out of competition due to risk of myocarditis.

### Reactive Airway Disease/Asthma/Exercise-Induced Bronchospasm (EIB)

- EIB is more common among ultramarathon runners than other athletes, although the exact reason is unclear.
- Predisposing factors include dust, allergens, cold weather, prolonged event duration, and high altitude.
- Runners with lower respiratory symptoms (shortness of breath and wheezing) should be evaluated for HAPE when participating at high altitude and/or exercise-associated hyponatremia (EAH).
- Concurrent URI may exacerbate or cause EIB and may increase the risk for HAPE.
- Spacer-delivered β2 agonists provide effective and portable treatment for asthma and may reduce pulmonary arterial pressure with HAPE.
- Athletes who can identify exercise/event triggers should consider pretreatment with β2 agonists.
- Long-acting β2 agonists may be more appropriate for pretreatment events lasting >5–6 hours.
- Asthma may actually improve at high altitude secondary to a reduction in pollen and pollutants at higher altitude.
- *Any runner requiring supplemental oxygen to maintain oxygen saturation should not be allowed to participate in/continue event.*

## Gastrointestinal Issues

- Gastrointestinal distress, including nausea, vomiting, diarrhea and gastroesophageal reflux disease (GERD), is prevalent among ultramarathon competitors.
- Vomiting is another common issue causing withdrawal from races and impacting race performance.
- Adherence to a diet low in fat and fiber prior to and during competition will reduce incidence of GI distress.

### Upper GI Tract

- Nausea and vomiting may be due to exercise-induced gastroparesis, overdrinking, or drinking hyperosmolar fluids, resulting in unabsorbed fluid in the upper GI tract.
- *Nausea may be an early sign of exercise-associated hyponatremia, altitude illness, dehydration, or heat illness, and should be further evaluated before returning the runner to competition.*
- Use of nonsteroidal anti-inflammatory drugs (NSAIDs) during the race increases risk of upper GI distress.
- Infective causes of nausea and vomiting are more likely to occur in multiday races and can spread quickly from person to person through unwashed hands, close contact with infected individuals, consumption of spoiled food, contaminated water, or unclean eating utensils.
- Proper hydration with appropriate fluids and electrolytes is essential.
- Oral ondansetron (dissolvable tabs) can effectively reduce nausea and vomiting.
- Some athletes may benefit from pre-event proton pump inhibitors.

### Lower GI Tract

- Dehydration and increased intake of simple sugars have been implicated in lower GI distress.
- Other risk factors include the female sex, younger age, history of abdominal surgery, history of irritable bowel syndrome, lactose intolerance, and dehydration.
- Transient lower gastrointestinal bleeding is common in endurance athletes but is usually self-limited.

## Cardiac Issues

### Athlete's Heart—Normal Adaptations

- Demands of endurance sport training lead to physiologic changes to cardiac structure in endurance athletes.
- Right ventricular (RV) and left ventricular (LV) hypertrophy are common.
- Left atrial enlargement may also occur.

### Proposed Mechanism of Cardiac Maladaptation

- Studies show that cardiac function (LV ejection fraction) is reduced and troponin release increases following prolonged endurance activity, which may be indicative of myocardial damage. These changes typically resolve within 72 hours.
- Prolonged exercise stress results in right ventricular (RV) strain, right atrial and right ventricular dilation, as well as diastolic dysfunction.
- Persistent, intense efforts without adequate rest to facilitate heart recovery, as well as long-term participation may lead to the development of scarring or fibrosis within the cardiac muscle.
- Patchy areas of fibrosis may increase risk for arrhythmias (atrial and ventricular) as well as sudden cardiac death (SCD).

### Cardiac Biomarkers

- Troponin I is elevated following prolonged endurance racing.
- Coronary calcium score is higher in endurance athletes compared to age-matched norms.

### Arrythmias

- Bradyarrythmias are very common and are typically benign in ultraendurance athletes if asymptomatic; sinus bradycardia, junctional bradycardia, first-degree AV block, and Mobitz type 1 block are common.
- Premature ventricular or atrial beats are common and typically benign.
- Tachyarrythmias, including atrial fibrillation (AF), may occur and may be more problematic in endurance athletes.
- Studies show veteran endurance athletes are at a 5-fold greater risk for atrial fibrillation.
- Majority of arrhythmias in endurance athletes originate in the right ventricle (possible exercise-induced arrythmogenic right ventricular cardiomyopathy [ARVC]).

### Sudden Cardiac Death in Ultraendurance Athletes

- Myocardial scarring may trigger lethal arrhythmias.
- Myocardial infarction more common in older athletes
- Autopsy data not always conclusive

### Cardiac Risk Stratification of Veteran Ultraendurance Racers

- No proven method to evaluate for myocardial fibrosis/scarring
- Reasonable approach includes ECG, sometimes a graded exercise test
- A coronary calcium score may be helpful for athletes >50 years of age
  - A coronary artery calcium (CAC) score <100 is generally regarded as low risk.
  - Athletes with CAC > 100 should be further risk stratified with an exercise test or catheterization as indicated.

## Orthopedic Issues

- While medical conditions result in the most withdrawals from competition in ultraendurance running events, orthopedic conditions, especially trauma, are a common reason for withdrawal from adventure races and UE mountain bike races.
- Major trauma in ultraendurance running races is rare but can result from falls, animal attacks, and lightning strikes.
- Traumatic musculoskeletal injuries tend to occur during the later stages of multiday races, likely due to fatigue.
- In adventure races, injuries are most likely to occur during the orienteering and/or mountain biking portions, with lower limb involvement most common.
- Incidence of injury from mountain bike sections is encouraged by the significant speed, downhill riding, rider inexperience, and competitive nature of participants.

### Acute Traumatic Musculoskeletal Injuries

UPPER EXTREMITY

- The arm/shoulder is the second most commonly injured area, with strains/sprains occurring in kayaking/paddling events and traumatic injuries occurring secondary to falls.

Arm/Shoulder

- Shoulder dislocation
  - 95% occur anterior secondary to external rotation and abduction
  - May occur secondary to a fall onto the arm or shoulder
  - Common in kayakers
- Clavicle fracture
  - Most commonly caused by a fall onto the shoulder, such as an "endo" injury on the mountain bike leg
  - Occur less commonly from a fall onto an outstretched arm
- AC separation
  - The classical cause of an AC joint injury is a direct blow to the acromion with the humerus in an adducted position
- Fractures
  - Scaphoid, distal radius, and radial head most commonly fractured
  - Mechanism is typically via a fall onto outstretched hand (FOOSH).

LOWER EXTREMITY (LE)

- Ankle: most common joint injured in the LE
- Ankle sprain:
  - Most common acute injury in adventure racing
  - Typically caused by inversion injury due to uneven terrain
- Distal fibula fracture/other ankle fracture
- Knee
- Patellar dislocation:
  - Caused by the powerful contraction of the quadriceps, combined with flexion and external rotation of the tibia on the femur (very steep downhill trail running), or by direct contact due to a fall onto the patella while knee is flexed
- Major ligament injury:
  - Anterior cruciate ligament
    - Typically caused by deceleration, pivot-type injuries

### Overuse Musculoskeletal Injuries

UPPER EXTREMITY

- Shoulder overuse injuries are common in kayak/paddling disciplines.
  - Experienced paddlers: excess volume of paddling
  - Inexperienced paddlers: too much, too soon, or improper technique
- Long course triathletes may experience shoulder overuse injuries from swimming
- Rotator cuff tendinopathy most common overuse shoulder injury

LOWER EXTREMITY

- Ankle
  - Achilles tendinopathy
    - Most common lower extremity overuse injury in adventure racers
    - Occurs in 11.5% of ultramarathoners yearly
    - Noninsertional or midsubstance most common
    - Made worse by uphill running
- Knee
  - Patellofemoral pain syndrome (PFPS)
    - Most common cause of anterior knee pain in active adults
    - Typically occurs in events with running such as ultraendurance racing, common in cycling
- Hip
  - Iliotibial band (ITB) syndrome
    - Common in events that require run training
    - Overuse injury that may present with pain at the lateral femoral epicondyle of knee

### Exercise-Associated Muscle Cramps

- Very common in ultramarathons
- Painful, involuntary muscle spasms during or immediately after exercise
- Occur primarily in gastrocnemius and soleus muscles, as well as the hamstring or quadriceps muscle groups
- Most common mechanism is neuromuscular fatigue and increased activity of neuromuscular units.
- Dehydration and sodium loss, particularly in salty sweaters, may also contribute.

## Renal/Electrolyte Issues

### Exercise-Associated Hyponatremia (EAH)

- Defined as a serum sodium level less than 135 mmol/L
- Often asymptomatic and found in up to 50% of ultramarathon (161 km) participants
- See Chapter 97: Mass Participation Endurance Events

### Dehydration

- Common during ultraendurance events
- Body weight losses of up to 8% or greater can occur.
- Oral hydration is the preferred route of fluid administration.
- IV fluids should only be administered in cases of significant hypovolemia and inability to tolerate oral fluid intake.
- In athletes who present with both dehydration and EAH, treatment should focus on EAH because isotonic IV fluids can worsen cerebral edema and lead to death.

### Rhabdomyolysis

- Ultraendurance events can lead to exertional rhabdomyolysis and acute kidney injury.

- Heat stress, dehydration, and NSAID use increase risk.
- Serum creatine kinase (CK) concentrations over 20,000 IU/L are common in athletes without evidence for rhabdomyolysis and renal injury.
- CK readings over 160,000 have been reported without clinical consequences.
- If an athlete presents with dark urine, a urine dipstick with 1+ or greater protein, 3+ or greater blood, and a specific gravity greater than 1.025, further renal evaluation is warranted if there is suspicion for rhabdomyolysis.
- If aggressive fluid resuscitation is to be undertaken, it is important to measure the serum sodium to assess for EAH.

### Vision/Eye Issues

**Description:** Transient vision problems have been reported among ultraendurance athletes

**History and physical examination:** "Hellgate eyes" is caused by corneal edema and drying. Symptoms typically begin during the nighttime hours with runners noticing halos around bright objects. Progresses to blurred vision and a slight gritty sensation in the eyes; the cornea may appear hazy in more severe cases.

**Prevention:** Protective eye wear, frequent blinking

**Treatment:** Stop activity and close eyes; this will resolve most symptoms within several hours. 5% hypertonic saline drops or ointment may also be helpful.

### Psychological Issues

- Fatigue, sleep deprivation, and a negative energy intake–expenditure balance can cause negative emotions during and after competition in ultraendurance athletes.
- Most ultraendurance athletes have a high pain tolerance.
- Sleep deprivation can lead to involuntary microsleeps while exercising, hallucinations (3D), and eventually psychosis.
- A "sleep strategy" for multiday events is important for a successful competition.

## EVENT COVERAGE ISSUES

**Description:** Event coverage for ultraendurance events, especially those held in remote areas, can be quite challenging.

**Communications:** Satellite phones or radios often necessary without cell phone coverage

**Allocation of resources:** Events can be spread over a large geographic area with varied terrain and conditions. Discuss with race directors ahead of time where injuries and illnesses can be anticipated to occur on the course. Personnel and first responders should be positioned in those locations. Examples would include at the bottom of a steep technical mountain bike descent, or at the end of a long trail section without water resources in anticipation of dehydration issues.

**Search and rescue:** Locating and extracting injured or ill competitors can be challenging. Have an emergency plan in place. It is helpful to have a search and rescue team available.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# 99 ROCK CLIMBING

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## INTRODUCTION

- There has been a rapid growth in rock climbing popularity and access in recent decades.
  - The Outdoor Recreation Resources Review Commission in 2007 noted that the number of recreational climbers had increased from 7.3 million to 9.2 million over the preceding decade.
  - The development of local climbing facilities and clubs continues:
    - Climbers are starting at younger ages.
    - Year-round climbing is available indoors.
- Climbing requires endurance, strength, and agility.
  - Athletes must be in good cardiovascular shape to climb.
  - Climbing-specific training reduces injury and increases ability.
- Equipment advances that accompanied the development of the sport have made climbing much safer.
  - Proper equipment maintenance optimizes safety.
  - The rule of thumb is to replace soft goods after 5 years and hardware after 10 years of use.

## GENERAL PRINCIPLES

### Terminology

**Belayer:** The one at the bottom or top of a climb who controls the rope with a belay device

**Rappeller:** The one who descends on the rope, controlling the rate with a figure 8 or another device

**Top rope:** The rope that is placed through chains or metal loops attached to the top of a climb; enables a higher level of safety, alleviating the need to place protection

**Lead climber:** The climber elevates the attached rope while climbing, periodically "clipping-in" to protection; task contains higher risk and difficulty. Subsequent climbers then climb using the top rope.

**Problem:** Bouldering involves the sequential dismantling and eventual conquering of a problem, which can be a particular route or even simply an extremely challenging segment or overhang.

**Pots and Keepers:** Canyoneering features carved by water out of sandstone, often filled with water. Pots are easy to swim or walk across and exit, while keepers are difficult to navigate without specialized techniques and gear

**Prusik:** A friction hitch or knot used to put a loop of cord around a rope, allowing sliding in one direction and a braking action in the opposite

### Types of Climbing

#### Free Climbing (Traditional Rock Climbing)

- "Free" indicates the absence of pulling or hanging on gear, rope, or stepping on anchors
  - True rock climbing with a rope simply used as protection for falls
- "Pro" (protection) indicates equipment used to facilitate free climbing; is removable (Fig. 99.1),
  - Passive: nut, hex (Fig. 99.2: Passive Protection)
  - Active: cam (see Fig. 99.2: Active Protection)
  - Aid climbing: climbing on placed or fixed pieces
- Climbers ascend in "pitches."
  - 20–50 meters per pitch
  - Gear and ropes are cleared and carried with climbers to be placed on subsequent pitches.
  - Climbers belay one another up each pitch.
  - Climbers rappel down from the top, controlling their own descent.
- Minimal impact on natural environment

#### Sport Climbing

OUTDOOR (see Fig. 99.1)

- Climbing on established routes, often with ratings and maps
- Fixed protection
  - Bolts: are anchored into rock at intervals of 2–4 meters
    - Climbers fix a carabiner onto bolts as they pass.
    - Lead climbers use bolts to safely ascend.
  - Chains: chains or welded loops anchored to the rock at the top
    - Following climbers clean the bolts as they ascend.
  - Bolts and chains must be inspected for the safety and stability of the route and rock surface.

INDOOR

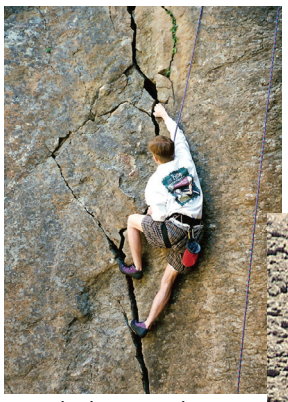
- "Rock gyms"
- Molded holds attached to climbing walls
- Risk of overuse injury, as climbers can do repeated routes, leading to excess fatigue
- Athlete skill level is augmented due to the presence of a safe environment free from natural hazards.

#### Mountaineering/Alpinism

- Often requires travel to austere environments in remote areas
- Climbers employ variable techniques, from hiking to rock and ice climbing over multiple days.
- Injury risk:
  - Serious injury or death from falls, usually related to fatigue and human error
  - Minor injuries can be more serious than major ones due to remoteness and limited rescue potential.
  - The most common injuries are acute and chronic musculoskeletal injuries.
  - Most of these occur in the hands and extremities.
- Environmental risks:
  - Hypothermia/exposure
  - Altitude sickness
  - Falling rocks
  - Wildlife

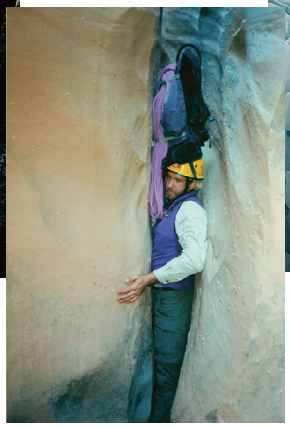
#### Bouldering

- Indoor and outdoor varieties (see Fig. 99.1)
- Climbers solo problems, which are usually 3–4 meters in height, and contain traverses and overhangs.
  - Optimal strategy is repetition, working on one or a few moves to complete the problem
- Spotting and mats provide adequate protection, but short falls can lead to lower extremities injury.
- High demand on upper extremities can lead to overuse injuries, especially to the fingers.
- Indoor and outdoor bouldering carries similar risks.


Free climbing, crack climber.

Outdoor sport climb, lead climber.

Bouldering.


Canyoneering.

Ice climber.

**Figure 99.1.** Types of climbing. (Figures courtesy of Matt Turley.)

Harness.

Climbing shoes.

Climbing gear.

Active protection.

Passive protection.

Quick draw.

Carabiner and ATC.

**Figure 99.2.** Climbing equipment.

- Outdoor bouldering presents added risks from the environment and remoteness.

## Rappelling

- Descent from cliffs using rappel devices on a rope (Fig. 99.3)
- Rappeller controls the rate of descent via friction between the rope and the device.
  - Increased by holding slack end of rope behind the back
  - Use of gloves prevents friction burns.
- A second safety rope can be attached with a belayer at the top or bottom of the cliff for redundant safety measures.
- Backup prusik cords may decrease the risk of injury or fatality.

## Canyoneering

- Canyoneers descend into canyons, narrow riverbeds, or channels, often with steep cliffs on the side.
- Slots cut deep trenches into sandstone due to running water and flash floods over centuries
  - Slot canyons are notoriously dangerous with limited access and contain stretches without escape routes.
- Combination of hiking, rappelling, climbing, swimming, camping, exploring, and courage
- Require "pots" and "keepers," which pose great danger to inexperienced explorers
- Pack rafting—canyoneering down to a river (such as the Colorado River in the Grand Canyon), floating down in a personal raft, then hiking or climbing out
- Weather presents a great danger that can lead to tragedy and death.

## Spelunking

- Cave explorers refer to less experienced counterparts as "spelunkers."

**Figure 99.3.** Rappelling from multiple pitch climb.

- Requires horizontal and vertical movement through caves with extensive use of ropes for pitches, crawling, and squeezing through narrow openings
- Experienced guides and acquiring knowledge of the terrain reduce risks
- Caving can require digging and diving (or even scuba diving for intense cavers).
- An excellent light source, specialized equipment, and redundant safety measures help avert potential disaster.

### Solo Climbing

- No ropes or gear
- Highest risk of injury
- Even elite climbers risk extreme danger, including death.
- Solo climbers may feel that the rewards of accomplishment outweigh the risks, enjoying unencumbered speed without ropes and gear.

### Ice Climbing

- Specialized equipment including crampons and axes (see Fig. 99.1) to ascend the ice
- Anchors are specially designed to drill into ice to ensure protection.
- Unfavorable weather conditions or unstable, melting ice leads to potential tragedy.
- Unique risks include exposure to cold and falling ice as well as the traditional risks of free climbing.

## Difficulty Rating Systems

- The Yosemite Decimal System (YDS) is the most common US climb difficulty rating system and was started in the 1930s by the Sierra Club.
  - **Classes**—rated by difficulty:
    - Class 1: Walking is sufficient
    - Class 2: Hiking with scrambling; occasional use of hands required
    - Class 3: Scrambling with some exposure and the use of handholds. A rope is not required, but falls can still be deadly.
    - Class 4: Simple climbing with natural protection and exposure; ropes are often used; falls may be deadly
    - Class 5: Technical free climbing with ropes; belaying and protection used. Solo climbs often result in severe injury or death. Rated from 5.0 through 5.15c, with letters (a–d) added after 5.10. Advanced climbs start at a rating of 5.10 and progress to expert levels.
    - Class 6: Aid climbing (designation is now rarely used; a separate scale from A0 to A5 is available)
  - **Grades**—rated by time
    - Grade I: One to two hours
    - Grade II: Less than half day
    - Grade III: Half day
    - Grade IV: Full day
    - Grade V: Two day climb
    - Grade VI: Multiday climb
    - Grade VII: One week or longer
  - **Protection**—rates spacing and quality of bolts/protection
    - Follows the movie rating system: G, PG, PG-13, R, and X
    - Significant variation exists. Climbers are advised to typically avoid anything beyond PG-13. R and X noted as caution.
- Bouldering Rating Systems
  - The Hueco Scale is the most commonly used rating system in North America. The scale spans from V0–V16 and is open-ended.
- Ice Climbing uses the Water Ice (WI) Scale, with numerals 1–11 applied, and a + option added. The Alpine Ice (AI) scale is used for glacial ice, which is less dense.
- International rating systems vary by country.

## Equipment and Safety

- Rope
  - Use of dynamic (high stretch) ropes lessen the impact of falls, often by 5%–7% of the length being used
    - Used in vertical rock/ice climbing
  - Static (low stretch) ropes are better for rappelling, as they are stronger and less supple.
    - Used for glacier travel and rappelling, as well as rescue
  - Dry ropes are specially treated to repel moisture, as water weakens ropes.
  - Nondry ropes are less expensive and suited for dry environments only.
  - Length choices include 50 m, 55 m, 60 m, and up to 70 m.
  - Diameter ranges from 7.5 mm to 11 mm.
  - The typical rope has a length of 60 m and a diameter of 10 to 11 mm.
  - Ropes are rated by diameter and number of falls.
- Harness (see Fig. 99.2)
  - Proper fit and sizing is essential for manufactured harnesses.
  - Should be inspected for damage
  - Climbers should inspect each other's harnesses to ensure proper fit and use.
- Shoes (see Fig. 99.2)
  - Tight-fitting, but should be bearable; smaller than a normal shoe
  - Creates a "hoof effect" of the foot to provide strength and grip, as well as protection from the rocks
  - Shoe types range from general use to slipper-like supple shoes for indoor climbing and bouldering, to stiffer boots for more vertical, outdoor climbing.
- Belay device
  - Controls rate of descent on the rope
  - ATC: "air traffic control"
  - Grigri: autolocking belay device
  - Figure-8

- Surface
  - Indoor, outdoor
  - Type of rock, condition
    - Loose rock, dirt, sand, or trees and brush in rock climbing
- Protection (see Fig. 99.2)
  - Active: cams with spring loading
  - Passive: chocks, nuts, hexes, tapers
- Carabiner (see Fig. 99.2)
  - C-shaped with hinged closure of the ring
  - Attaches the climber to a harness, bolt, or rope
  - Locking carabiners are stronger and safer.
- Quick draw (see Fig. 99.2)
  - Two carabiners attached by webbing, used to attach a rope to bolts while climbing
- Chalk
  - Held in a pouch behind the back; used to improve hand friction and reduce moisture
- Webbing, cord, slings
  - Used to carry gear, attach protection, or create a top rope with a carabiner by tying onto a rock, tree, or other solid structure
- Helmet
  - An essential piece of equipment that is often overlooked
  - Should also be worn by the belayer to protect them from falling rocks, ice, and debris

### Biomechanics, Training, and Physiology

- Training variables are more important than anthropometric determinants.
- Leanness, strength, flexible, and having low body fat are characteristics of more successful climbers.
- Energy expenditure:
  - Outdoor climbing expends greater energy than indoor
  - Metabolic Equivalent of Task (METs) values are less than those of running at the same heart rate.
  - $VO_2$ max reaches a plateau during climbing.
  - Handgrip fatigue lasts 20 minutes.

## EPIDEMIOLOGY

- Type of climb
  - Indoor climbing is safer and more controlled.
  - Proper use of protection; improved condition of bolts and chains
  - Regular inspection of equipment condition
  - Risk of death and serious injury greater in mountaineering (Table 99.1)
  - 3.1 injuries per 1,000 climber hours in sport competitions (Table 99.2)

**TABLE 99.1 ACCIDENTS AND INJURIES IN NORTH AMERICAN MOUNTAINEERING AND ROCK CLIMBING**

| Year | Accidents Reported | Injured | Fatalities |
|---|---|---|---|
| 2005 | 111 USA<br>19 Canada | 85 USA<br>14 Canada | 34 USA<br>7 Canada |
| 1951–2005 | 6,111 USA<br>958 Canada | 5,158 USA<br>715 Canada | 1,373 USA<br>292 Canada |

Modified from Williamson JE. Accidents in North American mountaineering. Golden: American Alpine Club Inc.; 2006, pp 97-98.

- Skill level
  - Elite climbers and higher difficulty climbs carry higher risk.
    - More overuse injuries to the upper extremities
    - Risk of death is higher in more exposed environments.
  - Most injuries, however, occur at or below the climber's usual level.
  - Inexperienced climbers experience lower extremity abrasions and lacerations.
  - Greater risk in male climbers who climb harder routes, have been climbing more than 10 years, and lead climb more than top rope climb.
- Environmental
  - Falling rocks
  - Isolation
  - Weather, sun, cold
  - Altitude
  - Lack of preparation
  - Plant and animals/insects (e.g., bees)

## CLIMBING INJURIES BY ANATOMIC REGION

### Upper Extremity

More commonly overuse injuries

#### Shoulder

- Rotator cuff tendonitis, tear, or impingement
- Rupture long head biceps
- SLAP (superior labrum, anterior to posterior) lesions
- Acromioclavicular (AC) sprain
- Dislocation
- Muscular strain: usually of the rhomboid, latissimus dorsi, or lower trapezius

#### Elbow

- Anterior
  - Climber's elbow: brachialis strain (see the Climbing-Specific Injuries section)
  - Distal biceps strain or tear
  - Posterior interosseous nerve compression
- Medial
  - Forearm flexor strain, overuse leads to medial epicondylitis
  - Pronator teres strain or tear
  - Ulnar nerve compression
- Lateral
  - Extensor strain: lateral epicondylitis
  - Radiocapitellar compression
  - Superficial branch of radial nerve compression
- Posterior
  - Triceps tendonitis
  - Osteoarthritis
  - Stress fracture, osteochondritis dissecans (OCD)

**TABLE 99.2 2005 WORLD CHAMPIONSHIPS IN ROCK CLIMBING INJURY RISK**

| Year | 2005 |
|---|---|
| Climbers | 443 |
| Countries | 55 |
| Climbing days | 520 |
| Acute medical issues | 18 |
| Serious medical issues | 4 (zero deaths) |
| Injury rate per 1,000 climbing hours | 3.1 |

Modified from Schoffl VR, Kuepper T. Injuries at the 2005 World Championships in rock climbing. *Wilderness Environ Med*. 2006;1(3):187-190, used with permission.

### Wrist

- Tendonitis/tenosynovitis
  - Flexor carpi ulnaris most common
- Sprains
- Carpal tunnel syndrome, tendon hypertrophy, edema
- Triangular fibrocartilage complex (TFCC) injury
- Retinacular injury

### Hand

- Climber's finger: flexor pulley injury (see the Climbing-Specific Injuries section)
- "Sausage fingers"
  - Joint effusion with synovial irritation, cartilage damage
- Flexor digitorum superficialis (FDS) and flexor digitorum profundus (FDP) tendonitis, strain, tear, laceration (Fig. 99.4)
  - Tenoperiostitis, tears at insertion
  - Force loads on FDS and FDP at a mechanical disadvantage with certain grips
- Extensor hood syndrome (EHS)
  - Induced by use of a crimp hold
  - Degenerative spurring to the joints results in irritation to tendon sheaths
  - Often associated with chronic flexor mechanism injuries
  - Clinically see 3–5-degree extension deficits, as well as morning stiffness
- Collateral ligament injury: pocket hold
- Flexion contracture
- Stress fracture
- Epiphyseal fracture
- Nodule/trigger finger (see Fig. 99.4)
- Dupuytren's contracture
- Ganglion cyst (see Fig. 99.4)
- Mallet finger
- Lumbrical tear/lumbrical shift (see Fig. 99.4)
- Amputation
- Hand grips affect injury patterns (Fig. 99.5)
  - Open grip: strain to the FDP is greater than to the FDS; is easier on joints, ligaments, and tendons
  - Crimp (with closed ring): strain on the FDS, the distal interphalangeal (DIP) joint, and the volar plate
  - Pocket grip: strain to the FDP and collateral ligaments
  - Vertical grip: strain to the FDS
  - Pinch grip
  - Finger locks
  - Hand stacks
  - Hand and fist jams

## Lower Extremity

### Hip

- Adductor strain: wide bridging between foot holds
- Strain to quadriceps or hamstring

### Knee

- Meniscus tear: common usage of the frog position with knee flexion and external rotation, pushing upward
- Lateral collateral ligament (LCL) complex injury, induced by a heel hook maneuver with downward force on the lateral heel in a hold, hip flexed in external rotation, and knee flexed
- Anterior cruciate ligament (ACL) tear: related to fall from bouldering heights

### Ankle

- Sprains are more common than fractures
  - Foot supinated in small shoes (dorsiflexion, plantarflexion–inversion)
  - Bouldering falls from up to 3 meters, risk can be reduced with adequate mat usage
  - Falling into climbing surfaces or tangling into ropes on an overhang
- Fracture or osteochondral injury, talar dome OCD, hard landings in climbing shoes

### Foot

- Neurologic symptoms of numbness, tingling from tight shoes
- Hallux valgus is common in climbers with 5 or more years of experience
- Blisters, toenail contusions/loss

## Spine/Abdomen

- Cervical: strains, degenerative changes, facet synovial impingement
  - Caused by falls, overhang extensions
  - Belay position, looking upward in extension; can wear prism glasses to mitigate
- Lumbar: strains and disc injury, degenerative changes, stress injury, or spondylolysis
  - Result from falls, impact
  - Repeated stress from falls in a harness, hyperextension
  - Pectoralis imbalance causing thoracic kyphosis and lumbar lordosis
- Abdominal: strains, contusions
  - Sustained in traverses, reaching
  - Falls and harness can contribute to strains and/or contusions, but these are less likely when harnesses are properly fitted and applied

### Head

- Impact
  - Falls can lead to concussion.
  - Rocks from above can impact the head.
  - Helmets should be used by the climber(s) and belayer.
- Eye
  - Hyphema from falling rocks or rope whip
  - Eye protection for the belayer is essential

### Skin

- Abrasion: "climber's rash" to lower extremities of less experienced climbers
- Laceration
- Sunburn
- Rope burns, blisters to hands or feet

# CLIMBING-SPECIFIC INJURIES

## Climber's Finger

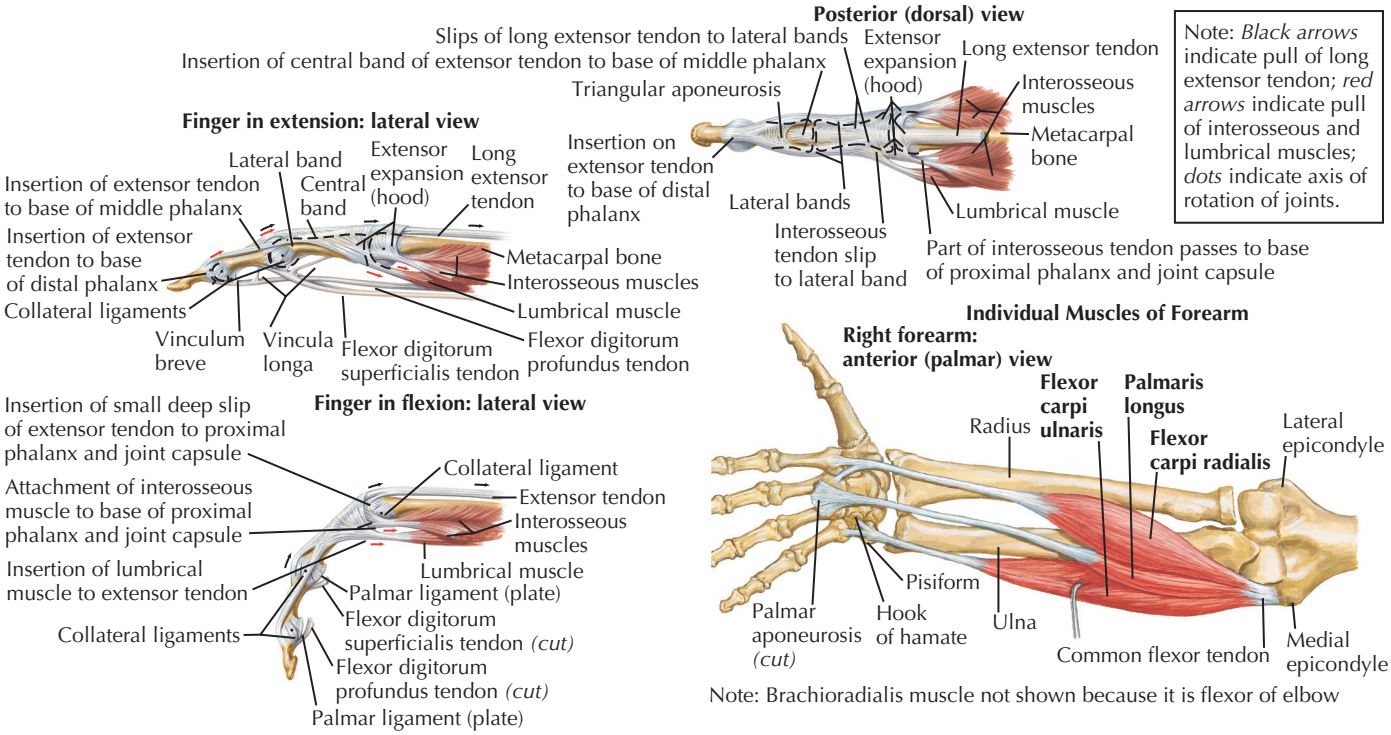
**Description:** A unique climber's injury to the flexor pulley system; injuries to the A2, A3, or A4 flexor pulleys; multiple pulley injuries more serious (Fig. 99.6); injury of the A2 flexor pulley is most common; caused by acute or chronic overuse. Hand grips and techniques contribute to the development of pulley injuries and other finger injuries. Accounts for 30% of finger injuries, and half of elite climbers have sustained some degree of pulley injury. At 90 degrees, the pulley sustains more tension than the tendon. Grading of the injury is very important (Table 99.3).

**Presentation:** The climber often presents with a history of feeling a "pop" related to the injury with associated swelling and pain. May present with a long-standing injury and/or chronic pain

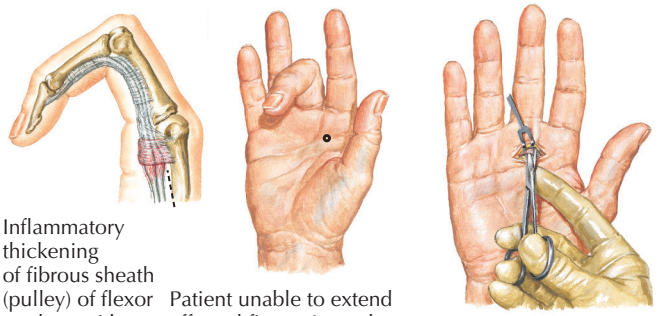
**Physical examination:** Bowstringing of the affected digit with acute pain and swelling. Most commonly affects the fourth digit of the nondominant hand (see Fig. 99.6)

**Diagnostics:** Magnetic resonance imaging (MRI), ultrasound. Dynamic ultrasound is 98% sensitive, 100% specific; user dependent (see Fig. 99.6).

## Flexor and Extensor Tendons and Fingers




## Stenosing Tenosynovitis of Flexor Tendons of Finger (Trigger Finger)

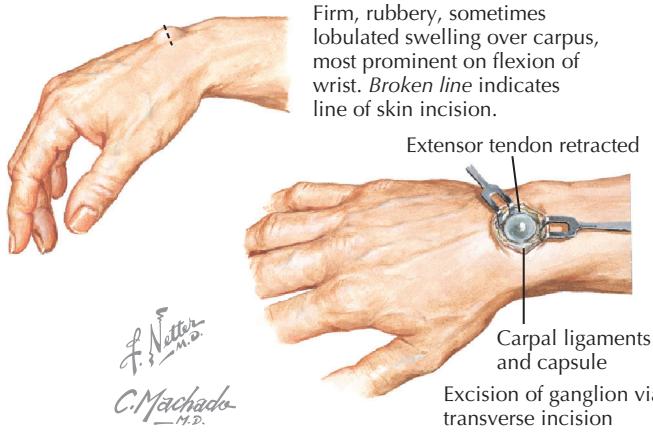


Inflammatory thickening of fibrous sheath (pulley) of flexor tendons with fusiform nodular enlargement of both tendons. *Broken line* indicates line for incision of lateral aspect of pulley.

Patient unable to extend affected finger. It can be extended passively, and extension occurs with distinct and painful snapping action. Circle indicates point of tenderness where nodular enlargement of tendons and sheath is usually palpable.

Incision of thickened pulley via small transverse skin incision just distal to distal flexion crease releases constriction, permitting flexor tendons to glide freely and inflammation to subside.

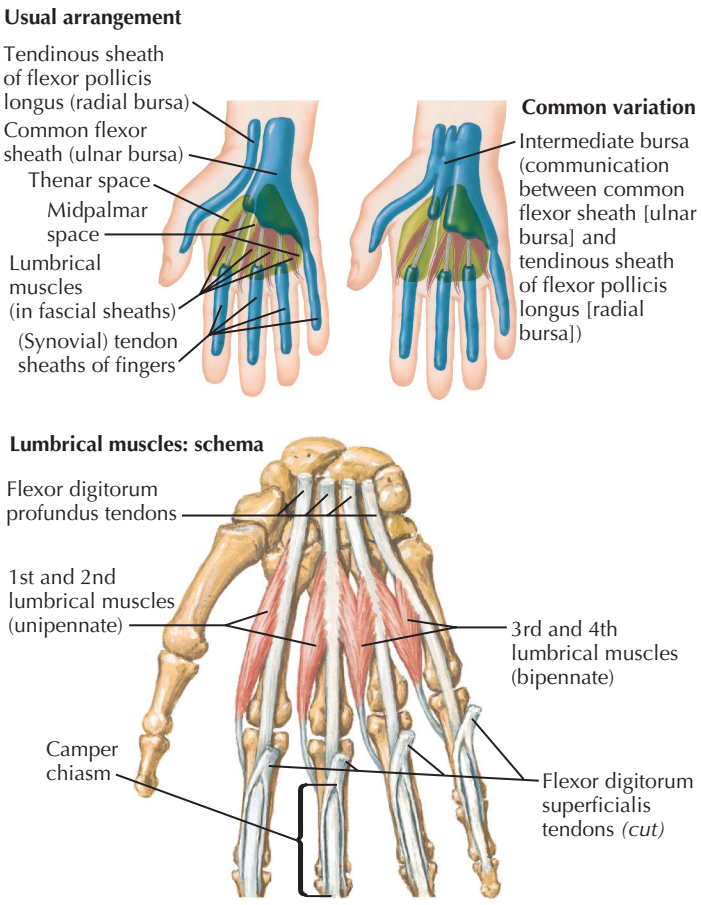
## Ganglion of Wrist




Firm, rubbery, sometimes lobulated swelling over carpus, most prominent on flexion of wrist. *Broken line* indicates line of skin incision.

Excision of ganglion via transverse incision

## Lumbrical Muscles and Bursae



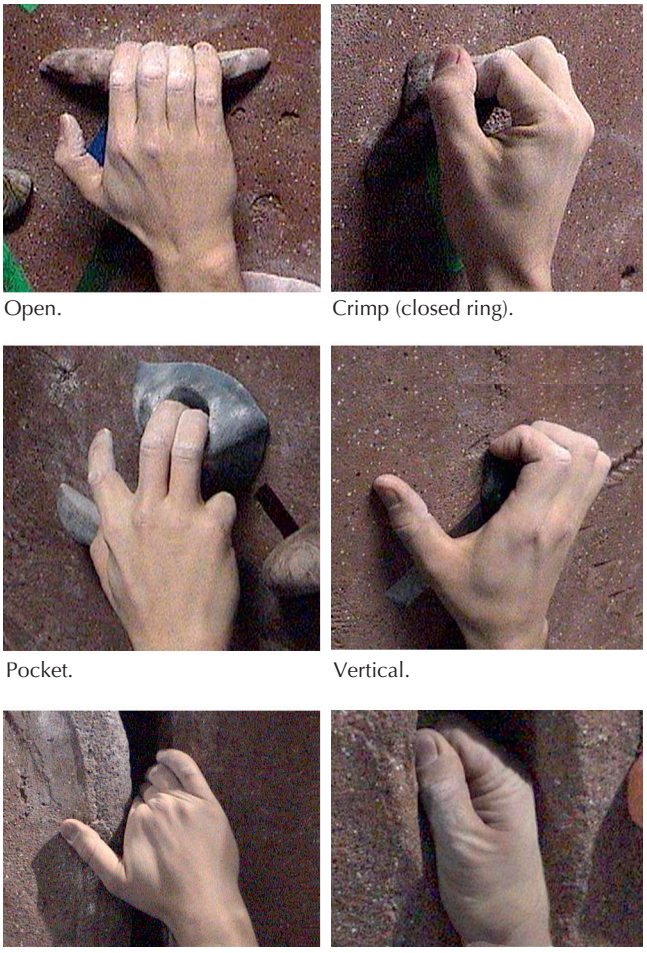

Note: Flexor digitorum superficialis and profundus tendons encased in synovial sheaths are bound to phalanges by fibrous digital sheaths made up of alternating strong annular (A) and weaker cruciform (C) parts (pulleys).

**Figure 99.4.** Upper extremity climbing injuries.

**Treatment:**

- **Nonsurgical treatment** (Table 99.4)
  - Grade 1: pulley strain
    - No immobilization
    - Physical therapy for 2 to 4 weeks
    - Return to light climbing at 4 weeks
    - Full climbing at 6 weeks
    - Taping for 3 months
  - Grade 2: complete rupture of the A4 pulley, or a A2 or A3 partial rupture
    - 10 days immobilization
    - Physical therapy for 2 to 4 weeks
    - Return to light climbing at 4 weeks
    - Full climbing at 6 to 8 weeks
    - Taping for 3 months
  - Grade 3: complete A2 or A3 rupture
    - 10 to 14 days immobilization
    - Physical therapy for 4 weeks
    - Use of a thermoplast or soft-cast ring
    - Return to light climbing at 6 to 8 weeks
    - Full climbing at 3 months
    - Taping for 6 months
- **Surgical treatment** (see Table 99.4, Fig. 99.6)
  - Grade 4: multiple pulley ruptures, or a A2 or A3 pulley rupture with lumbrical or ligament injury
  - Surgical repair:
    - Widstrom technique: loop and a half
    - Weilby repair can be used as an alternative
  - 14 days immobilization
  - Physical therapy for 4 weeks
  - Thermoplast or soft-cast ring should be used
  - Return to light climbing at 4 months
  - Full climbing at 6 months
  - Taping should accompany climbing for at least a year

**Prognosis:** Most climbers do well with conservative care. Ten percent experience persistent pain. Consider surgical treatment in climbers with residual pain or inability to return to prior climbing level.



Open. Crimp (closed ring). Pocket. Vertical. Finger jam. Fist jam.

**Figure 99.5.** Common hand grips.

**TABLE 99.3 GRADING OF FLEXOR PULLEY INJURIES**

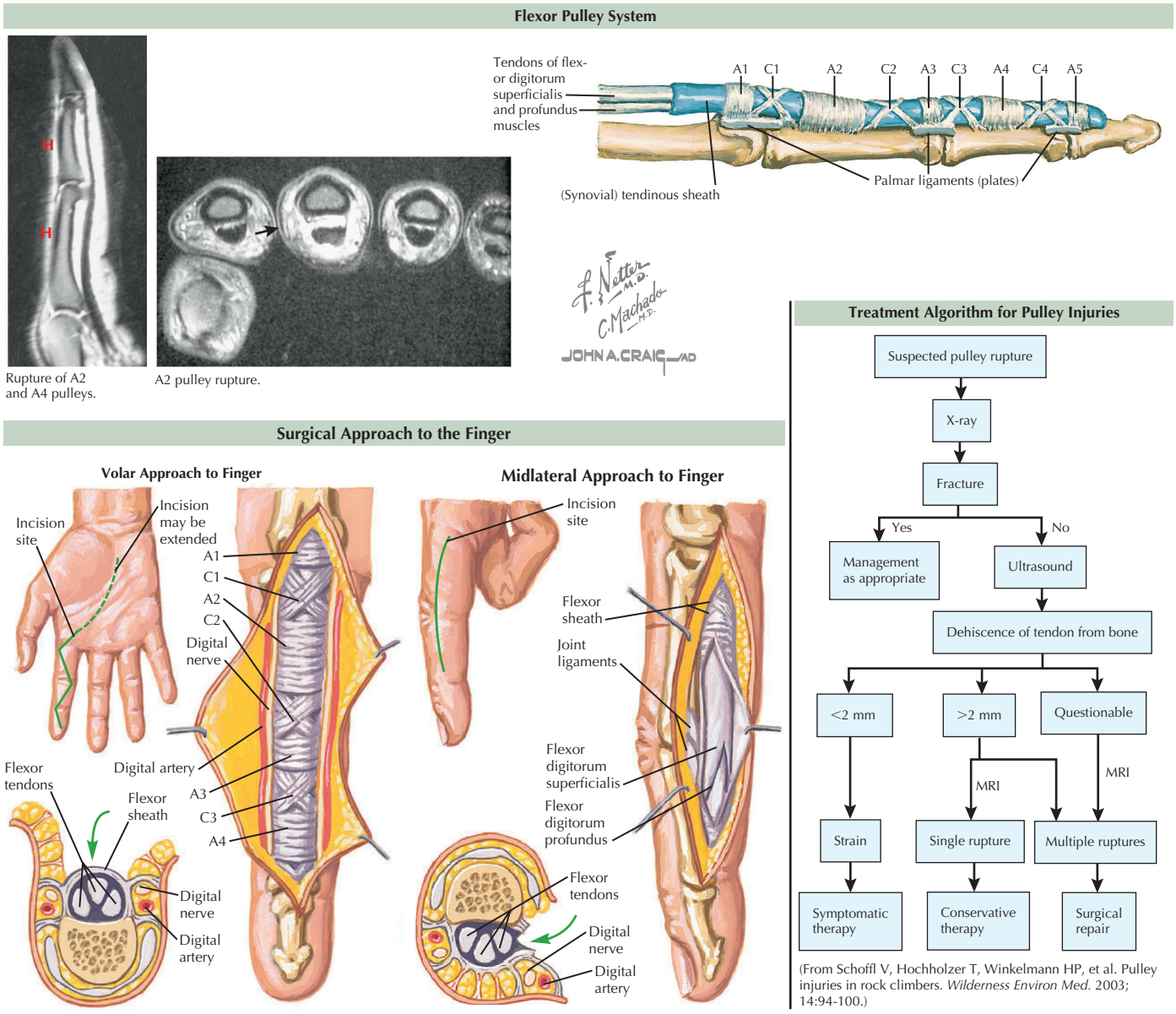
| Grade | Flexor Pulley Injury Pattern |
|---|---|
| Grade 1 | Pulley strain |
| Grade 2 | Complete rupture of A4 pulley or partial rupture of A2 or A3 |
| Grade 3 | Complete rupture of A2 or A3 pulley |
| Grade 4 | Multiple pulley ruptures or single pulley rupture with lumbrical or collateral ligament injury |

From Schoffl V, Hochholzer T, Winkelmann HP, Strecker W. Pulley injuries in rock climbers. *Wilderness Environ Med*. 2003;14(2):94-100.

**TABLE 99.4 TREATMENT OF PULLEY INJURIES**

| | Grade 1 | Grade 2 | Grade 3 | Grade 4 |
|---|---|---|---|---|
| Injury | Pulley strain | Complete rupture of A4 or partial rupture of A2 or A3 | Complete rupture of or A3 | Multiple ruptures, such as A2/A3, A2/A3/A4, or single rupture (A2 or A3) combined with lumbrical muscle or ligament damage |
| Therapy | Conservative | Conservative | Conservative | Surgical repair |
| Immobilization | None | 10 days | 10–14 days | Postoperative 14 days |
| Functional therapy | 2–4 weeks | 2–4 weeks | 4 weeks | 4 weeks |
| Pulley protection | Tape | Tape | Thermoplastic or soft-cast ring | Thermoplastic or soft-cast ring |
| Easy sport-specific activities | After 4 weeks | After 4 weeks | After 6–8 weeks | 4 months |
| Full sport-specific activities | 6 weeks | 6–8 weeks | 3 months | 6 months |
| Taping through climbing | 3 months | 3 months | 6 months | >12 months |

From Schoffl V, Hochholzer T, Winkelmann HP, Strecker W. Pulley injuries in rock climbers. *Wilderness Environ Med*. 2003;14(2):94-100.




(From Schoffl V, Hochholzer T, Winkelmann HP, et al. Pulley injuries in rock climbers. *Wilderness Environ Med.* 2003; 14:94-100.)

**Figure 99.6.** Climber's finger.

## Climber's Elbow

**Description:** Brachialis tendonitis or a tear at the muscle–tendon junction; caused by flexion and pronation of the elbow typical of climbing with traverses; insufficient firing of the biceps brachii in the flexed and pronated positions contribute to overuse of the brachialis (Fig. 99.7).

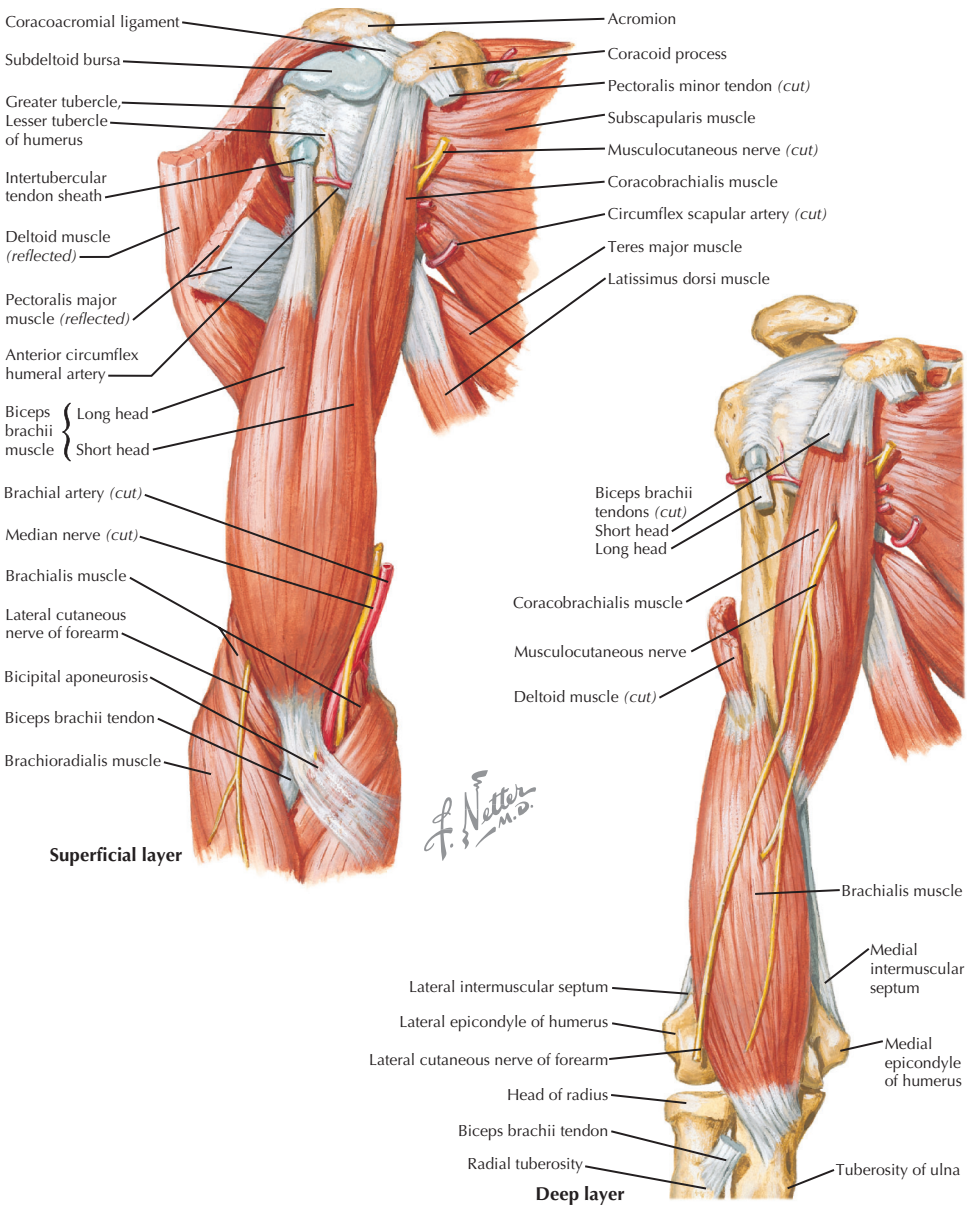
**Presentation:** Pain with climbing; typically presents after a prolonged, hard climb or a lengthier period of intense climbing with insufficient rest between climbs

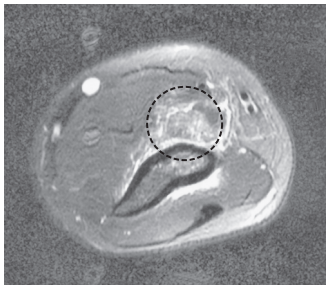
**Physical examination:** Presence of pain to the anterior elbow that is worsened with elbow flexion and pronation. A partial tear or rupture will typically have swelling and/or ecchymosis.

**Diagnosis:** Physical examination and history often sufficient to reveal climber's elbow. MRI or dynamic ultrasound can aid in identifying the presence of tendonitis or a tendon tear (see Fig. 99.7).

**Treatment:** Most athletes with climber's elbow can be treated conservatively. Rest is the mainstay of treatment; typically 2 to 4 weeks of rest is sufficient for a strain. Modified climbing may be attempted if milder injury is present, but the athlete must be pain-free and climbing with diminished frequency/intensity and at 2 to 3 levels below normal. Physical therapy is highly beneficial but requires an experienced physiotherapist. Modality care; administration of oral anti-inflammatories for inflammation and pain. Strengthening of opposing muscle groups. Complete ruptures should be treated with surgical repair within a week or two of injury for best results. Patients who fail conservative treatment should also have surgical consultation. Surgery requires months for recovery.

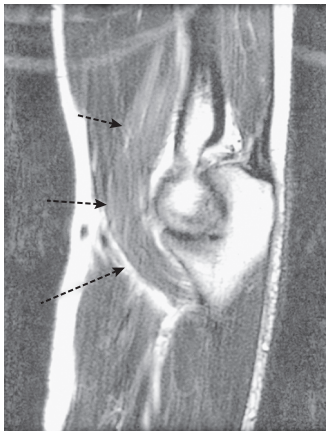
**Prognosis:** With adequate rest period, physical therapy, and strengthening of opposing muscle groups, many climbers return to full activity pain-free, whether conservative or surgical



Axial T2 FSE images with fat saturation circle outline intramuscular edema in the brachialis.

Coronal proton density fat saturated FSE image circle outlines edema in the dunal brachialis.



T2 FSE sagittal image through elbow. *Arrows* demonstrate increased signal intensity in the distal brachialis.

**Figure 99.7.** Climber's elbow.

treatment was used. Length of restricted activity depends on the severity of the injury and resolution of symptoms.

## INJURY PREVENTION, DIAGNOSIS, AND TREATMENT

### Medical Care of Climbers

- Physician inexperience or misconception
  - "What did you expect when you climb?"
  - Leads to delayed diagnosis and treatment
  - Many elite climbers find that health professionals do not understand climbing stresses and are not helpful in diagnosis or treatment
- Compliance is a significant issue with climbing overuse injuries.

### Prevention

- Tendon strengthening, stretching, inclusion of opposing muscle groups
- Low-weight, high-repetition endurance training
- Heat prior, ice following
- Taping: pulley protection, buddy taping when necessary
  - New method of "H-tape" produces added protection
  - Division of tape into two strips with a bridge in the middle, placing the bridge at the volar proximal interphalangeal (PIP) joint, and then the strips are placed circumferentially on either side of the dorsal PIP
- Adequate rest period between climbs

### Rest Guidelines

- Twenty-four hours should be taken for recovery after training.
- Forty-eight hours should be taken for recovery after climbing or hard training.
- Significant injuries require several months of rest.
- Splinting, ring to support pulley, taping
- Older climbers require more rest.

## Rehabilitation

- Can be for 2 to 3 months
  - Stretches
  - Range-of-motion exercises
  - Low weights
  - Gradually increased repetitions
- Strengthen muscle antagonists
- Change climbing technique (e.g., rely more on open than cling grip), slowly return to activity, start at lower level
- Formal physical therapy with an experienced therapist familiar with climbing injuries
- Rock climbing can aid the therapy of other injuries
  - Improves functional ankle instability with near-static movements, but care must be taken to not reinjure self with belaying to the ground

## Repair

- Indication is based on injury grade and injury type.
- Failure of conservative therapy
- Reconstruction of the fibro-osseous flexor sheath of fingers or repair of a brachialis tear; can fail with insufficient compliance/rehabilitation

## Counsel for Climbers

- Adequate instruction
- Climb with trusted companions
- Climb to your ability, know your limitations
- Impeccable equipment use and safety
  - Protect equipment from moisture, sun, heat, dirt, water, oil, abrasion
  - Use appropriate protection, knots, equipment
  - Date ropes and harnesses, check damage after each use, replace on time
- Plan and leave an itinerary
- Carry a cell phone, map, GPS
- First aid kit
- Food, water, sunscreen, eye protection, helmet

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Available at www.ExpertConsult.com.

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# 100 RODEO AND EQUESTRIAN SPORTS

Dawn Mattern • Michael Turner • Adrian McGoldrick

## EQUESTRIAN EVENTS

### Introduction

Data are sparse, but the risk of injury while riding or working with horses is higher than that in automobile racing, motorcycle riding, football, and skiing and is as the same as that in rugby.

### Events

- Olympic: dressage, eventing, show jumping
- Paralympic: dressage
- Racing: flat racing, harness racing, point-to-point racing, steeplechase, thoroughbred horse racing
- Others: combined driving, endurance riding, horseball, reining, tent pegging, vaulting, trail riding, sorting, rodeo

### Common Injuries and Medical Problems

- Chest trauma is the most common injury that is evaluated in hospitals.
- Head trauma and concussions are the second most common injuries evaluated in hospitals.
- Eventing witnessed 12 rider deaths in 18 months (2007–2008) and the highest rate of spinal injury.

## RODEO

### Introduction

- Rodeo is a competitive sport with participants of all ages and at all competition levels.
- Athletes may compete in a single event or multiple events and may attend anywhere from one to four or five events in a single weekend.
- Most events are derived from skills needed to work cattle.

### Epidemiology

- The composite injury rate for professionals is 2.3/100 competitor exposures and for high school competitors is 8.2/1,000 competitor exposures.
- The incidence of catastrophic injury is around 20/100,000, with a fatality rate of 7.29/100,000.
- The highest injury rates are found in roughstock events, with bull riding injury rates 2-fold greater than those in any other major event.
- When compared to the injury rates of all other contact sports, bull riding ranks as the most dangerous.

### Events

#### Roughstock

An 8-second duration is required for a qualified ride; one arm holds on to the animal, while the other is free and is not allowed to contact the animal. The score is based on the performance of the athlete and animal.

**Bull riding:** The rider hangs onto a rope tied to a bull (Fig. 100.1A).

**Saddle bronc:** The rider seated in a saddle, holding onto a rope attached to a halter.

**Bareback riding:** The rider holds on to a rigging attached to a horse's back (Fig. 100.1B-C).

**Steer riding:** The rider hangs onto a rope tied to the steer (usually an event for younger athletes prior to starting bull riding).

#### Timed Events

Quickest time wins.

**Steer wrestling:** While atop a galloping horse, the athlete slides his/her arms onto the neck of a steer and throws it to the ground.

**Calf roping:** While riding a horse, the athlete ropes a calf, dismounts, and ties three of the four legs together.

**Team roping:** The header ropes the head of a steer, while the heeler ropes the heels.

**Barrel racing:** The rider races around three barrels in a cloverleaf pattern.

**Steer roping:** Similar to calf roping, except with a steer

**Goat tying:** A youth event similar to calf roping

**Breakaway roping:** Similar to calf roping, but the calf is not thrown and tied

**Pole bending:** Riding a horse through six poles in a pre-established pattern

**Cutting:** The rider separates a single animal from a herd.

### Coverage

- Some rodeos are well covered by medical staff and services, but many are not covered at all, or maybe only by an ambulance crew.
- May be difficult to arrange follow-up care as many rodeo athletes travel to multiple sites
- Rodeo athletes pay entrance fees to each rodeo; a withdrawal due to injury results in the loss of money.
- An athlete may request a medical release if unable to perform, but medical personnel cannot prevent an athlete from participating.

### Common Injuries and Medical Problems

- Thoracic compression is the most common cause of catastrophic injury.
  - Unknown if a rodeo protective vest reduces injury
- Soft tissue contusions, sprains, and strains—most frequently reported injuries
- The true incidence of concussions is unknown as athletes do not usually seek care.
  - The head is the most commonly injured area across all ages.
  - "Rodeo SCAT" modifies Maddock's questions to the sport of rodeo.
  - Bareback riders experience 46 × *g* of head acceleration, while bull riders experience 26 × *g* (football players experience 21–23 × *g* consistently and may sustain hits of 98–102 × *g*).
  - Unable to establish impact threshold for concussions regardless of sport; helmet use does appear to reduce incidence of both catastrophic injury and fatality
- Thumb amputations—combination of crush and avulsion mechanisms occurs in roping athletes, with high incidence of infection and failed replantation
- Pectoralis major/latissimus dorsi tendon ruptures—occurs in steer wrestling, and is managed by surgical fixation
- Femoral acetabular impingement—exacerbated in the roughstock riding position
- Methicillin-resistant *Staphylococcus aureus* (MRSA)—livestock are frequently colonized, and infections can be transmitted in both directions

**A.** Bull rope.

**B.** Bareback rider's glove.

**C.** Glove in rigging.

**Figure 100.1.** Roughstock events.

# HORSERACING

## Introduction

- Horseracing is an immensely popular global sport with television audiences of over 100 million for major events (Melbourne Cup, Kentucky Derby); annual attendance figures are over 5 million in Great Britain alone.
- It is a very exciting and physically demanding sport with high injury rates.
- Career-ending injuries and fatalities are not uncommon.

## Epidemiology

- Horses weigh 1,000–1,200 lbs (450–550 kg) and travel at speeds of 20–40 mph (32–64 kph).
- Jockeys are seated approximately 6 ft (183 cm) above ground level.
- The jockey's head is approximately 8.5 ft (260 cm) above the ground during flat racing (higher when jumping).
- Concussion rates in horseracing are the highest in the recorded literature.
- Flat jockeys fall every 250 rides (concussion rate, 17.1/1,000 participant hours).
- Jump jockeys fall every 16 rides (concussion rate, 25.0/1,000 participant hours).
- Amateur jockeys fall every 8 rides (concussion rate, 95.2/1,000 participant hours).
- In flat racing, 0.41% of rides result in a fall, and 40% of falls result in an injury.
- In jump racing, 6.1% of rides result in a fall, and 17% of falls result in an injury.
- Fatality rates are roughly 460–900/100 million rides. Comparable fatality rates/100 million participant days in other sports are: mountaineering, > 780; air sports, > 640; motor sport, 146; water sports, 67.5; rugby union, 15.7; soccer, 3.8.
- In addition to the trauma caused by falls, the horse can inflict injuries by biting, pulling, kicking, standing, or rolling on the jockey, as well as hitting the rider in the face with a sudden movement of the head.

## General Principles

- Horseracing is broadly divided into flat racing and jump racing (sometimes referred to as National Hunt racing).
- Trotting is not included in this review but is very popular with fewer injuries.
- Jockeys can generally start race riding at 16 years of age and are usually referred to as "apprentice jockeys" at the beginning of their careers (usually the first 5 years).
- Jump jockeys retire around 40 years of age, but flat jockeys can continue past 50 years of age.
- Male and female jockeys compete on equal terms. The male:female jockey ratio tends to be closer to 50:50 in amateur racing and higher in professional racing.
- Flat racing generally takes place over a 12-month season, whereas jump racing tends to be limited to the winter months when the ground is softer.
- Flat jockey in Great Britain ride in an average of 600 races/year.
- Jump jockeys in Great Britain ride in an average of 300 races/year.
- Flat racing takes place over 0.625–2.75 miles (1–4.4 km).
- Jump racing takes place over 2–4.5 miles (3.2–7.2 km).
- The minimum riding weight varies considerably from country to country. In Great Britain, flat jockeys must weigh at least 112 lbs (51 kg) and jump jockeys must weigh 140 lbs (63.5 kg). This weight must be achieved by the jockey while wearing normal riding clothes, riding boots, and carrying a saddle.
- Jump racing requires horses to jump over either hurdles [3.5 feet high (101 cm)] or steeplechase fences [4.5 feet high (137 cm)].

## Safety Equipment

### HELMETS

- Helmets are designed to attenuate energy on impact through deformation of the helmet, and in particular the inner lining, which is usually constructed from EPS (expanded polystyrene) foam. The prevention of skull fractures and catastrophic injury is the primary aim. To date, no helmet has been proven to prevent concussions, but research is ongoing to develop a tangential impact test with a view to reducing concussions.
- When designing a helmet, the criteria used are:
  - Shock absorption
  - Penetration of the shell
  - Lateral deformation
  - Load distribution
  - Area of protection
  - Retention system strength and effectiveness

- Field of vision
- Weight
- Current helmet standards:
  - Helmet standards vary enormously from country to country, and riders wishing to compete outside the US must ensure that their helmets meet the required standards for participation.
  - Helmets in the US are required to meet ASTM or SNELL standards. In some situations, other standards may also be acceptable (e.g., the European Standard EN or the Australasian Standard AS/NZ)
  - US: ASTM F1163-13/ASTM F1163-04a/EN 1384:2012/AS/NZ 3838:2006/SNELL E2001

SAFETY VESTS

- Safety vests are purely designed to reduce chest wall injuries (i.e., rib fractures) and are not capable of preventing spinal injuries. Lightweight vests (Level 1) are licensed for use during race riding only, while heavier vests (Levels 2 and 3) are required while riding out/barrier trials/breeze ups, etc.
- Current safety vest standards:
  - As with helmets, safety vest standards vary enormously from country to country, and riders wishing to compete outside the US must ensure that their safety vests meet the required standard for participation. In the US, safety vests are required to meet an ASTM or SNELL standard. In some situations, other standards may also be acceptable (e.g., the European Standard EN or the Australasian Standard AS/NZ).
  - US: ASTM F2681-08/ASTM F1937-04/EN13158:2000 or 2009/BETA 2009 or BETA 2000 Body Protector Standard

GOGGLES

- There are currently no equestrian goggle standards, but high-impact plastic or polycarbonate lenses are recommended to reduce the risk of shattering and eye injury.

### Racecourse Medical Cover

- Medical cover at racetracks varies across the globe.
- In almost all countries, a minimum of two ambulances follow the riders. In many countries it is standard to have two doctors at the track also. However in some countries, such as New Zealand and Japan, only paramedics work at the track.
- Diagnostic equipment at the track varies from the very basic to full X-ray scanning facilities in Japan.
- Where medical staff members are deployed, it is important to ensure that they have the appropriate training and qualifications for prehospital care, and that they have access to the necessary emergency equipment and supplies.

### Racecourse Safety Arrangements

- Major changes have taken place over the last 10 years with the replacement of cement/steel running rails and railings with flexible plastic to reduce the risk of injury.
- Provision of access to the racing surface has improved with the creation of ambulance roads or the deployment of four wheel vehicles in situations where access is difficult.
- In jump racing, rules are in place to bypass fences if a rider or horse is injured and cannot be moved before the racing horses complete the circuit and return to the site of the accident.

### Insurance

- Race riding is a high-risk sport, and insurance can be difficult to arrange. In countries where there is an active Jockeys Association, insurance is usually available for death and disability, career-ending injuries, and private medical care.
- All medical staff must have suitable medical malpractice insurance if they wish to provide medical support on a racecourse.

### Race Track Surfaces

- Most racing in Europe takes place on turf during the summer. During the winter months, some racecourses provide an "all-weather" racing surface which is constructed from a mixture of artificial frost resistant materials.
- In the US and Australia, racing also takes place on dirt or cinder tracks.
- The injury rates on turf tracks are similar to those found on all-weather tracks.

## Common Injuries and Medical Problems

- Soft tissue injuries: 75%–80%
- Fractures: 10%–18%
- Dislocations: 1%–4%
- Concussions: 8% in professional racing (18% in amateur racing)

## Head and Neck

- Concussion
- Cervical spine fracture

## Upper Limb

- Fractured clavicle
- Dislocated shoulder

## Torso, Back, and Trunk

- Spinal injury
- Ruptured spleen

## Hip, Pelvis

- Fractured pelvis

### Lower Limb

- Fractured femur
- Fractured tibia and fibula

### Career-Ending Injuries

- In Great Britain from 1991 to 2005, there were approximately 1,113,500 rides, 32,445 falls, and 555 injuries. Of these injuries, 45 (8.1%) were career ending (including four fatalities).
- Career-ending injury location distribution: 24.3% torso–pelvis, 22.1% upper limb, 20% head, 17.7% lower limb, 13.3% neck and spinal cord

## Summary

- Horse racing is a very high risk activity, and jockeys should wear the highest standard of protective equipment when riding (helmets and safety vests).
- Injuries are common, and medical staff providing cover at racing events must be prepared to deal with fractures, dislocations, concussion, head injuries, and acute spinal trauma.
- All medical staff members must be suitably trained in prehospital care and have the appropriate equipment on site to manage the anticipated trauma. This should include a rapid method of transportation for critically injured jockeys (e.g., a fully equipped paramedic ambulance).

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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# THE EXTREME ATHLETE

# 101

Lior Laver • Omer Mei-Dan

## INTRODUCTION

- The definition of extreme sports (ES) extends to any sport featuring high speed, height, a real or perceived danger, a high level of physical exertion, highly specialized gear, or spectacular stunts and involves elements of increased risk. ES activities tend to be individual and can be pursued both competitively and noncompetitively.
- ES activities often take place in remote locations and in variable environmental conditions (weather, terrain). Little or no access to medical care may be present, and even if medical care is available, it usually faces challenges related to longer response and transport times, access to limited resources, limited care provider experience due to low patient volume, and more extreme geographic and environmental challenges.
- Popular ES include BMX and mountaineering; hang gliding and paragliding; free diving; surfing (including wave, wind, and kite surfing), personal watercraft; whitewater canoeing, kayaking, and rafting; bungee jumping, BASE jumping, and skydiving; extreme hiking and skateboarding; mountain biking; inline skating; ultraendurance races; alpine skiing and snowboarding; and ATV and motocross sports.
- In the last 2 decades, there has been a major increase in both the popularity of and participation in ES, with dedicated TV channels, internet sites, high-rating competitions, and high-profile sponsors drawing more participants.
- The popularity of ES has been highlighted in recent years by the success of the X Games, an Olympic-like multiday multisport competition showcasing the talents in ES.
- The risk and severity of injury in some ES is high, and participation in ES is associated with risk of injury or even death. Therefore, the extreme athlete, both amateur and professional, as well as the medical personnel treating these athletes, must consider the risk of injury and measures for injury prevention.
- Medical personnel treating the ES athlete need to be aware that there are numerous differences between the common traditional sports and this newly developing area. These relate to the temperament of the athletes themselves, the particular epidemiology of injury, the initial management following injury, treatment decisions, and rehabilitation.

## EPIDEMIOLOGY

- Injury mechanisms in ES are not well understood, particularly the injury pattern in many sports.
- The highest injury rates in ES are justifiably found in two groups: new and inexperienced athletes who have just started engaging in ES and experienced extremists.
- Reported injury rates in ES may be expected to increase during competition rather than training, a trend well recognized in common team sports as athletes are trying to push their limits even further for prizes, audience, or fame.
- In some ES disciplines, the injury and fatality rates are hard to establish due to a lack of formal recorded events.
- In many situations, the extreme athlete competes against oneself or the forces of nature, and the sport is practiced in relative isolation.
- Unlike expected terrain and environmental conditions that are similar in most traditional sports (i.e., soccer is played on a real or synthetic grass field), comparison of injury rates across ES is difficult given the large variance in terrain and environmental conditions, which often change variably during a single competition or event.

## SPECIFIC EXTREME SPORTS AND THEIR ASSOCIATED INJURIES

### Skydiving

- Skydiving is a major air sport of parachuting from an aircraft.
- It can practiced both competitively and recreationally, with over 5.5 million jumps performed annually by over a million jumpers worldwide, including tandem jumps.
- Injury rates are relatively high in sport skydiving.
- Majority of the collective total number of jumps is performed by a significantly smaller number of sports skydivers, whereas a larger number of participants perform fewer jumps each.
- Recent fatality estimations are about 1 per 16,300 jumpers and approximately 1 fatality per 88,000 jumps, which increases to 1 per 4,000 skydivers, excluding tandem jumps data.
- About 60% of fatalities are categorized as expert jumpers, while students account for about 20% of fatalities.
- Most fatalities (~70%) occur with the skydiver having at least one functional parachute on, and the majority have been caused by human error.
- Fatalities are mostly related to low or no pull of the parachute (~30%), malfunctions of the parachute system (~15%), reserve canopy problems (~15%), midair collisions (~20%), and landing errors (~20%).
- Fatalities are more common in experienced jumpers in their 4$^{th}$ to 5$^{th}$ decade of life and with on average 11 years of experience in the sport.
- More than half of fatalities occur in jumpers holding the highest parachute license (USPA D-License).
- Injury rates in skydiving are around 170 per 100,000 jumps, with only about 30% of these requiring a visit to an emergency department, and as few as 10% necessitating hospital admission.
- About two thirds of injuries in skydiving are minor, with around a third of these commonly being abrasions and contusions. Lacerations constitute an additional 20%–25% of all minor injuries.
- About 50% of injuries requiring emergency department follow-up treatment involve extremity trauma, with lower extremity injuries dominant (featured in as many as 80% of cases).
- Fracture rates in skydiving are estimated at 0.5 fractures per 100,000 jumps (mostly limbs but also spinal).
- The incidence of nonfatal events is estimated to be around 1 incident in every 2,000 jumps, decreasing to 1 per 3,200 jumps in licensed jumpers.
- About 90% of the nonfatal injuries occur around the landing, with about 50% of injuries involving the lower extremities (Fig. 101.1), about 20% involving the upper extremities (Fig. 101.2), about 20% involving the back and spine, and <10% involving the head.
- Injury severity is normally equally distributed between minor and moderate, with around 40% of each, with severe injuries accounting for a little over 10%.
- Most serious injuries are experienced by licensed skydivers, while students in training have a 6-fold higher injury rate.
- Women seem to be overrepresented, with a higher proportion of landing injuries than men.
- Although many parameters and participants have changed over the last 20 years, injury rates remain similar. Modern equipment

**Figure 101.1.** A hard stop landing in skydiving (or in paragliding), which can lead to a knee ligament injury. (Courtesy of Ori Kuper.)

**Figure 101.2.** An off-balanced landing in skydiving (or in paragliding) can result if a forearm fracture or even a pelvic injury. (Courtesy of Dr. Omer Mei-Dan.)

has decreased overall morbidity and mortality, but it has also led to faster landings with increased limb injuries.

## BASE Jumping

- BASE jumping [**B**uilding, **A**ntenna, **S**pan (a bridge, arch, or dome), and **E**arth (a cliff or other natural formation)] is a sport that developed from skydiving and uses specially adapted parachutes to jump from fixed objects (see Fig. 101.1).
- The sport is estimated to involve only about 2,500–3,000 active members worldwide but is considered among the most dangerous adventure sports in the world, and significantly more dangerous than skydiving, as BASE jumps are performed from much lower altitudes (often less than 500 feet above ground level).
- The sport appears to predominantly attract male participants.
- It has been banned from many popular launch sites, such as the Eiffel Tower in Paris and in many national parks in the US.
- Lower falling speeds with far less aerodynamic control and a high risk of losing flying stability leave little room for error. If the parachute is deployed while the jumper is unstable, there is a high risk of entanglement or malfunction. In such cases, the single canopy used may also be facing the wrong direction, which may not be problematic in skydiving.
- Off-heading opening of the parachute resulting in an object strike is the leading cause of serious injury and fatality in BASE jumping.
- As the sport takes place in close proximity to a cliff, a tower, or a building which is used as a jumping platform, the risk of collision with an object in BASE jumping is increased (Fig. 101.3).
- BASE jumping is associated with a 5- to 8-fold greater risk for fatality or injury when compared to regular skydiving.
- The fatality rate associated with BASE jumping was found to be 0.04%, although information on demographic characteristics or jumper experience levels was lacking.
- Several studies show that estimated injury rates are around 0.4%.
- The majority of accidents (>50%) involve the lower limbs, about 30% involve the back/spine, <20% involve the upper limbs, and about 3% are head injuries.
- Injury severity varies according to topographic conditions and altitudes involved, with higher altitudes (1,000 m and above) offering relatively safe jumping conditions, allowing for greater speed generation before parachute deployment and controlled landing, whereas lower altitude jumping sites could result in more severe injuries.

**Figure 101.3.** BASE jumping off a tall building. (Courtesy of Dr. Omer Mei-Dan.)

**Figure 101.4.** An aerial maneuver in surfing. (Courtesy of Shiran Valk.)

- The rate of injuries requiring hospitalization in BASE jumping is estimated at around 300 per 100,000 jumps and is sixteen times higher than the estimated rate of such injuries in freefall skydiving.
- Recently, a growing pattern of wingsuit-related fatalities has been shown in BASE jumping. It seems most wingsuit-related fatalities are attributed to cliff or ground impacts, mostly as the result of flying path miscalculations.

## Surfing

- The sport of wave surfing is ever growing, involving a huge market, commercialization of surfing apparel and the surfing lifestyle, fashion trends, and media coverage (Fig. 101.4).
- In 2009, it was estimated that there were more than 2.4 million surfers in the United States.
- There are four main board categories: longboards, shortboards, SUP boards, and tow-in boards.
- Surfing is considered relatively safe compared to more traditional sports, with estimates ranging between 2 and 3.5 "moderate to severe" injuries (resulting in lost days of surfing or requiring medical care) per 1,000 surfing days and around 0.25 injuries/surfer/year.
- The most common injuries requiring medical attention or resulting in AN inability to surf are lacerations (around 40%) and soft tissue injuries (around 35%).
- The majority of acute injuries are caused by striking either a surfboard (a sharp fin, the tail, or the nose of the surfboard) or another surfer, with the remainder caused by the sea floor.
- Lacerations, sprains, and contusions are the most commonly encountered injury types, but fractures and dislocations are also prevalent, with an estimated incidence of 10% for each.
- Injury rates in competitive surfing (professional and amateur) are higher, estimated at almost 6 per 1,000 athlete exposures, or 13 per 1,000 hours of competitive surfing, with >6 significant injuries per 1,000 hours of competitive surfing. In this regard, surfing rates favorably compared to injury rates in American collegiate football (33 per 1,000 hours), soccer (18 per 1,000 hours), and basketball (9 per 1,000 hours).
- The relative injury risk is estimated to be 2.4-fold greater when surfing in waves overhead or bigger, and 2.6-fold greater when surfing over a rock or reef bottom.
- More than one-third of acute injuries involve the lower extremities, and similar rates are found with relation to the head and neck.
- A considerable proportion of head injuries is found in surfing, possibly related to the fact that very few surfers use protective headgear.
- Fatality rates are not well documented in surfing. However, they have been reported in low numbers in some ocean-related drownings.
- As 50% of a surfer's time is spent paddling, and another 45% spent remaining still, while only 3%–5% is spent actually riding waves, most overuse injuries derive from paddling.
- Overuse injuries in surfing are mostly found in the shoulder (~20%), back (~20%), neck (~10%), and knee (~10%).
- Injury prevention in surfing is practiced by following basic safety recommendations, such as maintaining adequate swimming skills (the ability to swim 1 km in less than 20 min and being comfortable swimming alone in the ocean), being familiar with the surfing environment and conditions (entry and exit points, currents, and underwater hazards), avoiding surfing to exhaustion, and safely practicing breath-holding training.
- Using adequate equipment is also essential, including temperature-appropriate wetsuits protecting against hypothermia, as well as protected, rounded, and shock-absorbing surfboard noses and fin trailing edges. A board leash should be used to keep the board close at hand, and the board can be used as a flotation device should a surfer become exhausted or injured.

## Paragliding

- Paragliding is a recreational and competitive flying sport.
- It is defined as a sport using a single seater, nonmotorized, foot-launched flexible aircraft that is steered aerodynamically and able to start from ground level without requiring a free-fall phase.
- The paraglider, an advanced form of the parachute, consists of an upper and lower sail, with "ribs" dividing it into numerous separate compartments that are stabilized by air pressure.
- Accurate maneuvers are enabled by using two steering lines attached to the rear corners of the parachute.
- Despite not having an engine, paraglider flights performed by an experienced pilot can last many hours and cover large distances (up to many hundreds of kilometers).
- The paraglider pilot also has the ability to gain altitude using air thermals, often climbing to a few kilometers over the surrounding countryside/geographic surface.
- Paragliding accidents present a completely new injury pattern, not comparable to injuries associated with other air sports or even ground traffic accidents.
- Most accidents are caused by pilot errors, unpredictable meteorologic changes, or an incorrect appreciation of environmental conditions.
- Accidents may occur in difficult terrain, often making rescue operations beyond the capacity of ground rescue services, necessitating helicopter rescue operations.
- The spine is considered the "Achilles heel" of the sport, sustaining the majority of impact during accidents and consequent injuries.
- **Spinal injuries** are relatively common with an incidence rate ranging from 25% up to around 50%.
- The thoracolumbar transitional region is most commonly involved (T12–L1).
- Spinopelvic dissociations can occur at a higher odds ratio than in the general trauma population, and even pelvic injuries can occur in up to 19%.
- **Lower extremity injuries** are also relatively common, with an incidence rate ranging from 35% to 54%.
- The ankle is the most frequently involved region, with relatively high rates of fractures, fracture–dislocations, and ligamentous injuries, often requiring surgical management.
- **Upper extremity injuries** occur in up to around 20% of all injuries: mainly distal radius fractures, acromioclavicular separations, shoulder dislocations, elbow fracture–dislocations, and soft tissue injuries.

- Head injuries are also encountered at rates up to 24% and could be fatal. Thoracic and abdominal injuries are also encountered, and while less common, may pose an immediate fatality risk.
- Fatality rates are very low, ranging between 0.03% and 0.06%.
- Most paragliding accidents are the result of pilot error.
- Other important factors are an awareness of potential risk factors, unexpected weather conditions, and the level of training and experience. Equipment failure is a rare cause.
- Beginners and recreational pilots with less than 100 flights are the most accident-prone group, especially during takeoff and landing, compared to reasonably experienced pilots with up to 200 flights.
- The 2 years immediately after gaining the pilot's license are the most dangerous period, irrespective of the number of completed flights, as well as the length of the training period.
- Paragliding accidents can occur in different phases of flying, from takeoff/launch to landing.
- Takeoff accidents often occur in uneven terrain due to insufficient sail expansion and lift.
- Major spinal injuries can occur in this phase when the pilot is sitting back too early due to overestimation of the lifting airstream, crashing on ones buttocks.
- Inflight accidents are more influenced by weather changes and the pilot's level of experience and skills, and rarely equipment failure.
- Accidents in this phase often result in crashes from a great height, potentially leading to multiple injuries.
- Landing accidents are more common and are primarily a result of forceful landing.
- Causes for landing accidents include misjudgment of conditions, pilot inability to adjust to abrupt changes in the thermals and wind conditions, and other technical errors all leading to incorrect or uncontrolled landing.
- General main causes of accidents are: collapse of the glider, collapse or deflation of the airfoil, incorrect use of the break lines resulting in a stall, and oversteering, or pilot error

## SAFETY REQUIREMENTS AND PREVENTION MEASURES

- Avoid excessive wind speed of over 24 kph (15 mph).
- Avoid wind directions that will not facilitate a takeoff (or landing) into the wind.
- Pilots should avoid tailwind takeoffs at all cost.
- Avoid flying into heavy rain or snow, since the paraglider wing is made from fabric and can absorb moisture, thus affecting the weight of the wing.
- Mandatory routine preflight checks should be performed, with a thorough inspection of all the equipment, including the reserve parachute and protective gear.
- Protective helmets and sturdy footwear reaching above the ankle joint are indispensable pieces of equipment. The use of protective gloves is highly recommended.
- New generation back protection devices provide the best prophylaxis for pilots against pelvic and spinal cord injuries. Foam multichamber and airbag harnesses are considered the best protection against spinal and pelvic fractures.
- Careful prelaunch observation of other pilots in the air to evaluate conditions should be performed.
- Taking advanced courses that simulate different flying incidents, teaching pilots to cope with hazardous situations that may arise during flight, deliberately inducing (under guidance) major collapses, stalls, spins, etc., in order to learn recovery techniques and maneuvers.

## SUMMARY

- ES are increasing in popularity, being fun to participate in and exciting to watch.
- The management of the injured ES athlete is a challenge to surgeons and sports physicians. The margins for error in these sports are small, and athletes as patients are more likely to return to their activities prematurely than the general sporting population following injury.
- A wide variety of disciplines and sports are represented in ES, and this is ever expanding. Future research must progress alongside the development of these sports to allow the sports to be analyzed and made safer without detracting from their extreme nature.

## RECOMMENDED READINGS

Available at www.ExpertConsult.com.

## RECOMMENDED READINGS


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