SPORTS MEDICINE FOR THE PRIMARY CARE PHYSICIAN

Third Edition

Edited by

RICHARD B. BIRRER
FRANCIS G. O'CONNOR

CRC PRESS

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Foreword by Timothy David Noakes

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FOREWORD

While Harold Abrahams was honing his sprinting talents at Caius College, Cambridge University, in preparation for the 1924 Paris Olympic Games, under the watchful eye of his professional Italian coach, Sam Massabini, his elder brother Dr., later Sir, Adolphe Abrahams was well advanced in his medical career. By then he had already served as the first Honorary Medical Doctor to the British Track and Field team at the 1912 Stockholm Olympic Games. So it was perhaps natural that 49 years later, when Sir Adolphe published *The Disabilities and Injuries of Sport,* one of the first English-language textbooks of sports medicine of the modern (post-Second World War) era, the foreword should have been written by his brother, the 1924 Olympic 100m gold medalist.

In his foreword, Harold pays tribute to his elder brother's ability as a "runner quite capable of winning prizes in races from fifty yards to ten miles" so that although the elder Adolphe perhaps "lacked the innate potentiality to reach the top class," yet "he was assuredly unrivalled in his enthusiasm." But no such qualifications were needed to defend the quality and importance of his brother's writings. Thus Harold's opinion was that although "… many books and articles have been written about injuries incurred in sport … most of these seem to me to suffer from the shortcoming of attempting to be academic scientific treatises and to neglect the practical details." But Adolphe's book was different: "This book breaks new ground, for not only are the injuries which may be encountered in athletics and sport generally considered with the proper regard for their nature, causation, diagnosis, and treatment, but for the first time disabilities that athletes meet, such as possible injuries to vital organs, sickness, stitch, cramp, and the like are fully examined. The author writes with the pen of the scientist, but in language which will be easily understood by the lay reader." Finally Harold concludes: "That this is so should surprise no one, since he has qualifications for his task which must be unique. … Many an international athlete would hasten to pay tribute to the part which the author has played with his knowledge, advice, and understanding, in their success." This was praise indeed from the first British Olympic runner to accept professional training support; an intellectual shift that brought the younger Abrahams into blunt conflict with the prevailing code of scrupulous amateurism of his college, university, and nation.

Sir Adolphe's slim 93-page textbook first came to my attention only many years later, occasioned perhaps by the enthralling story of the athletic experiences of his brother and Scottish missionary Eric Liddell, so faithfully captured in David Putman's classic 1981 movie, *Chariots of Fire*. The Abrahams were clearly unusually talented brothers.

In retrospect, whatever its worth in 1961, the great value of Sir Adolphe's book for modern scholars is that it provides one measure of the state of sports medicine experienced by a leading practitioner, at least in his part of the English-speaking world, in the first half of the 20th century. There is certainly good evidence, developed especially in John Hoberman's compelling book, *Mortal Engines: The Science of Performance and the Dehumanization of Sport*, that sports medicine was more advanced in Germany and France than it was at that time in the English-speaking countries, including Britain and the United States.

But for Britain certainly and perhaps also for North America at that time, *The Disabilities and Injuries of Sport* was probably the benchmark. Yet with all the compassion in the world, *The Disabilities and Injuries of Sport* can really only be described as "quaint;" perhaps little better than a simple first aid booklet of the modern era. Although Sir Adolphe establishes the tradition that it is best to live your subject if you are to know it properly, his book falls some way short of being the proverbial "Bible." From the security of the 21st century, it is heartening to see how far the profession has progressed in the past 50 years.

A year after *The Disabilities and Injuries of Sport* was published, the first edition of the British *Sports Medicine***,** co-written by English authors John Williams and Peter Sperryn was released. It is said that Williams had written the book on the basis of his early experiences gained from directing the first dedicated sports injury clinic in the United Kingdom. It would be the last time a textbook of sports medicine would be attempted by only two authors, for there was a clear acceleration in the

rate at which sports medical knowledge was developing in the English-speaking world.

The first sports medicine textbook that I read in detail enjoyed the same title, *Sports Medicine,* but was co-authored by the North Americans Fred Allman of Atlanta, Georgia, and Allan Ryan from Madison, Wisconsin, the latter the first editor of a journal, *The Physician and Sports Medicine*, that achieved great influence under Ryan's editorship. The book written with 17 co-authors significantly expanded the perceived scope of sports medicine by including chapters covering the medical supervision of the athlete including medical and orthopaedic care; the care of those requiring special physical education; and preventive medicine and therapeutic exercise. It introduced the concept that sports medicine includes the promotion and prescription of exercise for the treatment and prevention of disease, a concept that would set the trend for the further expansion of sports medicine in the final quarter of the 20th century.

Two years later, the second edition of the British *Sports Medicine* was released. It represented a giant leap forward for European sports medicine. It also included multiple co-authors who produced a book "based on the concept of Sports Medicine as an integrated multi-disciplinary field embracing the relevant areas of clinical medicine (sports traumatology, the medicine of sports, and sports psychiatry) and the appropriate allied scientific disciplines (including physiology, psychology, and biomechanics)". Despite the greatly increased scope of the book, the editors still felt that "it is clearly impossible to cover the whole subject in such a way as to satisfy each potential reader." An interesting aspect of this text was that, unlike its North American equivalent, it was silent on the role of exercise prescription for the prevention and treatment of disease. Rather, the full emphasis was on the care of those who are already active, usually at the higher levels of competence and commitment. Another fascinating difference was that whereas Ryan and Allman's text contains 22 index references to the word *female*, neither the word *female* nor *woman* appears in the index of the British text written by Williams and Sperryn.

All these publications are important because they provide an infallible measure of the growth of sports medicine in the past half-century since the publication of Sir Adolphe's original. And so it is from this historic tradition that the third edition of *Sports Medicine for the Primary Care Physician* makes its welcome appearance.

Written for those primary care physicians who provide first-contact care for the physically active, this multi-authored text follows the North American tradition of providing a comprehensive resource for those working at all levels of sport, whether it be with professional athletes or with those whose exercise is confined to the weekends. The third edition remains faithful to its forerunners in that the goal of its editors remains unashamedly to provide physicians with the information that will assist in the promotion of the 1990 United States National Objective for increased levels of physical fitness and exercise.

In my view, this will become an increasingly important role for sports medicine in the coming decades and will begin to outweigh the more utilitarian, albeit currently more alluring, role of providing expert medical care to the sporting elite. That this goal was already established by Dr. Birrer in the first edition of this text, published in the early 1980s, is a tribute to his vision and unwavering commitment. This concept extends beyond the Olympian goals to which the Abrahams brothers and many others aspire, and which is the true measure of the new maturation of this discipline.

The text is current and concise yet sufficiently inclusive to provide the background information required confidently to practice this discipline. This must be so since the chapters are contributed by those whose expertise has been acquired by working at the coal face, learning, as did Sir Adolphe, by living their profession. The scope is comprehensive with equal weight given to the medical and orthopaedic requirement of the profession. Special features are the chapters devoted to the needs of specific groups of exercisers - runners, swimmers, throwers, and children, among many others. There is also a surfeit of information (22 index entries) about the special sports medical requirements of that 50% of the population who are female.

So where is sports medicine at the birth of the 21st century?

Study this textbook and marvel.

Sir Adolphe and his brother would have approved.

Timothy David Noakes, MBChB, MD, DSc

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PREFACE

"And is not bodily habit spoiled by rest and idleness but preserved for a long time by motion and exercise?"

Plato, ***Theaetetus***

Exercise in the natural form of work was a necessary and regular part of life before the Industrial Age. With the advent of work-saving machinery, people turned more and more to the "sedentary studious life."[1] Despite the clairvoyant exhortations of some of our nation's political leaders (Thomas Jefferson and Benjamin Franklin) and the world's medical giants (George Cheyne and Joseph Addison), the "pamper'd Race of Men" prospered.[2] The tempo of the past several decades has shown the wisdom and benefits of regular exercise. Serendipity has played a significant role in the observations that physically active individuals do not suffer proportionately from atherosclerosis, strokes, myocardial infarctions, and other chronically debilitating diseases as do their sedentary counterparts. Rudimentary exercise research has begun to yield the secret successes of fitness training. Established health benefits of regular physical activity include a reduction of coronary heart disease, hypertension, non-insulin-dependent diabetes, colon cancer, anxiety, depression, and all-cause death rates; maintenance of appropriate body weight and functional capacity; and increased bone mineral content. The general sociocultural swing back to naturalism, homeopathy, holism, and self-help has embraced exercise and sports activities as a necessary component and has once again brought us full circle to our ancestors' philosophies.

Because the world of sports and recreational activity encompasses all ages and both genders, as well as the entire health spectrum, it is fitting that the primary care physician should be the front-line sports medicine specialist. It is this individual who first sees the injured athlete at home, on the field, or in the office. Very often the physician recognizes the family or personal problem affecting a person's performance. Above all, it is the primary care physician who is best suited to integrate the patient's work, sport, family, and school environment so that maximum exercise potential and function under the safest health conditions can be realized.

The first edition of this book sprang from the Society of Teachers of Family Medicine sports medicine panel in 1980. This invigorating panel and its chairman, Daniel Garfinkel, M.D., designed the sports medicine curriculum for the family practice programs of this country. The second edition moved to a problem-oriented, field-side format and became a practical authoritative guide for primary care physicians involved in the study and practice of sports medicine. This third edition brings further evolutionary changes, including several new chapters (complementary/alternative sports medicine options, the radiologic evaluation of the athlete, and a revised and streamlined medical illness section) and incorporates the growing science of evidence-based medicine. In addition, Francis O'Connor, M.D., a recognized family physician who is a sports medicine authority, becomes the associate editor.

While there are a number of professional resources for evidence-based medicine, this third edition utilizes a grading system described in the *American Family Physician* in 2002.[3-5] *Level A* evidence is derived from a high-quality, randomized, controlled trial (RCT)/meta-analysis using comprehensive search strategies. *Level B* evidence is from a nonrandomized, well-designed clinical trial utilizing a nonquantitative systematic review with appropriate search strategies. *Level C* evidence represents expert opinion as consensus viewpoint.

Much work remains to be done in achieving the 2010 national objectives for physical fitness and exercise.[6] While the number of employer-sponsored fitness programs has significantly increased and the majority of primary care physicians include a careful exercise history as part of their initial examination of new patients, fewer than 50% of children ages 10 to 17 regularly participate in appropriate physical activities, particularly cardiorespiratory fitness programs that can be carried into adulthood. Only 10 to 20% of adults ages 18 to 65 are participating in vigorous physical exercise, and fewer than 8% of adults 65 years or older engage in appropriate physical activity (i.e., regular walking, swimming, or other aerobic activity). Only 5% of adults can accurately identify the variety and duration of exercise thought to promote cardiovascular fitness most effectively. Over 60% of adults

and up to 15% of children are overweight or obese. These are profound challenges that must be addressed in the next decade through clinical practice and scholarly research. The message is clear: "Exercise is the easiest way to preserve health."[7] We are the medium. This textbook is one small effort in making the message happen. Read, learn, enjoy, and profit.

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ABOUT THE EDITORS

Richard Birrer received his M.D. degree from Cornell University Medical College in 1975. He did his internship in family medicine at the Hunterdon Medical Center in Flemington, New Jersey, and completed his training at the State University of New York, Health Sciences Center of Brooklyn. As chief resident he was the recipient of the Parke-Davis Teacher Development Award. In 1982 he completed a Master of Public Health degree in international health policy and management from Harvard University. He was a senior Fulbright scholar in 1984, introducing the principles and practice of family medicine in Egypt. He was chosen as a Health Exchange Scientist to the former U.S.S.R. under the Fogarty program in 1985 and became Salzberg Fellow in 1986. He is the CEO and President of St. Joseph's Healthcare System, Inc. He is Professor of Medicine at Cornell. He is a diplomat and fellow in the specialties of family practice, sports medicine, geriatrics, emergency medicine, and medical management (certified physician executive). Dr. Birrer is the author of more than 300 peer-reviewed articles and monographs and 10 textbooks and serves on a number of editorial boards.

Francis G. O'Connor graduated from the United States Military Academy, West Point, New York, in 1981, and received his medical degree from the Upstate Medical Center, Syracuse, in 1985. He completed a residency in family medicine at St. Joseph's Hospital Health Center in Syracuse, and then entered the military where he became Chief of Family Medicine, Walson Army Community Hospital, Fort Dix, New Jersey. He completed a sports medicine fellowship in 1992 with Dr. Robert Nirschl at the Virginia Sports Medicine Center, Arlington Hospital. He currently is the director of the sports medicine fellowship program, Uniformed Services University of the Health Sciences, Bethesda, Maryland, where he is an associate professor of family medicine. He is a fellow of the American College of Sports Medicine and a board member with the American Medical Society of Sports Medicine and the American Medical Athletic Association. He has authored nearly 40 peer-reviewed articles and monographs and is the co-editor of *The Textbook of Running Medicine*. He has a special interest in running medicine and exercise-associated collapse/sudden death. He is a senior medical consultant to the Army Ten Miler, Marine Corps Marathon, and DC Marathon.

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CONTENTS

SECTION ONE
GENERAL SPORTS MEDICINE

PART I
THE ROLE OF THE PHYSICIAN IN SPORTS

1 THE HISTORY OF SPORTS MEDICINE

Richard D. Smith and Richard Levandowski

The first application of medicine to sports is unknown, but the earliest recorded example of a sports-contest-related injury is the Biblical account of Jacob's wrestling match (Genesis 32: 24–32). Jacob received the Lord's blessing but apparently no medical attention: He passed the place called Penuel "limping because of his thigh." An early Egyptian surgical treatise (ca. 2600–2200 BC) describes treatment for a variety of injuries, many of which could have been sustained in sporting contests.[1]

The first known treatise on athletic training, written in approximately 444 BC by pentathlon champion Iccus of Tartum, is now lost but mentioned in extant texts. The first sports medicine professional may have been Herodicus of Selymbria, a contemporary of Socrates. The pioneer of "medical gymnastics," Herodicus insisted that athletic training be systematized, balanced in exercise and diet, and have a medical component. Both Iccus and Herodicus recommended massage, as do many modern trainers and sports medicine professionals.[2]

Even in ancient times, it was evident that natural talent alone could not win the laurels against talent improved by training.[3] Athletes of the Greek classical period were required to train under supervising physicians (*gymnastes*) 10 months before competing in the Olympics.[4] But, skepticism remained about applying medical knowledge to sport. Plato himself, writing in a later era, characterized Iccus and Herodicus as "gymnastic sophists."[5] In his *Gymnastikos*, Philostratus the trainer accused physicians of allowing athletes too luxurious a diet and not demanding they bathe in cold mountain streams and sleep on the ground. (Philostratus was not a mindless taskmaster, however. He also offered cautions on heat exhaustion and overtraining.[4])

By the late classical period, treatment of certain athletic injuries had become quite specific. Quintus of Smyrna (ca. fourth century AD) described the treatment of ankle sprains with ointment and dressings and boxing wounds by draining, suturing, and applying topical medications.[4]

The famous second-century Roman physician Claudius Galen, whose work exercised an influence well into the Christian era, was a true sports medicine practitioner. Galen had been appointed physician to the gladiators in Pergamum by Pontifex Maximus and subsequently published his methods of treating injuries suffered in the arena.[2] He also advocated weight lifting and other resistance exercises as a means of promoting health.[6]

The Greco–Roman linkage of exercise and health influenced philosophers and physicians into the medieval and Renaissance periods. For example, Bergerius (1370–1440) and later Montaigne espoused the classical notion of regular exercise as part of children's education.[7]

The first illustrated book on sports medicine has been credited to Gerolamo Mercuriale (1530–1606), a distinguished professor of medicine at the universities of Padua, Bologna, and Pisa. His *Artis Gymnasticae apud Antiquos Celeberrimae nostris Temporibus Ignoratae* (1569) contains woodcuts showing rope climbing, medicine ball exercises, and "activities for women." Mercuriale considered the relationships of medicine to sports and exercise to health and discussed classical attitudes toward them.[8]

Although Greco–Roman traditions exerted the most influence on Euro–American sports medicine, classical Indian hatha yoga and Chinese gung fu systems (the latter encompassing methods of exercise, health, and energy regulation as well as self-defense) may represent the earliest systematizations of exercise therapy, as witnessed by the texts of the Hindu Atharva-Veda and the Chinese book of gung fu written around 1000 BC.[2]

The writings of Hakim Ibn-e-Sina (979–1037), known in the West as Avicenna and the father of

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Islamic medicine, excited great interest during the cultural exchange of the post-Crusade period. Many of Hakim Ibn-e-Sina's recommended treatments would be familiar to today's sports medicine professionals: massage, movement exercises, and warm baths to promote rehabilitation from injury.[2] But, from the eighteenth century until well into the twentieth, most European, British, and American sports medicine providers were not doctors but self-taught trainers. A physician might be called in the event of injury, but most learned persons seemed to have lost interest in applying medical knowledge to athletics.[9] Attitudes toward the body itself had changed, and the concept of *mens sana in corpore sano* was no longer dominant. Many schools of philosophy agreed that the mind and body were separate entities, and dominant religious views considered the body an object of contempt, a source of temptation and weakness.

Benjamin Franklin practiced resistance exercises and measured their effect on his cardiovascular system. Writing in 1772 from London to his son, he reported that, "The dumbbell is another exercise of the latter compendious kind. By use of it, I have in forty swings quickened my pulse from sixty to one hundred beats in a minute, counted by a second watch; and I suppose the warmth generally increases with quickness of pulse."[6]

By the mid-nineteenth century, classical attitudes about the contribution of physical development to intellectual and moral development reasserted themselves. Physicians on staff and faculty at major universities became directly involved with athletics. Credit for the advent of collegiate sports medicine can be given to Edward Hitchcock, M.D., who was appointed instructor of physical education and hygiene at Amherst College in 1854. Hitchcock instituted anthropomorphic studies of the students, published a textbook and 161 articles on topics in athletics and sports, and was probably America's first college team physician.[2]

Other landmarks of this transitional period included English physician John Morgan's mid-nineteenth century study of the longevity of former oarsmen as compared to the general population; an 1899 paper in the *Boston Medical and Surgical Journal* by E.A. Darling of Harvard entitled "The Effects of Training," which studied the fitness of Harvard crews; and Augustus Thorndike's 1938 textbook *Athletic Injuries, Prevention, Diagnosis and Treatment*, considered to be the first general American text of sports medicine.[2]

At the time of the 1896 revival of the Olympic games by Baron Pierre de Coubertin, however, training was still dominated by former athletes and not medical professionals. Greek doctors were in attendance at the first modern Olympiad in Athens, following the marathoners in ambulances and tending to exhausted runners, but no formal provision was made for medical attention for the athletes.[4]

The death of Portuguese runner Lazaro from heat stroke after the 1912 Stockholm marathon led to the requirement that all marathoners undergo a physical examination, the first such requirement in modern Olympic history; however, the United States did not send official trainers with its team until the 1920 Antwerp games, nor a team doctor and nurse until the 1924 Paris games.[4]

SPORTS MEDICINE ORGANIZATIONS

In 1928 at the St. Moritz Winter Olympics, 33 physicians met to prepare for the establishment of an International Congress of Sports Medicine at the summer games and to create a permanent International Assembly on Sports Medicine, which later became the Federation International Medico-Sportive (FIMS). Other international groups that have advanced sports medicine were founded over the succeeding years. These include the Conseil International du Sport Militaire (established 1948), the South American Congress on Sports Medicine (1953), the UNESCO International Council of Sport and Physical Education (1960), the Groupement Latin de Medicine Physique et Sport (1961), and the International Committee for the Standardization of Physical Fitness Testing (1964).

In the United States, the National Athletic Trainers Association (NATA) was created in 1949. In 1954, the American College of Sports Medicine (ACSM) was founded, and the American Medical Association (AMA) created the *ad hoc* Committee on Injuries in Sports. The AMA followed up with a Committee on Exercise and Physical Fitness in 1964. Serious academic study of sports medicine was boosted when the ACSM commenced publication in 1969 of the *Journal of Medicine and Science in Sport* and first published the *Encyclopedia of Sport Science and Medicine* in 1971.

In 1962, the American Academy of Orthopedic Surgeons established the Committee on Sports Medicine. The American Academy of Family Physicians,

the American Academy of Pediatrics, and the American Physical Therapy Association also have committees on sports medicine. Other important groups in the field of sports medicine and physical fitness are the American Association for Health, Physical Education, Recreation, and Dance; the American College Health Association; the Women's Sports Foundation; and the National Strength and Conditioning Association.

A landmark in U.S. government efforts to promote physical fitness through sports came in 1956 with President Eisenhower's establishment of the President's Council on Youth and Fitness. It was reorganized under President Kennedy and renamed the President's Council on Physical Fitness and Sport under President Johnson. In 1978, Congress established the Office of Physical Fitness and Sports Medicine with passage of Public Law 95-626 and also passed Public Law 95-606, the Amateur Sports Act.

In 1978, the U.S. Olympic Committee instituted the Volunteer Physicians Program to prepare primary care and orthopedic physicians for working with American athletes at international competitions, including the Olympics. The same year, an active sports medicine department was founded at the Olympic Training Center in Colorado Springs. A sports medicine department was opened at the Lake Placid training center in 1982 and one at the San Diego training facility in 1994.[10] Today, the majority of national sports governing bodies have sections on sports medicine, and most states and cities have established councils on physical fitness.

RECENT DEVELOPMENTS AND FUTURE TRENDS

Contrary to earlier views, research and practical experience have demonstrated that vigorous participation in athletics is as safe for women as it is for men.[11] Performance levels for both men and women have risen to the point where yesterday's world records are often today's qualifying marks. Sports medicine has been called upon increasingly not only to heal athletes but also to make them — as the Olympic motto exhorts — swifter, higher, stronger. As a result, ergogenics has become a field for intense research.[12] Negative publicity about steroid experimentation had tainted the Soviet bloc's work in sports medicine, but with the successes of the Eastern Europeans and the emergence of the People's Republic of China as an Olympic power, the results of biomechanical research done in the Communist countries and the priority given to involving sports medicine specialists in every stage of their athletes' development have come to Western notice.[13,14]

Another major boost for sports medicine occurred in the 1970s when fads for tennis, running, weight training, and aerobics proved to be lasting trends. The personal involvement of such physicians as Allan Ryan and George Sheehan helped spread information on exercise, as well as the increasing number of exercise-related injuries being seen by primary care providers.

Predictions are risky, but the sports medicine of the future — like the sports medicine of classical times — will probably be as concerned with conditioning, performance, nutrition, and skill development as with treating injury, and other trends are likely: a continued increase in sports participation by the general population, including children and the elderly; demands for standardization and certification of sports medicine training programs; the challenge of bringing the latest knowledge to the attention of non-elite athletes; and, of course, the need for the primary care physician to have expertise in sports medicine.

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2 THE SPORTS MEDICINE PHYSICIAN

Richard B. Birrer

One of the most enjoyable aspects of primary care is providing care to active children and adults and encouraging activity in those who are not. The biology of the human organism requires physical activity for optimum performance. Competent clinician, inveterate enthusiast, and consummate diplomat describes the prototypical picture of a "team doc." Sports medicine for a local grammar school, high school, or college is a natural extension of a primary care physician's practice. More than 30,000 physicians now serve as team physicians or medical consultants for teams or school systems. It is estimated that 85% of those physicians in this capacity are primary care physicians.

Certainly, primary care physicians will see sport- and recreation-related injuries throughout their clinical careers. Family physicians, pediatricians, internists, adolescent medicine specialists, and generalists are best suited as team physicians to care for the needs of the active individual. Non-primary care physicians (e.g., those in emergency medicine, orthopedics, obstetrics/gynecology) should be paired with a primary care physician in order to provide comprehensive, continuous coverage. Balancing the needs of the athlete, team, and institution and its representatives is the ethical responsibility of the sports physician.

THE SPORTS MEDICINE TEAM

The primary members of the sports medicine team are the athlete, physician, coach, and athletic trainer (Figure 2.1). Secondary members include support systems for the athlete's family, friends, and teammates; clinical consultants (e.g., dentist, podiatrist, orthopedist, nutritionist, psychologist), administrative support; and officials. The athlete is the focal point of the team. The team is as strong as its weakest member and sinks or swims based on its dynamic equilibrium being maintained through communication. It is helpful to view the sports team metaphorically as a family. In this mobile, heterogeneous, ever-changing, industrial society, the sports team represents a valid surrogate family. The 1979 Pittsburgh Pirates world championship baseball team proudly referred to themselves as "The Family." The structure of most athletic organizations generates a strong emotional base; therefore, the prototypical roles of the nuclear family appear time and again. In a metaphoric sense, the manager or coach will be viewed as a father or other authority figure. The athletic trainer typically plays the role of a nurturing mother, and the assistant coaches play the role of big brothers or big sisters. Team members represent siblings, and ancillary personnel and cheerleaders can be considered the extended family. Putting the team in this perspective is helpful in two ways: (1) it facilitates an understanding of the emotional and organizational relationships among team members, and (2) it provides a working perspective for the team physician.

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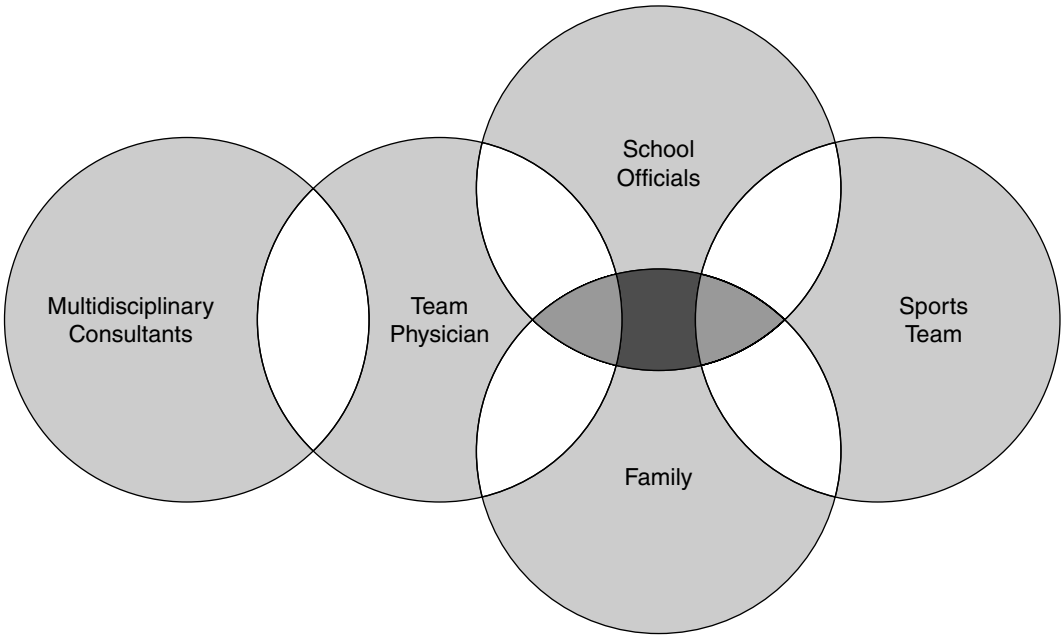

Figure 2.1 The sports medicine team.

ATHLETIC TRAINER

A certified athletic trainer is available in about 35% of high schools.[1] The trainer's role is to promote safety by preventing and caring for athletic injuries and coordinating the school athletic program.[2,3] The physician's assistant, the athletic trainer, should hold certification from the National Athletic Trainer's Association and the American Red Cross advanced first aid and basic life support (BLS) courses. In conjunction with the physician, the trainer designs, coordinates, and implements the athletic training and prevention program, obtains pertinent medical history, and maintains current injury profiles. The team trainer should have a working knowledge of taping, wrapping, padding, and bracing; should be able to recognize and evaluate injuries; and should be prepared to render appropriate first aid if the physician is not present. The athletic trainer should make decisions regarding further athletic participation and ensure that athletes with significant injuries receive appropriate care. The athletic trainer supervises the transportation and rehabilitation of injured athletes and stays in close contact with the team physician at all times. The athletic trainer serves as the liaison among the team, athlete, athlete's family, physician, and coaching staff. Because the athletic trainer is the physician's agent, guidelines and rapport are paramount, as negligence on the part of the trainer will often be attributed to the doctor.

COACH

Responsibilities include the general care and well-being of athletes through solid leadership and coaching, ensuring a proper playing environment, and preparticipation screening. Certification in advanced first aid and BLS through the American Red Cross is mandatory. In the absence of an athletic trainer, the coach's role in the prevention and care of injuries is proportionately greater. A good coach reinforces the rules and reaffirms the value of sportsmanlike conduct. Modern weight-training methods and a ban on performance-enhancement agents are promoted. Proper preseason clearance, injury and rehabilitation follow-up, and return-to-play criteria are enforced. Playing conditions (e.g., temperature or humidity) and scheduling and canceling practices or competitions are under the purview of the coach. Periodic inspections of equipment and training areas and emergency planning are the responsibilities of the coaching staff.

OFFICIALS

The officials are responsible for consistent implementation of stipulated rules and regulations during competitive sports. Duties include pregame preparations (equipment, weigh-ins, and playing

field conditions) and game officiating (conditions or injuries). In non-professional sports, a liability policy may be appropriate.

TEAM PHYSICIAN

The term *sports physician* is a general term referring to a physician who cares for individuals involved with any sporting or recreational activity. This involvement may entail only preparticipation clearance, weekend games, and/or the care of athletic injuries in a clinical setting. The term *team physician* is defined by specific requirements and responsibilities.[4] The team physician plays a very influential role in the sports community and is expected to provide not only physical aid but also philosophical and psychological counseling to athletes who may be concerned about poor sports performance, schoolwork, or career and psychosocial issues. The comprehensive, continuous care provided by primary care physicians makes them uniquely qualified to be a natural complement to the sports team "family" and cornerstones of sports medicine teams. The average number of years served in this role at the amateur level is 11.

REQUIREMENTS[4]

How do you provide the best medical care for athletes at all levels of participation? The sports physician must have a medical degree (M.D. or D.O.) with an unrestricted license to practice medicine and surgery in the state of record and be in good standing with the state department of health and licensing board. Board Certification in a specialty and the Certificate of Added Qualification in Sports Medicine (American Boards of Family Practice, Internal Medicine, Pediatrics, and Emergency Medicine conjoint sponsorship since 1993) is recommended. The physician must be certified in BLS and have a working knowledge of sports emergency and trauma care with particular proficiency in the management of musculoskeletal injuries and medical conditions encountered in sports. Additional training (e.g., fellowship, continuing medical education) and certification in sports medicine, a significant clinical practice of sports medicine (>50%), certification in advanced life support (ALS) and advanced trauma life support (ATLS), membership and participation in a sports medicine society, involvement in academic sports medicine (research, teaching and scholarly publication), and a knowledge base of compensation, disability, and medicolegal issues are desirable. Continuing medical education (CME) is available from local, regional, and national sources, particularly the American Academy of Family Physicians, American Academy of Orthopaedic Surgeons, American College of Sports Medicine, American Medical Society of Sports Medicine, American Orthopaedic Society for Sports Medicine, and American Osteopathic Academy of Sports Medicine. Additional opportunities exist through medical societies, peer review journals, electronic media (CD-ROM, websites), and other organizations such as the U.S. Olympic Committee, National Basketball Association Professional Team Physicians' Society, Association of Ringside Physicians, National Football League Team Physicians' Society, or major league baseball.

Sports physicians should consider writing articles on sports medicine for the lay press and medical journals. The process of identifying a researchable hypothesis, collecting data, and presenting the findings at any of the many sports medicine meetings (American College of Sports Medicine, American Osteopathic Association of Sports Medicine, or American Medical Society of Sports Medicine) promotes continued evolution and refinement of the specialty and enhances the practitioner's knowledge base and skill set.

Being a strong motivator and having excellent verbal and written communication skills are essential. Academic problems can be identified and their management discussed within the context of the sports medicine team. Equilibrium is best achieved by regular weekly meetings; midweek is a good time for meetings, as light practice sessions generally prevail then. The team physician also facilitates and coordinates interactions with a panel of multidisciplinary consultants. In such a manner, warmth and trust are promoted among the team members, allowing for better integrated performance of all members. Equanimity ensures that professional judgment will prevail over personal enthusiasm. Availability (e.g., having a well-organized coverage system) during regular rounds on and off the field (sidelines, training room, office, nights, and weekends) as well as competitions, whether home or away, plus some unstructured time is essential. Finally, involvement in a sporting activity on a competitive or recreational basis provides practical insights into the world of the athlete. Training, competition, and injury at a personal level help one understand the athlete's mindset and promote a healthy lifestyle.

RESPONSIBILITIES

The fundamental responsibilities of the job of team physician require ensuring that athletes are able to participate in a sports activity with the assistance of health care that is confidential, maximizes prevention of and protection from injury, and does not allow those needs to become subordinate to the team or institution.[4,5] At the trainer, coach, team, and institution levels, enjoyment, education, facilitation of success, and protection from liability are the goals.

How do we distinguish a committed sports medicine physician from a physician who covers only official competitions (i.e., a game doctor)? Sports physicians must commit sufficient time and expertise in order to optimize the potential of each athlete under their care. Duties fall into two broad categories: administrative and clinical. Administrative duties include defining roles for involved parties; establishing a chain of command; emergency drill planning and training; event coverage protocols; medicolegal and malpractice responsibilities; assessing environmental or playing conditions, as well as the supply and status of equipment; and regular education of and communication with athletes, athletic trainers, coaches, parents, and officials regarding athlete-related issues.

Clinical duties involve preparticipation screening and clearance; event coverage (grammar school, high school, college, amateur, masters, Olympics), including on-field and off-field management of injuries and rehabilitative and return-to-play processes; proper medical record documentation and security; appropriate preventive counseling and education regarding athletic problems (e.g., substance abuse, conditioning/training, ergogenic aids, nutrition, medical problems); and coordination of all aspects of the sports medicine team. The latter requires ultimately assuming the responsibility of clinical decision making for an athlete's safe participation in a sport. Performance of a sport-specific exam by a physician should be treated as a confidential matter, just as when caring for a private patient.

Coaches and trainers can be taught to do yearly performance tests such as flexibility, strength, agility, endurance, and speed. Preventive guidance includes weight-training programs, defining fluid requirements, and equipment fitting. Coaches' and trainers' knowledge of first aid should be assessed and updated whenever necessary, particularly with regard to basic life support. The training room (Athletic Medicine Unit) must have a separate area with controlled access and appropriate equipment (see Appendix 4).[6] In addition, a crash kit for cardiopulmonary resuscitation (CPR) and a sports medicine bag should be prepared and maintained by the trainer and periodically checked by the physician (Appendix 4). During away events, appropriate equipment and supplies must be available either through regular channels or from the host facility.

PROBLEM AREAS

When defining the relationship of the physician to the team, league, or institution of record, an explicit formal agreement or contract, preferably in writing, should be made, particularly in cases of monetary compensation. Having a written job description and performance criteria is desirable. At a minimum, the physician must have professional autonomy with respect to all clinical decisions. Relationships with colleagues, malpractice coverage, and financial incentives are common challenges facing sports physicians. The physician must be sure that his malpractice policy covers clinical sports medicine issues, including emergencies and trauma, outside the office setting. Most traveling physicians, for instance, are not covered outside their home state. Collegial relationships require regular attention. Communication and follow-up require closure in order to maximize involvement and optimize outcomes. For instance, failure to obtain a consultant's report or lack of feedback to the athlete's primary care physician leaves a palpable gap in the athlete's care in addition to endangering relationships with colleagues. Playability issues require an open, clear discussion in advance of a decision.

Much of a team physician's work at the grammar and high-school levels will be *pro bono*, or on a volunteer basis. Reimbursement for preparticipation clearances and game coverage is usually nominal. Some programs have special support grants available, and compensation can be received for injury evaluation and management. At the college, amateur, and professional levels, insurance coverage is usually available. Community service and the ability to work with young motivated patients provide immense personal satisfaction, and the prestige and credibility within the community afford significant marketing opportunities for practice building.

HANDLING TEAM INJURIES[6]

Athletes are injured on and, more frequently, off the playing field. Informed consent for treatment must be sought at the patient/athlete (or parent/guardian) level, and an injury control

contract or letter of understanding must be established early between the physician and administrative officials that clearly defines and delineates responsibilities and chain of command for all involved parties. It is of utmost importance that the team physician have an emergency response plan for practice and competition in place, with arrangements being made beforehand with the help of the trainer, coaches, and the opposing team's physician. The plan should be based on an integrated medical system that includes preseason and game-day planning as well as postseason evaluation. It should be regularly practiced. The telephone numbers of ambulances, hospitals, physicians, and parents should be readily available, in either the training kit or training room. In some communities the ambulance is visible, in some it is hidden in order to reduce parental apprehension, and in others the emergency medical technicians (EMTs) or paramedics (EMT-Ps) are on short call and respond readily if they are needed. The emergency medical response plan should be reviewed with the medical staff of the opposing team, communication equipment secured and checked, and examination and treatment sites identified. If the team physician has difficulty remembering the players and their health history, it is advisable to have the medical records secured safely and taken along with the team.

Game-day planning includes coordination by the team physician of medical operations, administrative policies, and sports bag/medical supplies. Before actual competition, the team physician is responsible for the final clearance of all injured or ill athletes. On game day, assessment and management of injuries and medical illnesses and return-to-play determinations, including follow-up care instructions and notifications, are essential.

When the physician is not present, the trainer assumes responsibility for the athletic supervision. When at an athletic contest, team physicians should stay in the background and not lose perspective of their primary functions; they must be alert to the action on the field, as injuries can be more easily assessed if the mechanism of injury is known. However, the physician must avoid becoming so wrapped up in the competition that he forgets to be available and prepared to take action. Team protocol requires that only the trainer go out onto the field to assess an injury, thus reducing parental apprehension, especially for minor injuries. If the physician is needed, the trainer should use a prepared signal to call the physician onto the field, at which point the injured player becomes the physician's patient. The physician's initial responsibility involves assessing the extent of injury and deciding if, when, and how to move the player. Because performance outcomes, scholarships, and professional opportunities for individuals and the team may hinge on a medical call, decisions made at this time are critical. If an injury is judged not to be life threatening, the player should be moved to the sidelines for further evaluation. It is unwise to determine the disposition of an athlete who has not had his uniform removed for a thorough examination.

A prompt decision on diagnosis and treatment is essential before the desire to win clouds judgment regarding the athlete's playability. It is not unusual for team physicians to take a helmet away from an injured or overly anxious player in order to prevent the player from returning to the game. On the other hand, it is important to allow the player to return to the game as soon as possible when an injury is minor. A good rule of thumb is "*If in doubt, keep the athlete out!*" The physician's words concerning injuries and playability are final.

The team physician should always follow up on the injured athlete during halftime, immediately after the game, and the following day in the dressing room or private office or by telephone, depending on the seriousness of the injury and whether the player wishes to be consulted. If a serious injury occurs, the team physician should try to accompany the injured athlete to the hospital. Per prior arrangement, the physician for the opposing team, if available, should then be called upon to serve both teams during the game. All those concerned — parents, coaches, and players — should be advised of the location of the hospital to which an injured athlete is to be taken in order to expedite the situation and reduce anxiety. A follow-up visit and notification of appropriate parties are appropriate. Careful documentation of all findings and medical record-keeping are essential. On the field, a portable dictation machine is useful. Transcription and filing of important data are a must and are functions usually overseen by the athletic trainer.

Team physicians must always bear in mind that their demeanor is being witnessed by an anxious crowd of parents and spectators. The ability to act immediately and with authority helps

dispel the fear that often permeates the stands after an injury. Living with the media is a reality; referral of the press to the team coach or trainer allows the physician to perform his duties.

Under the coordination of the team physician, post-season follow-up benefits the care of injured or ill athletes. Medical and administrative protocols are improved, strategies to improve sideline preparedness can be implemented, and the season's illnesses and injuries can be compiled and summarized. Post-season physicals and care, as well as off-season conditioning programs, are appropriate.

CONCLUSIONS

Sports and recreational activities during the formative years help build character in youngsters, provide preventive health care during adulthood, and improve the quality of life in the elderly. Coordinated involvement by primary care physicians can favorably enhance appreciation of the possibilities and limitations of athletics across the entire life cycle. Specifically, a committed team physician can wield a great deal of influence and should use these strengths for the enrichment of the active individual as well as the good of the sport. Success can be measured by a lifelong commitment to sports and recreational activities. For the committed physician, it is an awesome challenge and special privilege.

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3 THE PREPARTICIPATION PHYSICAL EXAMINATION*

Katherine M. Walker and James B. Tucker

INTRODUCTION

The preparticipation examination is an important aspect of athletic competition. Its purpose is to identify athletes with medical conditions that limit participation or predispose to injury, to assess physical maturity and physical fitness, to assess general health and identify health-risk behaviors, and to meet legal and insurance requirements.[1–7] It was not designed to prevent participation and rarely does it do so. A recent study of 2739 athletes resulted in disqualification of only 1.9% from participation in sports.[8] Another study of 596 athletes resulted in only a 0.2% clearance failure rate.[9] The exam should be completed approximately 4 to 8 weeks prior to the beginning of the sports season in order to allow adequate time for rehabilitation and to order further diagnostic tests if necessary.[2–6,10]

At the high school level, all states except Rhode Island require a formal preparticipation physical exam. While most states recommend that physicians perform the examinations, 21 allow medical exams to be done by non-physicians, such as physician assistants and nurse practitioners. Eleven states allow medical professionals with little or no cardiovascular training (chiropractors and naturopathic clinicians) to perform the sports physical.[11]

Some physicians believe that the preparticipation exam should be performed annually. Many authorities, however, believe that a complete exam should be performed with each entry into a higher level of competition (e.g., middle school to high school, high school to college), with interim histories and limited physical examinations completed in between.[3–5,10] The American Heart Association recommends that a complete preparticipation examination be performed for high school athletes every 2 years and for collegiate athletes upon entering the institution. Histories and blood pressure measurements should be obtained in the interim years, and any abnormalities should

* In the second edition, this chapter was authored by James T. Marron and James B. Tucker.

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be further investigated[12] (Level of Evidence 5, expert opinion).

The three main formats of the examination are mass screening, locker room, and individual evaluations. Advantages and disadvantages exist for each. Mass screening is usually more efficient, as it involves many different stations staffed by various health professionals. It has been likened to an assembly line. The stations usually consist of vital signs, medical, and orthopedic stations. Usually the examiners are more sports minded than the average primary care physician and theoretically are more likely to recognize abnormalities than an individual physician because they are responsible for only one section of the physical. When Durant et al.[13] compared both single and multiple examiners, they found that multiple examiners were more likely to detect abnormalities on exam and to recommend further testing. It was postulated that, because each examiner is responsible for only part of the exam in mass screening, he or she is more likely to find an abnormality than an individual performing the entire physical exam.[13] Disadvantages to mass screening include a noisy and hurried environment, lack of a personalized approach, poor communication with the patient's primary care physician and parents, and lack of privacy or confidentiality.[1–7]

A second type of exam is conducted in the locker room. This is probably the least effective, as it focuses more on exam than on history. Privacy is a concern, as athletes are undressed and examined in front of their peers. Confidentiality is also an issue, as usually a private area for questioning is not available. The participants are therefore less likely to disclose personal information regarding health and high-risk behaviors.[4,14]

Primary care physicians are responsible for a large number of individual preparticipation examinations. One major advantage of this style is that the physician usually knows the personal and family history as well as the family setting. The primary physician is more likely to provide continuity of care and has an accessible medical record. Athletes have an established relationship with their primary physicians and therefore may be more likely to confide in them regarding risky behaviors that can affect competition. Many parents and athletes use the preparticipation athletic examination as their only contact with a doctor. The office setting is much better at providing adequate privacy than is mass screening. Unfortunately, this type of evaluation has disadvantages, as well. Often, a primary care physician is less knowledgeable regarding sports requirements than physicians who volunteer to do the exams. Individual exams are also more costly and time consuming than mass screening. Some athletes do not have access to primary care physicians, making an individual exam difficult. Communication among the athlete's primary care physician, coach, and athletic trainer is sometimes poor, making coordination of care difficult.[1–7,10]

HISTORY

The history portion of the preparticipation exam (PPE) is used to identify the majority of medical problems that will affect athletes. The physician needs to inquire about chronic medical illnesses, surgical history, allergies, and current medications, including both prescription and over-the-counter medicines. Answers appear to differ depending on who completes the history form. Carek et al.[15] compared the answers given to historical questions by athletes alone with those given by athletes and parents together. The two groups disagreed on questions related to cardiovascular, neurologic, musculoskeletal, and weight issues. Interestingly, the athletes alone responded with "yes" answers more than their parents. Peltz et al.[16] devised an efficient method of obtaining data via the Internet for athletes at Stanford University. Regardless of how it is collected, the medical history is by far the most important aspect of the preparticipation examination.[1,3,9,10] The Preparticipation Physical Examination Task Force is an excellent guide to use to obtain a medical history as well as for recording the physical examination (see Figure 3.1).[1]

HEENT

Many times the head and neck, eye, ear, nose, and throat (HEENT) portion of the exam is deemphasized. An important aspect of the eye exam is to note any differences in pupil size (anisocoria) at baseline. This becomes important if the athlete sustains a head injury. The physician should also examine visual acuity. An athlete should have corrected vision of 20/40 or better if engaging in collision and contact sports. Protective eyewear should be worn by all athletes participating in sports that are high risk for eye injuries, especially those athletes with poor vision or absence of an eye. The presence of a single eye is a contraindication to participation in sports in which eye protection cannot be effectively worn such as boxing and wrestling. Examination of the mouth may show evidence of bulimic activity and/or

Preparticipation Physical Evaluation

HISTORY **DATE OF EXAM** _____

Name _____ **Sex** _____ **Age** _____ **Date of birth** _____

Grade ___ **School** _____ **Sport(s)** _____

Address _____ **Phone** _____

Personal physician _____

In case of emergency, contact

Name _____ **Relationship** _____ **Phone (H)** _____ **(W)** _____

Explain "Yes" answers below.
Circle questions you don't know the answers to.

	Yes	No
1. Have you had a medical illness or injury since your last check up or sports physical?	☐	☐
Do you have an ongoing or chronic illness?	☐	☐
2. Have you ever been hospitalized overnight?	☐	☐
Have you ever had surgery?	☐	☐
3. Are you currently taking any prescription or nonprescription (over-the-counter) medications or pills or using an inhaler?	☐	☐
Have you ever taken any supplements or vitamins to help you gain or lose weight or improve your performance?	☐	☐
4. Do you have any allergies (for example, to pollen, medicine, food, or stinging insects)?	☐	☐
Have you ever had a rash or hives develop during or after exercise?	☐	☐
5. Have you ever passed out during or after exercise?	☐	☐
Have you ever been dizzy during or after exercise?	☐	☐
Have you ever had chest pain during or after exercise?	☐	☐
Do you get tired more quickly than your friends do during exercise?	☐	☐
Have you ever had racing of your heart or skipped heartbeats?	☐	☐
Have you had high blood pressure or high cholesterol?	☐	☐
Have you ever been told you have a heart murmur?	☐	☐
Has any family member or relative died of heart problems or of sudden death before age 50?	☐	☐
Have you had a severe viral infection (for example, myocarditis or mononucleosis) within the last month?	☐	☐
Has a physician ever denied or restricted your participation in sports for any heart problems?	☐	☐
6. Do you have any current skin problems (for example, itching, rashes, acne, warts, fungus, or blisters)?	☐	☐
7. Have you ever had a head injury or concussion?	☐	☐
Have you ever been knocked out, become unconscious, or lost your memory?	☐	☐
Have you ever had a seizure?	☐	☐
Do you have frequent or severe headaches?	☐	☐
Have you ever had numbness or tingling in your arms, hands, legs, or feet?	☐	☐
Have you ever had a stinger, burner, or pinched nerve?	☐	☐
8. Have you ever become ill from exercising in the heat?	☐	☐
9. Do you cough, wheeze, or have trouble breathing during or after activity?	☐	☐
Do you have asthma?	☐	☐
Do you have seasonal allergies that require medical treatment?	☐	☐

	Yes	No
10. Do you use any special protective or corrective equipment or devices that aren't usually used for your sport or position (for example, knee brace, special neck roll, foot orthotics, retainer on your teeth, hearing aid)?	☐	☐
11. Have you had any problems with your eyes or vision?	☐	☐
Do you wear glasses, contacts, or protective eyewear?	☐	☐
12. Have you ever had a sprain, strain, or swelling after injury?	☐	☐
Have you broken or fractured any bones or dislocated any joints?	☐	☐
Have you had any other problems with pain or swelling in muscles, tendons, bones, or joints?	☐	☐

If yes, check appropriate box and explain below.

☐ Head	☐ Elbow	☐ Hip
☐ Neck	☐ Forearm	☐ Thigh
☐ Back	☐ Wrist	☐ Knee
☐ Chest	☐ Hand	☐ Shin/calf
☐ Shoulder	☐ Finger	☐ Ankle
☐ Upper arm		☐ Foot

13. Do you want to weigh more or less than you do now?	☐	☐
Do you lose weight regularly to meet weight requirements for your sport?	☐	☐
14. Do you feel stressed out?	☐	☐

15. Record the dates of your most recent immunizations (shots) for:

Tetanus _____ Measles _____

Hepatitis B _____ Chickenpox _____

FEMALES ONLY

16. When was your first menstrual period? _____

When was your most recent menstrual period? _____

How much time do you usually have from the start of one period to the start of another? _____

How many periods have you had in the last year? _____

What was the longest time between periods in the last year? _____

Explain "Yes" answers here: _____

I hereby state that, to the best of my knowledge, my answers to the above questions are complete and correct.

Signature of athlete _____ Signature of parent/guardian _____ Date _____

Figure 3.1 Preparticipation Physical Evaluation forms. (From Smith, D.M., Kovan, J.R., Rich, B.S., and Tanner, S.M., *Preparticipation Physical Examination*, 2nd ed., American Academy of Family Physicians, American Academy of Pediatrics, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and American Osteopathic Academy of Sports Medicine, Minneapolis, MN, 1997. With permission.)

Preparticipation Physical Evaluation

PHYSICAL EXAMINATION

Name _____ **Date of birth** _____

Height _____ **Weight** _____ **% Body fat (optional)** _____ **Pulse** _____ **BP** ___/___ (___/___ , ___/___)

Vision R 20/ ____ **L 20/** ____ **Corrected: Y N** **Pupils: Equal** ____ **Unequal** ____

	NORMAL	ABNORMAL FINDINGS	INITIALS*
MEDICAL			
Appearance			
Eyes/Ears/Nose/Throat			
Lymph Nodes			
Heart			
Pulses			
Lungs			
Abdomen			
Genitalia (males only)			
Skin			
MUSCULOSKELETAL			
Neck			
Back			
Shoulder/arm			
Elbow/forearm			
Wrist/hand			
Hip/thigh			
Knee			
Leg/ankle			
Foot			

*Station-based examination only

CLEARANCE

❑ **Cleared**

❑ **Cleared after completing evaluation/rehabilitation for:** _____

_____

_____

_____

❑ **Not cleared for:** _____ **Reason:** _____

Recommendations: _____

_____

_____

_____

Name of physician (print/type) _____ **Date** _____

Address _____ **Phone** _____

Signature of physician _____, **MD or DO**

Figure 3.1 (Continued)

tobacco use and may help tip off the examiner to delve into these topics. A high, arched palate may be a clue to a patient with Marfan's syndrome. The ear examination can reveal a ruptured tympanic membrane, which is a risk factor for participation in swimming and diving.[1–3,5,10,17]

CARDIAC

The cardiac history and physical exam are most critical and require extra attention because of the severity of the consequences if abnormalities are overlooked. Sudden cardiac death among high school athletes is estimated to occur at a frequency of 1/100,000 to 1/300,000 high school athletes per year.[1,5,18–23] These numbers are even higher among older athletes. The most common cause of sudden cardiac death among high school athletes is hypertrophic cardiomyopathy. In athletes over the age of 35, atherosclerotic disease accounts for the majority of deaths. The next most common cause of sudden cardiac death in young athletes is coronary anomalies. Other causes include ruptured aorta from Marfan's syndrome, aortic stenosis, myocarditis, dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia, mitral valve prolapse, long QT syndrome, and intramural coronary arteries.[18–23]

The appropriate screening methods for cardiac disease have been debated. Most agree that a cardiac history and physical exam is the most cost-effective approach for young athletes in detecting cardiac abnormalities that may predispose to sudden cardiac death. Electrocardiograms and echocardiograms have been used but are not generally considered to be cost effective and are not specific.[18,23] While the sensitivity of electrocardiograms in the setting of hypertrophic cardiomyopathy is high, many normal hearts will have abnormal electrocardiograms. In many structurally normal hearts of athletes, repolarization abnormalities, ST-T wave changes, findings consistent with left ventricular hypertrophy, and varying degrees of atrioventricular block will be observed.[22] As with electrocardiograms, echocardiograms can lead to many more false positives than true positives, as the incidence of cardiovascular abnormalities that lead to sudden cardiac death is so low. This could potentially lead to wrongful disqualification of athletes from participation. It has been estimated that the incidence of hypertrophic cardiomyopathy is approximately 1/500.[18,22] At an average cost of $600 per echocardiogram, it could cost up to $300,000 to detect one previously undiagnosed case.[22] If the incidence of sudden death is 1/200,000, and assuming the echocardiogram would detect all predisposing conditions (not true), the cost would skyrocket to $120,000,000 to prevent one death. Additionally, many cardiac abnormalities that predispose to sudden cardiac death, such as arrhythmogenic right ventricular dysplasia, cannot be diagnosed with an echocardiogram alone.[18] Therefore, at this time, routine screening of all young athletes with electrocardiography and echocardiography is not recommended; however, athletes with positive answers to the cardiac history questions and/or abnormalities on physical examination should be further evaluated.

Questions regarding the cardiac history are very important, as it carries the highest risk for sudden death on the athletic field. A number of questions need to be addressed, including a history of exercise-induced lightheadedness, dizziness, or syncope; family history of sudden cardiac death before the age of 50; chest pain; shortness of breath or excessive fatigue with exercise; and a history of murmur, irregular heart beats, or palpitations. Many cardiac abnormalities can be familial and include Marfan's syndrome, hypertrophic cardiomyopathy, and atherosclerosis. Arrhythmias, conduction abnormalities, anomalous coronary arteries, and valvular disease may result in symptoms of fatigue, lightheadedness, dizziness, or syncope.[1–3,5,6,10,14,18–23] Syncope during exercise is a much more worrisome finding than syncope after exercise. If it occurs during exercise, it is much more likely to be related to an underlying medical condition that could be life threatening. Syncope after exercise is more likely associated with exercise-associated collapse or exercise-related syncope. If exercise and muscle contraction cease abruptly, the pumping action of the skeletal muscle is lost. This, in combination with a reflex vasodilation, results in syncope.[21,24] A history of illegal drug use becomes important, as cocaine use can cause sudden cardiac death.[23]

Older individuals require a more in-depth cardiac examination if they plan on engaging in regular strenuous exercise or competitive sports competition. Maron et al.[25] make the following recommendations for older athletes. Men over the age of 40 to 45 and women over the age of 50 to 55 with one or more risk factors for coronary artery disease should undergo exercise stress testing. These risk factors include hypercholesterolemia or dyslipidemia, systemic hypertension, current or recent cigarette smoking, diabetes mellitus, or history of myocardial infarction or sudden cardiac death in a first-degree relative less than 60 years old. All athletes 65 years of age and older and all athletes with symptoms of coronary disease should undergo an exercise test. Athletes younger than 65 who are asymptomatic and without cardiac risk factors do not need to undergo exercise stress testing. Echocardiography should be performed on all patients if the history or

TABLE 3.1
Coronary Artery Disease Risk Factor Thresholds for Use with ACSM Risk Stratification

Risk Factors	Defining Criteria
Positive	
Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or other male first-degree relative (i.e., brother or son) or before 65 years of age in mother or other female first-degree relative (i.e., sister or daughter)
Cigarette smoking	Current cigarette smoker or those who quit within the previous 6 months
Hypertension	Systolic blood pressure of >140 mmHg or diastolic of >90mm Hg, confirmed by measurements on at least two separate occasions, or on antihypertensive medication
Hypercholesterolemia	Total serum of cholesterol of >200 mg/dL (5.2 mmol/L) or high-density lipoprotein cholesterol of <35 mg/dl (0.9 mmol/L), or on lipid-lowering medication; if low-density lipoprotein cholesterol is available, use >130 mg/dL (3.4 mmol/L) rather than total cholesterol of >200 mg/dL
Impaired fasting glucose	Fasting blood glucose of >110 mg/dL (6.1 mmol/L) confirmed by measurements on at least two separate occasions[7]
Obesity[a]	Body mass index (BMI) of >30 kg/m^2 [8] or waist girth of >100 cm[9]
Sedentary lifestyle	Persons not participating in a regular exercise program or meeting the minimal physical activity recommendations[b] from the U.S. Surgeon General's report[10]
Negative	
High serum HDL cholesterol[c]	>60 mg/dL (1.6 mmol/L)

[a] Professional opinions vary regarding the most appropriate markers and thresholds for obesity; therefore, exercise professionals should use clinical judgment when evaluating this risk factor.

[b] Accumulating 30 minutes or more of moderate physical activity on most days of the week.

[c] It is common to sum risk factors in making clinical judgments. If high-density lipoprotein (HDL) cholesterol is high, subtract one risk factor from the sum of positive risk factors because high HDL decreases risk of coronary artery disease.

Source: Adapted from Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, *JAMA*, 269, 3015–3023, 1993.

physical is suggestive of valvular heart disease, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, or prior myocardial infarction.[25] The American College of Sports Medicine (ACSM) has different guidelines for older athletes that are more stringent and rely on risk stratification and moderate vs. strenuous exercise (Tables 3.1 to 3.4).[26] If cardiac abnormalities are identified in both older and younger athletes, the guidelines from the 26th Bethesda Conference[27] are an excellent reference regarding recommendations for participation for various cardiac problems. All recommendations are level C (consensus/expert opinion).

The heart exam is the single most important aspect of the physical exam because undetected cardiac abnormalities can lead to sudden death. The examiner should listen in a quiet area with the patient in the sitting, squatting, and supine positions. A murmur that is louder with a Valsava maneuver or with a position that decreases venous return should be further investigated, as such a finding is associated with hypertrophic cardiomyopathy. Any diastolic murmur and any murmur that is higher than a grade 3/6 should also be further evaluated. Femoral and brachial arteries should be palpated to rule out coarctation of the aorta. The patient should also be evaluated for signs of Marfan's syndrome, as this syndrome is associated with aortic rupture and sudden cardiac death. The physical exam findings include an unusually tall person with a wide arm span, pectus excavatum, long fingers, myopia, and displaced lenses.[1,3,5,10,18,19,21–23]

Blood pressure readings should be taken preferably in a quiet, relaxed setting. If it is initially elevated, the reading should be taken again after a few minutes. If it is elevated a second time, the athlete should lie down and rest for 10 to 15 minutes. Unfortunately, this is sometimes difficult to do, especially in a mass screening type of examination format. Hypertension itself does not disqualify an athlete from participation unless it is severe and untreated. Published guidelines can help to determine the severity of the hypertension

TABLE 3.2
Major Signs or Symptoms Suggestive of Cardiovascular and Pulmonary Disease[a]

Pain, discomfort (or other angina equivalent) in the chest, neck, jaw arms, or other areas that may be due to ischemia
Shortness of breath at rest or with mild exertion
Dizziness or syncope
Orthopnea or paroxysmal nocturnal dyspnea
Unusual fatigue or shortness of breath with usual activities
Ankle edema
Palpitations or tachycardia
Intermittent claudication
Known murmur

[a] These symptoms must be interpreted in the clinical context in which they appear because they are not all specific for cardiovascular, pulmonary, or metabolic disease. For clarification and discussion of the clinical significance of the signs or symptoms, see Glover and Maron.[11]

Source: Adapted from Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, *JAMA*, 269, 3015–3023, 1993.

TABLE 3.3
Initial ACSM Risk Stratification

Low risk	Younger individuals[a] who are asymptomatic and meet no more than one risk factor from Table 3.1
Moderate risk	Older individuals (men ≥45 years of age; women ≥55 years of age) or those who meet the threshold for two or more risk factors from Table 3.1
High risk	Individuals with one or more signs/symptoms listed in Table 3.2 or known cardiovascular,[b] pulmonary,[c] or metabolic[d] disease

[a] Men <45 years of age; women <55 years of age.

[b] Cardiac, peripheral vascular, cerebrovascular disease.

[c] Chronic obstructive pulmonary disease, asthma, interstitial lung disease, or cystic fibrosis (see American Heart Association[12]).

[d] Diabetes mellitus (types 1 and 2), thyroid disorders, renal or liver disease.

Source: Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, *JAMA*, 269, 3015–3023, 1993.

TABLE 3.4
ACSM Recommendations for Current Medical Examination and Exercise Testing Prior to Participation and Physician Supervision of Exercise Tests

	Low Risk	Moderate Risk	High Risk
Current Medical Examination[a] and Exercise Testing Prior to Participation			
Moderate exercise[b]	Not necessary[c]	Not necessary	Recommended
Vigorous exercise[d]	Not necessary	Not necessary	Recommended
Physician Supervision of Exercise Tests			
Submaximal test	Not necessary	Not necessary	Recommended
Maximal test	Not necessary	Recommended[e]	Recommended

[a] Within the past year (see Metzl[2]).

[b] Absolute moderate exercise is defined as activities that are approximately 3 to 6 METs or the equivalent of brisk walking at 3 to 4 mph for most healthy adults.[13] Nevertheless, a pace of 3 to 4 mph may be considered to be "hard" to "very hard" by some sedentary, older persons. Moderate exercise may alternatively be defined as an intensity well within the individual's capacity, one that can be comfortably sustained for a prolonged period of time (about 45 minutes), has a gradual initiation and progression, and is generally noncompetitive. If an individual's exercise capacity is known, relative moderate exercise may be defined by the range 40 to 60% maximal oxygen uptake.

[c] The designation of "Not necessary" reflects the notion that a medical examination, exercise test, and physician supervision of exercise testing would not be essential in the preparticipation screening: however, they should not be viewed as inappropriate.

[d] Vigorous exercise is defined as activities of >6 METs. Vigorous exercise may alternatively be defined as exercise intense enough to represent a substantial cardiorespiratory challenge. If an individual's exercise capacity is known, vigorous exercise may be defined as an intensity of >60% maximal oxygen uptake.

[e] When physician supervision of exercise testing is recommended, the physician should be in close proximity and readily available should there be an urgent need.

Source: Adapted from Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, *JAMA*, 269, 3015–3023, 1993.

TABLE 3.5
Classification of Hypertension by Age-Group

Age Group (years)	Significant Hypertension (mmHg)	Severe Hypertension (mmHg)
Children (6–9)	Systolic, ≥122	Systolic, ≥130
	Diastolic, ≥78	Diastolic, ≥86
Children (10–12)	Systolic, ≥126	Systolic, ≥134
	Diastolic, ≥82	Diastolic, ≥90
Adolescents (13–15)	Systolic, ≥136	Systolic, ≥144
	Diastolic, ≥86	Diastolic, ≥92
Adolescents (16–18)	Systolic, ≥142	Systolic, ≥150
	Diastolic, ≥92	Diastolic, ≥98

Source: Task Force on Blood Pressure Control in Children, National Heart, Lung, and Blood Institute, Bethesda, MD, Report of the Second Task Force on Blood Pressure Control in Children — 1987, *Pediatrics*, 79(1), 1–15, 1987.

and can be used in making clearance decisions (Table 3.5).[28]

PULMONARY

Exercise-induced asthma is the most common medical consideration in young athletes and can be discovered by asking questions pertaining to wheezing and/or coughing during exercise. Positive answers to these questions should lead to further peak flow testing. Baseline readings can be obtained and compared to readings taken after exercise. A decrease of 10 to 15% is suggestive of exercise-induced asthma. This can prove to be an invaluable diagnosis because appropriately diagnosed athletes can be optimally treated to perform at their best. The lung exam is normal in most cases. Even those with exercise-induced asthma will usually have a normal exam at rest. Participation is allowed for all sports if the asthma is under control. Only athletes with severe asthma will need restrictions on activity. Classic findings are increased shortness of breath relative to others, bronchospasm, and cough 10 to 15 minutes into exertion or occasionally shortly after cessation of exercise.

A less common abnormality is primary spontaneous pneumothorax. Those who have undergone only conservative management should be counseled on the risks of recurrence if they are involved in strenuous or contact sports. Athletes who have undergone thoracotomy or other invasive procedures should be allowed to return to all sports in 2 to 4 weeks. In either case, if a pneumothorax does recur, the athlete should be advised not to participate in contact sports.[29]

ABDOMEN

The abdominal exam should be done with the athlete in a supine position, and all four quadrants should be palpated. This allows the examiner to evaluate the athlete for organomegaly. An acutely enlarged liver or spleen is a contraindication to collision/contact or limited-contact sports. Infectious mononucleosis can cause acute splenomegaly and puts the athlete at risk for splenic rupture. An athlete should be kept out of competition in strenuous noncontact sports for 3 weeks and out of contact/collision sports for a full month after infectious mononucleosis.[1–3,5,10] Another important finding that should not be missed in young female athletes is the presence of a gravid uterus.

NEUROLOGIC

It is important to ascertain a past history of concussions, including the severity and frequency of occurrence, as well as loss of consciousness. This may help to identify those individuals at greater risk for recurrent injury. The many guidelines regarding return-to-play criteria following concussions all agree that an athlete should not be allowed to return to play if still symptomatic from a concussion. This can put an athlete at risk for second impact syndrome, which results from a second concussion that occurs before the athlete is asymptomatic from the first. This can lead to vascular engorgement and death.[29] All primary care physicians should be familiar with guidelines for continued participation in contact/collision sports after repeated concussions. Guidelines proposed by Cantu,[30] the Colorado Medical Society,[31] and the American Academy of Neurology[32] (Level 5 evidence, expert opinion) are most commonly used. History of a seizure disorder is also important as it can affect the athlete's eligibility for certain sports such as archery, swimming, and riflery. Burners/stingers or pinched nerves are other neurologic complications that should be addressed and recorded. They usually are the result of stretching or compression of the cervical nerve roots or brachial plexus. The physician should inquire about transient quadriplegia, which is a rare problem that presents as burning pain, numbness or tingling, and weakness or paralysis of all four extremities. If an athlete has recurrent burner/stingers or transient quadriplegia, further workup should be performed to rule out a structural abnormality. If such an abnormality exists, competition in contact/collision sports should be restricted.[1,2,5,6,29]

MUSCULOSKELETAL

The examiner should record a history of previous orthopedic injuries. The purpose of this portion of the history is to identify musculoskeletal abnormalities that may predispose to future injury. This information can also be used to help individualize rehabilitation programs. A recent study showed that, of the athletes who were disqualified from participation, the majority were not cleared secondary to musculoskeletal problems.[8]

Most musculoskeletal injuries are detected by the history, and it is rare to find an abnormality in an asymptomatic athlete without a history of a previous injury. Thus, a general screening approach (commonly termed the "2-minute orthopedic exam") is recommended, with a more detailed exam of areas of prior injury. A general screening examination has been developed and has been accepted by many major medical associations.[1] This screening examination in combination with an orthopedic history should be used for asymptomatic athletes with no previous injuries (Level of Evidence 1, randomized controlled trial/meta-analysis).[33] Joint-specific examinations become important and should be performed if a joint has been previously injured. Some physicians may prefer to do the entire joint-specific examination on all athletes; however, it is usually unnecessary because it is low yield in asymptomatic individuals. Another variation of the musculoskeletal exam is to do sport-specific examinations. For example, an examiner may spend more time on the shoulder exam of baseball players and swimmers in order to maximize strength and conditioning programs. Joint-specific and sport-specific exams are more time consuming than the general screening exam. The sport-specific examination is usually done by sports medicine physicians, as it requires more in-depth knowledge of individual sports.[1–3,5,7,10,14,17]

In general, clearance is denied to an athlete with a musculoskeletal injury who has persistent effusion or edema, loss of functional ability, strength that is less than 85 to 90% of the unaffected side, and significantly decreased range of motion.[1] Spinal conditions that are cause for disqualification include symptomatic spondylolysis, spondylolisthesis, functional cervical spinal stenosis, spear tackler's spine, and herniated discs with cord compression.[1,17] Referral to a specialist is warranted when the examining physician is unsure of the clearance guidelines for particular musculoskeletal disorders.

DERMATOLOGIC

The physician should inquire about certain skin infections, such as herpes simplex, impetigo, boils, scabies, and molluscum contagiosum. When an athlete is contagious, participation in sports that involve mats (such as wrestling, gymnastics, and martial arts) as well as contact/collision sports or limited-contact sports should not be allowed.[1,2,5,10,17,34]

GENITOURINARY

Athletes with a single kidney or testicle require special consideration. The American Academy of Pediatrics formerly recommended that a single kidney was a contraindication to participation in high-contact sports.[6] This is no longer true. Now the recommendation is for athletes with a single kidney to be assessed on an individual basis to determine eligibility for high-contact sports.[35] The use of a "flak" jacket is recommended for moderate-contact sports.[1,6] A single testicle is not a contraindication to athletic participation, but a protective cup should be worn. The athlete and parents must be informed of the risks of injury or loss to the remaining testicle.[1,2,5,10,17]

HEAT ILLNESS

A prior episode of heat-related illness may predispose to future problems. It becomes more apparent in hot, humid environments. Coaches and trainers need to be aware of the risks of heat stroke and allow adequate time for rehydration. Athletes at risk need to be especially cautious so as not to become overheated. The physician should also inquire about medications such as diuretics, antihistamines, amphetamines, and ergogenic aids such as ephedra and creatine that may predispose to dehydration. Assessments for clearance need to be done on an individual basis if there is a history of heat illness. A restriction to cooler environments might be warranted in certain cases.[1,5,7]

SPECIAL CONSIDERATIONS FOR THE FEMALE ATHLETE

Certain questions should be addressed during the preparticipation physical examination of women. The female athlete triad refers to a pattern of disordered eating, amenorrhea, and abnormal bone mineral density and is a common problem encountered among women athletes. It is seen in higher frequency in sports where appearance is important or thinness is an advantage such as gymnastics and distance running. The examiner should inquire about age at menarche, frequency

TABLE 3.6
Classification of Sports by Contact

Contact/Collision	Limited Contact	Noncontact
Basketball	Baseball	Archery
Boxing[a]	Bicycling	Badminton
Diving	Cheerleading	Body building
Field hockey	Canoeing/kayaking (white water)	Canoeing/kayaking (flat water)
Football	Fencing	Crew/rowing
Flag	Field (high jump, pole vault)	Curling
Tackle	Floor hockey	Dancing
Ice hockey	Gymnastics	Field (discus, javelin, shotput)
Lacrosse	Handball	Golf
Martial arts	Horseback riding	Orienteering
Rodeo	Racquetball	Power lifting
Rugby	Skating (ice, inline, roller)	Race walking
Ski jumping	Skiing (cross-country, downhill, water)	Riflery
Soccer	Softball	Rope jumping
Team handball	Squash	Running
Water polo	Ultimate frisbee	Sailing
Wrestling	Volleyball	Scuba diving
	Windsurfing/surfing	Strength training
		Swimming
		Table tennis
		Tennis
		Track
		Weight lifting

[a] Participation not recommended by the American Academy of Pediatrics; the American Academy of Family Physicians, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and American Osteopathic Academy of Sports Medicine have no stand against boxing.

Source: From American Academy of Pediatrics Committee on Sports Medicine and Fitness, *Pediatrics*, 94(5), 757–776, 1994. With permission.

of menstrual periods, history of stress fractures, and eating patterns. The benefit of hormonal therapy for amenorrheic athletes remains in question, but most researchers and the authors agree that oral contraceptive pills should be given in order to try to increase bone mineral density and therefore prevent stress fractures. While the presence of eating disorders is rarely cause for disqualification, recognizing them can aid in getting the athlete the appropriate counseling. Left untreated, eating disorders can lead to menstrual irregularities, osteoporosis, poor cold tolerance, bradycardia, electrolyte abnormalities, gastrointestinal problems, acid–base disorders, cardiac abnormalities, infertility, organ failure, and even death.[1,5,6,36,37]

ROUTINE SCREENING TESTS

Routine laboratory tests such as a urinalysis or complete blood count, are not recommended (Level of Evidence B, other evidence).[3,4,14,33] If, however, the history or physical examination raises concerns, then further tests should be ordered. For example, a female with a history of heavy menstrual flow, poor eating habits, and fatigue should have her hematocrit checked and perhaps iron studies evaluated. A person with a family history of sickle cell disease should also have a complete blood count with evaluation of the peripheral smear. Urinalysis should be performed if the athlete provides a history of hematuria, dysuria, or polycystic kidney disease. A fasting lipid profile should be performed on those with a family history of premature coronary artery

TABLE 3.7
Classification of Sports by Strenuousness

High-to-Moderate Intensity			Low Intensity
High-to-Moderate Dynamic and Static Demands	High-to-Moderate Dynamic and Low Static Demands	High-to-Moderate Static and Low Dynamic Demands	Low Dynamic and Low Static Demands
Boxing[a]	Badminton	Archery	Bowling
Crew rowing	Baseball	Auto racing	Cricket
Cross-country skiing	Basketball	Diving	Curling
Cycling	Field hockey	Equestrian	Golf
Downhill skiing	Lacrosse	Field events (jumping)	Riflery
Fencing	Orienteering	Field events (throwing)	
Football	Ping pong	Gymnastics	
Ice hockey	Race walking	Karate or judo	
Rugby	Racquetball	Motorcycling	
Running (sprint)	Soccer	Rodeoing	
Speed skating	Squash	Sailing	
Water polo	Swimming	Ski jumping	
Wrestling	Tennis	Water skiing	
	Volleyball	Weight lifting	

[a] Participation not recommended by the American Academy of Pediatrics; the American Academy of Family Physicians, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and American Osteopathic Academy of Sports Medicine have no stand against boxing.

Source: From American Academy of Pediatrics Committee on Sports Medicine and Fitness, *Pediatrics*, 94(5), 757–776, 1994. With permission.

disease to evaluate for familial types of hyperlipidemia.[3] Controversy exists regarding neck imaging in athletes with Down's syndrome. The Special Olympics supports a policy that all athletes with Down's syndrome undergo cervical radiographs with extension and flexion views to rule out atlantoaxial instability; however, the American Academy of Pediatrics no longer supports this policy. In conclusion, routine screening tests are not recommended, but further tests should be done if warranted by the history and physical.

CLEARANCE

Determining clearance for an athlete is a difficult task for physicians. The preparticipation examination form should include a section that indicates whether or not the athlete has been cleared for sports participation. An athlete should not be cleared for participation if the sport constitutes a health risk for the individual or puts other athletes at risk for injury. Sometimes limited competition may be allowed during treatment or if protective equipment is used. The physician may choose to allow clearance pending further investigation of specific problems. Sports are generally classified by the amount of contact and how strenuous they are (Tables 3.6 and 3.7). Determination of clearance should take into account the type of sport in which the individual will be competing. The 26th Bethesda Conference guidelines are an excellent resource when deciding on clearance for cardiac abnormalities, and general clearance guidelines can be found in Figure 3.2.[1] Most of the time, clearance must be determined on an individual basis. Occasionally, an athlete or an athlete's parents will disagree with the doctor's recommendation to deny clearance. Under the Rehabilitation Act of 1973 and the Americans with Disabilities Act of 1990, the athlete may actually have the right to participate against medical advice. If the athlete does decide to participate, an exculpatory waiver should be signed by the athlete and/or parent(s)/guardian(s) that indicates awareness of the risks of participation and prohibits bringing suit against the physician in the event of injury. Unfortunately, the validity of this type of waiver varies from state to state, so it may not completely protect the physician from legal action.

CONCLUSION

The preparticipation physical examination is important to help ensure safety in athletic participation. It is not intended to and rarely does it result in disqualification of athletes from competition. The

Medical Conditions and Sports Participation*

Condition	May Participate
Atlantoaxial instability (instability of the joint between cervical vertebrae 1 and 2)	Qualified yes
Explanation: Athlete needs evaluation to assess risk of spinal cord injury during sports participation.	
Bleeding disorder	Qualified yes
Explanation: Athlete needs evaluation.	
Cardiovascular disease	
Carditis (inflammation of the heart)	No
Explanation: Carditis may result in sudden death with exertion.	
Hypertension (high blood pressure)	Qualified yes
Explanation: Those with significant essential (unexplained) hypertension should avoid weight and power lifting, body building, and strength training. Those with secondary hypertension (hypertension caused by a previously identified disease) or severe essential hypertension need evaluation. The National High Blood Pressure Education Working group[3] defined significant and severe hypertension.	
Congenital heart disease (structural heart defects present at birth)	Qualified yes
Explanation: Those with mild forms may participate fully; those with moderate or severe forms or who have undergone surgery need evaluation. The 26th Bethesda Conference[4] defined mild, moderate, and severe disease for common cardiac lesions.	
Dysrhythmia (irregular heart rhythm)	Qualified yes
Explanation: Those with symptoms (chest pain, syncope, dizziness, shortness of breath, or other symptoms of possible dysrhythmia) or evidence of mitral regurgitation (leaking) on physical examination need evaluation. All others may participate fully.[5]	
Heart murmur	Qualified yes
Explanation: If the murmur is innocent (does not indicate heart disease), full participation is permitted. Otherwise, the athlete needs evaluation (see congenital heart disease and mitral valve prolapse[5]).	
Cerebral palsy	Qualified yes
Explanation: Athlete needs evaluation.	
Diabetes mellitus	Yes
Explanation: All sports can be played with proper attention to diet, blood glucose concentration, hydration, and insulin therapy. Blood glucose concentration should be monitored every 30 minutes during continuous exercise and 15 minutes after completion of exercise.	
Diarrhea	Qualified no
Explanation: Unless disease is mild, no participation is permitted, because diarrhea may increase the risk of dehydration and heat illness. See fever.	

Figure 3.2 Medical Conditions and Sports Participation form. (From American Academy of Pediatrics Committee on Sports Medicine and Fitness, *Pediatrics*, 107(5), 1205–1209, 2001. With permission.)

Eating disorders	Qualified yes
Anorexia nervosa	
Bulimia nervosa	
Explanation: Patients with these disorders need medical and psychiatric assessment before participation.	
Eyes	Qualified yes
Functionally one-eyed athlete	
Loss of an eye	
Detached retina	
Previous eye surgery or serious eye injury	
Explanation: A functionally one-eyed athlete has a best-corrected visual acuity of less than 20/40 in the eye with worse acuity. These athletes would suffer significant disability if the better eye were seriously injured, as would those with loss of an eye. Some athletes who previously have undergone eye surgery or had a serious eye injury may have 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.[6,7]	
Fever	No
Explanation: Fever can increase cardiopulmonary effort, reduce maximum exercise capacity, make heat illness more likely, and increase orthostatic hypertension during exercise. Fever may rarely accompany myocarditis or other infections that may make exercise dangerous.	
Heat illness, history of	Qualified yes
Explanation: Because of the increased likelihood of recurrence, the athlete needs individual assessment to determine the presence of predisposing conditions and to arrange a prevention strategy.	
Hepatitis	Yes
Explanation: Because of the apparent minimal risk to others, all sports may be played that the athlete's state of health allows. In all athletes, skin lesions should be covered properly, and athletic personnel should use universal precautions when handling blood or body fluids with visible blood.[8]	
Human immunodeficiency virus infection	Yes
Explanation: Because of the apparent minimal risk to others, all sports may be played that the athlete's state of health allows. In all athletes, skin lesions should be covered properly, and athletic personnel should use universal precautions when handling blood or body fluids with visible blood.[8]	
Kidney, absence of one	Qualified yes
Explanation: Athlete needs individual assessment for contact, collision, and limited-contact sports.	
Liver, enlarged	Qualified yes
Explanation: If the liver is acutely enlarged, participation should be avoided because of risk of rupture. If the liver is chronically enlarged, individual assessment is needed before collision, contact, or limited-contact sports are played.	

Figure 3.2 (Continued)

Malignant neoplasm	Qualified yes
Explanation: Athlete needs individual assessment.	
Musculoskeletal disorders	Qualified yes
Explanation: Athlete needs individual assessment.	
Neurologic disorders	
History of serious head or spine trauma, severe or repeated concussions, or crainotomy.[9,10]	Qualified yes
Explanation: Athlete needs individual assessment for collision, contact, or limited-contact sports and also for noncontact sports if deficits in judgment or cognition are present. Research supports a conservative approach to management of concussion.[9,10]	
Seizure disorder, well-controlled	Yes
Explanation: Risk of seizure during participation is minimal	
Seizure disorder, poorly controlled	Qualified yes
Explanation: Athlete needs individual assessment for collision, contact, or limited-contact sports. The following noncontact sports should be avoided: archery, riflery, swimming, weight or power lifting, strength training, or sports involving heights. In these sports, occurrence of a seizure may pose a risk to self or others.	
Obesity	Yes
Explanation: Because of the risk of heat illness, obese persons need careful acclimatization and hydration.	
Organ transplant recipient	
Explanation: Athlete needs individual assessment.	
Ovary, absence of one	Yes
Explanation: Risk of severe injury to the remaining ovary is minimal.	
Respiratory conditions	
Pulmonary compromise, including cystic fibrosis	Qualified yes
Explanation: Athlete needs individual assessment, but generally, all sports may be played if oxygenation remains satisfactory during a graded exercise test. Patients with cystic fibrosis need acclimatization and good hydration to reduce the risk of heat illness.	
Asthma	Yes
Explanation: With proper medication and education, only athletes with the most severe asthma will need to modify their participation.	
Acute upper respiratory infection	Qualified yes
Explanation: Upper respiratory obstruction may affect pulmonary function. Athlete needs individual assessment for all but mild disease. See fever.	
Sickle cell disease	Qualified yes

Figure 3.2 (Continued)

Explanation: Athlete needs individual assessment. In general, if status of the illness permits, all but high exertion, collision, and contact sports may be played. Overheating, dehydration, and chilling must be avoided.	
Sickle cell trait	Yes
Explanation: It is unlikely that persons with sickle cell trait have an increased risk of sudden death or other medical problems during athletic participation, except under the most extreme conditions of heat, humidity, and possibly increased altitude.[11] These persons, like all athletes, should be carefully conditioned, acclimatized, and hydrated to reduce any possible risk.	
Skin disorders (boils, herpes simplex, impetigo, scabies, molluscum contagiosum)	Qualified yes
Explanation: While the patient is contagious, participation in gymnastics with mats; martial arts; wrestling; or other collision, contact, or limited-contact sports is not allowed.	
Spleen, enlarged	Qualified yes
Explanation: A patient with an acutely enlarged spleen should avoid all sports because of risk of rupture A patient with a chronically enlarged spleen needs individual assessment before playing collision, contact, or limited-contact sports.	
Testicle, undescended or absence of one	Yes
Explanation: Certain sports may require a protective cup.	

* This table is designed for use by medical and nonmedical personnel. "Needs evaluation" means that a physician with appropriate knowledge and experience should assess the safety of a given sport for an athlete with the listed medical condition. Unless otherwise noted, this is because of variability of the severity of the disease, the risk of injury for the specific sports listed in Table 1, or both.

Figure 3.2 (Continued)

exam should be scheduled prior to the beginning of the sport season, allowing adequate time for further evaluation of identified abnormalities. The authors favor individual exams by qualified primary care or sports medicine physicians. Whenever doubt arises as to whether an athlete should be allowed to compete in a particular sport, it is recommended to refer to appropriate resources/specialists to aid in the decision process. Because many athletes use this exam as their only interaction with healthcare professionals, it should be used as an opportunity to establish trust and foster a physician–patient relationship.

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4 LEGAL ASPECTS OF SPORTS MEDICINE*

Lauren M. Simon

INTRODUCTION

With the growth of participation in sports, the sports medicine team and the medicolegal aspects of sports medicine have also grown in importance. Although the legal aspects of sports medicine are constantly evolving, having an understanding of the basic legal principles involved in providing medical care to athletes of all ages is vital to the successful practice of sports medicine. That knowledge will also help to dispel fears about liability that otherwise may prevent some physicians from engaging in the practice of sports medicine. This chapter is designed to familiarize physicians with the legal regulations affecting sports medicine.

ROLE OF THE TEAM PHYSICIAN

Although they come from a variety of backgrounds, team physicians should practice sports medicine according to relevant standards of care for both emergency and non-emergency situations. They should keep updated on the treatment of individual athletes and teams.[1] Some physicians are hired by a school or team to provide medical services for a specific athletic contest or series of events, whereas others volunteer to provide those services *gratis* (without charge). A spectator at an

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athletic event who happens to be a physician may also aid an athlete in case of an emergency (see The Good Samaritan section).

A team physician consensus statement, which outlines the duties of a team physician, has been developed by a sports medicine project-based alliance of six major professional sports medicine organizations.[2] The major responsibilities of the team physician include preparation of preparticipation evaluations of athletes, treatment and management of injuries, supervision of rehabilitation, and education of athletes, teams, athletic staff, and schools on multiple topics such as protective equipment, injury prevention, minimizing injuries, conditioning guidelines, infection precautions, and nutrition. The team physician should be familiar with the injury patterns commonly seen in specific sports. It is also important for the team physician to be cognizant of the rules regarding the athletes' medical eligibility and to be vigilant when prescribing any medication for any potential impact on an athlete's ability and eligibility to play. The team physician may also be involved in decisions about athletes or teams playing in various environmental conditions. An extremely important function of the team physician is to ensure that a well-conceived plan is in place to handle emergencies. Although the details of actual emergency situations vary, ideally a trial run should be performed to smooth out any apparent wrinkles in the emergency plan before an actual emergency arises.

TEAM PHYSICIAN AGREEMENT

Team physician responsibilities can vary depending on the sport, level of competition, sports governing body, school league, university, age of athlete, etc. In addition, the team physician may be treating the athlete in a variety of situations from the relative calm of the office to the intense pressure of an important game. In order for the team physician to be able to treat the athletes according to the standard of care, no matter what the situation, it is strongly recommended that prior to beginning to serve as a team physician the physician should have a written agreement or contract that delineates the team physician's duties and role particularly regarding return-to-play decisions. The agreement should be between the physician and the team or governance organization for the team such as the school district, university, or sports league.[1]

SPORTS MEDICINE LIABILITY

Some physicians are hesitant to practice sports medicine because of fear of being sued. If a sports medicine physician is sued for medical treatment given or failure to treat an athlete and if the case results in a trial, the key question will be, "Did the physician abide by the standard of care?" The standard of care in sports medicine is constantly evolving, and the plaintiff and the defense will bring in experts to testify on the issue. To be found liable for medical malpractice, a sports medicine physician must be found to have been negligent. For a litigant to prevail against the physician on a claim of medical malpractice, four elements of negligence must be considered: duty, breach, causation, and damages.

The first element, *duty*, refers to whether the team physician has a physician–patient relationship, an obligation recognized by law that results in a duty to care for the athlete in question. Where such a relationship or duty exists, the physician must competently care for the athlete at the minimum skill level of other physicians who provide medical treatment for such an injury.

The second element, *breach*, reflects a failure to conform to the requisite standard of care (expected of a reasonably competent sports physician in similar circumstances) for the athlete. Much of the testimony in medical malpractice cases consists of expert witnesses for the patient/plaintiff and the physician/defendant who attempt to demonstrate that the physician's diagnosis/treatment did not or did conform to the standard of care owed to the patient.

The third element, *causation*, is proven if the plaintiff demonstrates that the physician's breach of the duty of care (that is, the failure to provide the minimum skill level to the patient) resulted in (that is, had a sufficient causal connection to) the specific harm befalling the patient/athlete. A physician cannot be found negligent by causation unless the medical treatment (or failure to treat) fell below the standard of care and resulted in harm or injury to the patient. For example, if a team physician fails to diagnose a fractured fibula in a football player who returns to the game and subsequently dislocates his shoulder, causation has not been proven. This is because the treatment (or lack thereof) involving the fibula did not cause the injury to the shoulder.

The fourth element of negligence is *damages*. To have a viable case against a physician, the patient/athlete must have sustained some quantifiable damages, whether they be physical, financial, or emotional costs.[1] This means that if the

team physician renders medical treatment below the standard of care that proximately causes an injury but no damages result, a negligence claim should not hold up in court. A sports physician who engages in care of athletes must possess a similar degree of skill, learning, and care compared to reasonably competent sports physicians acting under similar circumstances. For example, a reasonably competent sports physician should be aware that an athlete who sustains a head injury and is knocked unconscious should be assumed to have an associated cervical spine injury and an immediate airway, breathing, circulation, and appropriate neurological evaluation plus immobilization should be performed.[3] If a sports medicine physician does what a reasonably competent sports medicine physician would do under the same or similar circumstances, an athlete/plaintiff will be unlikely to prevail on a claim of negligence against the physician.

Medical Malpractice Insurance

It is essential that a team physician's malpractice insurance cover the scope of the physician's practice of sports medicine. Because many of the patients treated by team physicians (particularly in a school setting) are minors (who in certain circumstances can file lawsuits upon reaching the age of majority), careful consideration must be given to the type of insurance coverage selected by the team physician.

Malpractice insurance can be divided into two main types: *occurrence* and *claims-made*. Occurrence insurance provides coverage for alleged malpractice events that occur during the policy period but which can be brought to court during or after the period of the policy. Because occurrence insurance covers the physician for the alleged events, even after the time period of the policy may have ended, it does not require tail coverage. In contrast, claims-made insurance provides coverage for alleged malpractice events that occur *and* were brought to court during the time that insurance policy was in effect. When a physician has claims-made insurance, it is recommended that the physician acquire tail coverage to ensure malpractice coverage in case a claim is filed at a later date.

Some malpractice carriers place restrictions that deny coverage for certain activities. If a physician's malpractice insurance does not cover sports medicine activities, he should obtain additional insurance coverage before participating in uncovered medical practice. It is important for the physician to be knowledgeable about potential exclusions on the malpractice policy, such as the practice of medicine outside the physical confines of an office or hospital, that may preclude the physician from conducting examinations at a gymnasium or covering athletic events. Additionally, if a physician travels with a team, it is important to check the "territory" section of a malpractice policy to see if the locale to which the physician is traveling is within the scope of coverage of the insurance policy.

THE TRAVELING TEAM PHYSICIAN

It is possible that when a team physician accompanies a team out of state (where the team physician is not licensed to practice medicine) that the physician might be perceived as practicing medicine without a license. In order to avoid this problem, team physicians must become familiar with the laws governing the practice of medicine in other states. A good place to gather information or resources concerning another state's medicolegal policies is through that state's medical board. Some states allow physicians who are licensed in another state to practice medicine occasionally in their state, as long as the physician does not have an office in that state or purport to be licensed in the visiting state; others do not.[1] It is the responsibility of team physicians to know the laws of the state that they are visiting.

When traveling to other states, physicians should familiarize themselves with the protocols and standard of care for sports medicine providers in that domain. It is important to learn the emergency policies and procedures before the visiting athletes engage in sports activities to provide for their safety. It is also recommended that the local sports medicine providers be apprised of any special medical needs of traveling team members. Physicians repeatedly traveling with a university or professional teams often form informal relationships with physicians in the same sports leagues who can help inform them of local policies and provide for any special medical needs of their athletes. To be safe, physicians should refer to the local medical board or legal counsel when in doubt.

Physicians traveling domestically must also be aware of travel restrictions on the contents of the sports medicine bag. For example, airline carriers restrict flammable substances such as ethylene glycol spray and some sprays used for athletic taping, as well as sharp objects that are frequent components of a physician's medical bag. Internationally, some prescription medications such as

narcotics may be illegal to carry into some foreign countries and may place the physician at risk of being accused of drug trafficking. The U.S. Embassy can provide such information before foreign travel. Depending on where the team travels internationally, a ready supply of emergency equipment that we have come to expect domestically, such as spineboards and external defibrillators, may not be available. Another aspect of traveling with teams domestically and abroad arises when the team is comprised of minors. Traveling team physicians should make sure that each player's parent or guardian has completed *written* treatment authorization forms in case the need for treatment arises and the parent or guardian cannot be reached.

THE GOOD SAMARITAN

Many physicians volunteer their time to serve as team physicians and/or to perform preparticipation evaluation. Other physicians fear legal liability so they do not volunteer their time. Because the United States is such a litigious society, it is quite reasonable for prospective team physicians to question whether they are protected from a lawsuit under a Good Samaritan statute. Good Samaritan statutes were enacted to protect persons who offer medical assistance to those in need of treatment from liability[1] and exist in some form in all 50 states.

Unfortunately, it is not possible for a team physician to be totally protected from liability. Anyone can be sued; however, just because a case is filed does not mean it has any merit or will proceed to trial.[4] It is not uncommon for cases to be dismissed or verdicts to be found in favor of the physician in those instances where the doctor voluntarily came to the aid of another person in an emergency situation. In such cases, the Good Samaritan law has been successfully invoked as a defense.[4]

It is essential that team physicians know the parameters of the Good Samaritan law in each state where they will be rendering athletic coverage. Some states, in order to encourage the rendering of volunteer medical care by physicians, have passed Good Samaritan legislation that specifically protect from liability those physicians who volunteer as athletic team physicians and/or who render care at athletic events or in other emergency situations. California is one such state.[4,6]

The parameters of Good Samaritan laws can vary from state to state. As a general rule, however, most state Good Samaritan statutes protect a particular class of persons (i.e., some states protect all persons rendering assistance, while other statutes specifically protect particular healthcare providers such as physicians and nurses) acting in good faith in an emergency setting, where the provider's assistance has conformed to the profession's conduct standards under the circumstances and where the assistance has been provided without compensation.[1] In other words, the Good Samaritan must rescue or aid with honest intentions (good faith) at an emergency scene (such as an automobile accident, sporting event, or other locations specified by statute, normally not in a hospital or office), performing the aid with the minimum standard of care (without negligence) and performing the aid gratuitously (for free). Most significantly, team physicians must ensure that they are not receiving compensation for their service, or their protection under a state's Good Samaritan statutes could be invalidated. Receiving a team shirt, a free meal, or bus ride with the team has not been deemed to be monetary compensation.[1]

THE PREPARTICIPATION EVALUATION

Physicians are often asked to perform preparticipation evaluations (PPEs) to certify if an athlete can participate in certain sports or activities. Many school systems and states require these evaluations.[7] It is important to be aware that although many physicians perform PPEs without charge, most state Good Samaritan statutes do not protect physicians from potential liability, such as failure to diagnose a medical condition or injury, as a PPE is not an emergency evaluation.[4,5] One area of the PPE that poses the greatest liability risk is failure to diagnose a cardiac abnormality, such as an arrhythmia or other anomaly, that can be associated with sudden death in athletes. This is one reason why physicians must be in a setting during PPEs where they can adequately auscultate the heart. Unfortunately, many PPEs are conducted in mass settings that may be quite noisy. One of the most publicized legal cases involving cardiac sudden death in an athlete surrounded the death of basketball star Hank Gathers.[8] The cardiac anomaly known as hypertrophic obstructive cardiomyopathy (HOCM) has been associated with sudden death in athletes and can be a significant challenge for sports physicians because HOCM is often asymptomatic and thus not easily identified during a medical history and/or physical examination.[4] HOCM may be identified in some cases by echocardiography; however, it may

only be visible under exercise conditions. Another cardiac anomaly associated with death in athletes is rupture of the aorta in an athlete with Marfan's disorder. An athlete who has a prior injury that was inadequately rehabilitated and is subsequently re-injured by participating in sports poses another source of liability for physicians. Athletes or their families have been known to sue sports medicine physicians, alleging negligent failure to discover latent injuries or physical defects.[5] The best protection from liability is for the physician to adhere to accepted sports medicine practices under the circumstances. Sometimes athletes fail to disclose information about medical conditions or prior injuries on their history or, in the case of minors, they may have signed their history sheet without their parent's knowledge and input. A physician can be held liable if found negligent in determining fitness or giving sports clearance that subsequently results in a causal injury.

Being aware of the potential liability from performing PPEs, physicians should pay close attention to their selection of venue in which to perform those evaluations. Some of the venues include individual visits by athletes to the doctor's office; a location such as a gymnasium, where multiple athletes are seen individually at sequential stations where each examiner performs one section of the exam; or locker room *en masse* examinations. Although station-based rotations or locker room examinations allow a large number of athletes to be examined efficiently, drawbacks of these methods include noise and lack of privacy, which may preclude an adequate history taking and make it difficult to auscultate the heart and lungs effectively. In the station-based type of examination, someone must sign the ultimate clearance determination, and this raises the question as to whether a physician wants to assume potential liability from another provider's assessment, particularly the cardiovascular evaluation.

Preparticipation Evaluation Standards

In order to guide preparticipation clearance determinations, many professional medical organizations have issued standards that address common medical problems that athletes desiring preparticipation clearance may have and which may increase their risk if participating in certain sports.[7,9–11] Sports can be classified according to the two subsets of degree of physical contact between players and strenuousness.[7,9] The contact subset divides sports into three categories: contact/collision, limited contact, or non-contact (see Appendix II). For example, football is considered a contact/collision sport, while baseball is a limited contact sport, and swimming is categorized as a non-contact sport. The strenuousness subsets consist of four classifications based on intensity and the dynamic and static aspects of the sport: high- to moderate-intensity dynamic and static demands (e.g., football); high- to moderate-intensity dynamic demand and low-intensity static demand (e.g., soccer); low-intensity dynamic demand and high- to moderate-intensity static demand (e.g., karate); and low-intensity dynamic demand and low-intensity static demand (e.g., golf).[7]

Another source for athletic clearance guidance can be found in the guidelines from the 26th Bethesda Conference which are used for clearance of athletes with cardiovascular problems. When evaluating adults for cardiovascular clearance, it is helpful to utilize the American College of Sports Medicine's *Guidelines for Exercise Testing and Prescription*[12] to ensure that appropriate risk screening for cardiovascular diseases is being conducted. Even using appropriate guidelines to help determine clearance, some athletes may incur serious injury or death before the standard cardiovascular screening period, such as the death of 33-year-old St. Louis Cardinal pitcher Darryl Kile in the summer of 2002.[13]

A physician who has completed the history and physical examination of a person desiring participation in sports should render conclusions about clearance or the need for specific evaluations before a clearance determination can be finalized. The physician should document the decision in writing and include discussion of decision-making points if an athlete is not given clearance. Sometimes members of a school athletic staff or a sports organization may help to expedite any further evaluations that are needed, but physicians are limited by confidentiality as to what medical information they can divulge without the express consent of the adult athlete or the parent/guardian of a minor athlete.

Preparticipation Evaluation: Athletic Clearance

After the PPE and appropriate follow-up evaluations (such as an echocardiogram) are performed, the clearance determination is made. The four types of clearance assessments for sports participation are (1) unrestricted clearance, (2) clearance after completing further evaluation or rehabilitation, (3) restricted clearance for certain sports, or (4) not cleared for any sports. The sports classification guidelines (contact/strenuousness) are

very useful for determining the sports in which an athlete is physically capable of participating and for counseling an athlete who may have a restricted clearance as to which sports to consider. Considering the findings of the PPE, the 26th Bethesda classifications, and the effect of various medical conditions on sports participation[9] can help physicians to determine which activities, if any, are safe for a particular athlete. For example, if an athlete has a medical condition that warrants restricted clearance for a contact/collision, high-dynamic sport such as soccer, that athlete may be cleared for a non-contact/low-dynamic and static sport such as golf.

Determining risk is essential when clearing athletes. For example, during the musculoskeletal assessment portion of the exam, the physician should carefully examine previous injury sites, as this is a frequent source of liability. Sport-specific evaluation should also be performed, such as examining a soccer goalie for broken fingers or deformities or evaluating the shoulder range of motion and stability of a baseball pitcher's throwing arm. When evaluating minors, physicians should also assess the physical maturity level of the athlete, which can be helpful when discussing risks of participation in sports where age cutoffs are used to divide athletes rather than size.

Physicians should make clearance determinations based on the standard of care and in the best interests of the athlete. The physician should not be influenced by outside considerations such as a team official or recruiter. Even though physicians exercise their best medical judgment to make clearance determinations, some special situations require additional documentation, such as athletes with unpaired organs or cardiac abnormalities. An athlete with unpaired organs or cardiac abnormalities who wants to participate in any contact/collision or limited contact sports should be given a thorough explanation of the choice of sports and the inherent risks. For example, an athlete with only one eye who desires to play a racquet sport should wear eye protection for that eye.

The decision to exclude an athlete from participation in a particular sport, like the decision to permit an athlete to play a particular sport, can be fraught with unexpected legal repercussions. Some athletes use legal avenues to reverse a team physician's or school district's clearance determinations.[4] In *Pace v. Dryden Central School District* (1991), a 17-year-old male with a solitary kidney was prohibited by the school district physician from participating in the high school football and basketball interscholastic athletic programs. His parents filed a lawsuit to compel the school to permit him to play. As part of their case, the parents submitted evidence showing that the particular health risks involved with the selected sports had been discussed with the family and that the student's own physician as well as a urology specialist had attested that the student's participation in contact sports was, in the opinion of the physicians, "reasonably safe." The court reversed the school district's decision and permitted the student to play.[14] Most significantly, the court held that, by virtue of its legal challenge to the restriction, the family waived any future liability claim against the school district in the event that the athlete's kidney was injured. The import of this decision is that litigation is often inevitable whether an athlete is cleared or not cleared for participation in sports. Team physicians should always rely on the clearance guidelines when making their determinations even if they are subsequently challenged in court.[4,5] It is important to note that if a court overrules a physician's clearance decision and permits an athlete to play or if an athlete has been conditionally cleared after the athlete has signed a written waiver of risk, the team physician continues to have the responsibility to help the athlete participate in that sport as safely as possible. Thus, if an athlete with an arrhythmia is playing a particular sport, whether by court order or by the athlete's decision after waiving the risks of participation, the team physician must ensure that a defibrillator is within immediate reach and that people are present who are trained in its use.

Although athletes do not have a constitutionally protected right to participate in sports,[15] they need to be given due process under the law if they are excluded from participation due to medical reasons. Thus, constitutional problems can occur if a high-risk athlete is barred from play due to medical condition.[16] Multiple laws prohibiting discrimination, arbitrary classifications, and disparate treatment can be applied to the athletic context.[16] These include the Federal Rehabilitation Act of 1973, the Individuals with Disabilities Education Act of 1989, and the Americans with Disabilities Act of 1990.[17–19] Athletes have utilized these laws to challenge team physicians' decisions to exclude athletes from participation. For example, in *Lambert v. The West Virginia State Board of Education* (1994), a school district excluded a deaf student from participating in basketball. The student took the district to court and the court decided that the Federal Rehabilitation Act and

the Individuals with Disabilities Education Act barred such discrimination against the handicapped student. The court ruled that the student could participate in the sport by using the services of a signer.[20]

Of particular concern are conditions involving cardiovascular conditions. A high school football player passed out at practice and was found on evaluation for the syncope to have a heart defect (hypertrophic cardiomyopathy); when he was subsequently restricted from playing football he sued his high school and the Archdiocese of Cincinnati in order to be permitted to play. The court in *Larkin v. Archdiocese of Cincinnati* (1990) ruled that schools have the power to enforce medical standards, even if that means barring some athletes from competition.[1,16,21] The Americans with Disabilities Act has also been used to challenge potentially discriminating practices by sports leagues to inhibit the ability of an athlete with a physical impairment to compete. In *PGA Tour, Inc. v. Martin* (2001), the court ruled that denying the use of a golf cart to Casey Martin (a professional golfer with a circulatory disorder that impairs his ability to walk), per the policy of the PGA Tour that all golfers must walk during tournament play, violated the Americans with Disabilities Act and Martin's request for a waiver to the "walking rule" should have been granted.[22]

RISK RELEASES/WAIVERS OF LIABILITY

In school systems and universities and in professional sports, athletes may be at risk by choosing to participate in specific sports. One of the responsibilities of the sports physician is to inform prospective athletes (or their parents or guardians) of the risks involved in participating in that sport. Schools and athletic organizations often have preprinted written handouts that include sport-specific information. In cases in which athletes have increased risks participating in sports due to known medical conditions, physicians should carefully document those risks and the potential consequences of participation on the risk release sheet that the adult athlete or parent or guardian of a minor must sign.

The rationale for these waivers is to try to protect the school or athletic organization and its staff from being sued. The premise is that if an athlete (or parent or guardian) is aware of the dangers or inherent risks of a particular sport and chooses to participate in the sport anyway, then the adult athlete (or parents, if a minor) have assumed the risk and the school district, team, athletic organization, or activity sponsor and physician are insulated from liability. Waivers and risk releases do not always protect the parties from liability. Some states do not accept some types of waivers of risk as legally binding. As with any contract, the courts are often used to determine whether a waiver is valid and binding. The courts consider whether the waiver was clearly written, specific, and not subject to different interpretations.[16] Additionally, if a physician is negligent regarding treatment of an injury that occurs in that sport, the waiver of risk in the decision to play does not absolve the physician or athletic personnel from liability regarding injury negligence.

Sports medicine physicians should be knowledgeable about the laws in the states where they are practicing because they differ in the legal interpretation of risk release waivers or contracts. The safest approach is for physicians to use their best medical judgment when determining whether a person should be cleared to play a particular sport.

RETURN-TO-PLAY DECISIONS

A major area that exposes a sports physician to potential liability from negligence is making return-to-play decisions after injury. The sports physician is the one who must determine whether or not an injured athlete may safely resume participation in the sport. Even though there are often pressures from the athlete or coach to reduce the time an athlete is restricted from participating in the game, the team physician must never compromise the athlete's safety. The physician must inform the athlete of the risks of playing injured if the athlete is allowed to return to play. The standard of care to be used when making return-to-play decisions is to consider what a reasonably prudent physician would do under the same or similar circumstances.[1] One of the most common return-to-play decisions concerns players with head injuries. When a football player sustains a grade 1 concussion and initially complains of some dizziness when exiting the playing field but no loss of consciousness occurs and the dizziness subsequently resolves despite a mild headache, should the player be allowed to resume play?[23] Utilizing the reasonably prudent physician standard, the existence of the postconcussive headache would be a significant factor causing the athlete to be barred from play, at a minimum, for the remainder of the game.[1,23] Prior to engaging in athletic coverage, it is helpful for the physician to clarify, in writing, the role of the

team physician for making the ultimate return-to-play decisions. This can minimize the return-to-play conflicts that can invariably arise during a game.

TREATMENT

In contrast to emergency treatment that may occur on the sidelines, elective or non-emergency treatment requires informed consent from an adult athlete or from a parent or guardian if the athlete is a minor. In situations where a parent is unavailable, a permission-to-treat form signed by the parent may be used for basic treatment of a minor. Informed consent means giving the athlete sufficient information to make an informed decision about whether or not to accept medical advice. When an athlete allows himself to be treated by a team physician without objection, it is considered implied consent.[3] The elements of informed consent include explaining the nature of the medical condition or diagnosis and treatment, inherent risks of the condition or treatment, and the likelihood of success of the treatment, alternatives to the treatment, or risks of not accepting treatment. An example of informed consent would be to inform an athlete of options for treating a painful injury with an anti-inflammatory medication such as ibuprofen plus discussing potential risks that the drug can have on various organ systems and the likelihood that it will work vs. other options for treatment or lack of treatment.

CONFIDENTIALITY

Until the advent of the Health Insurance Portability and Accountability Act of 1996 (HIPAA), it was understood by the legal community that the communications between a doctor and patient were privileged unless the privilege was expressly waived by the patient. Sports physicians had a legal responsibility to protect an athlete's medical information, even about injuries, from athletic staff, teammates, and press unless given specific permission to release that information. Now, under the HIPAA, not only physicians but also health plans, covered schools and universities can all suffer significant civil and criminal penalties for failure to protect the confidentiality of the patient (athlete).[24,25] Health providers may only disclose specific patient information to other health providers solely for healthcare treatment purposes. HIPAA also limits the information a physician may share with a school or university or that they in turn may share with the public. This presents a dilemma when an athlete sustains a severe injury in a nationally televised athletic contest and sports teams and the public want information about the athlete's condition and the injury.[24,25] Physicians must be very careful not to release any information without the express consent of their patients.

The HIPAA's *Privacy Rule,* which has the greatest impact on the practice of sports medicine, creates national standards to protect an individual's personal health information, gives patients increased access to their medical records, and affects the way health information is shared (see Section 45 CFR, Parts 160 and 164).[24] Sports medicine providers, schools, and sports teams need to confer with their legal counsel to determine how they will comply with HIPAA. Some specific items from the HIPAA Privacy Rule that will be discussed here include the Notice of Privacy Practices (NPP) and the Authorization To Release Protected Health Information (PHI) because they affect the practice of sports medicine and applicable consent procedures.

Notice of Privacy Practice (NPP)

Healthcare providers are mandated to make a good faith effort to get a *written* acknowledgement of receipt of the NPP from a patient. The NPP describes the uses and disclosures that may be made of a person's PHI. The HIPAA does not prescribe a specific form for the NPP or require that the patient sign the actual NPP form, so the patients can sign a separate sheet or cover sheet to acknowledge they have read the NPP. Emergency treatment situations are exempted from the good faith effort requirement. However, as soon as is reasonably practicable after the emergency situation, the good faith effort to obtain patient acknowledgment of receipt of the NPP applies. [45 CFR 164.520(c)(2)(ii)].

Authorization to Release Information

Healthcare providers (including sports medicine providers) and other covered entities are required to obtain authorization for disclosure of protected health information (PHI) and for non-routine uses of PHI to parties that are *not* part of the chain of healthcare providers (e.g., the media, teammates, athletic directors). According to the National Athletic Trainers Association Governmental Affairs Committee, it is unclear whether athletic trainers fall under the category of "covered entities" (in their various employment settings) under the HIPAA. While the legal standard is evolving, it is recommended that all teams, sports governing bodies, schools, healthcare providers, and athletic staff consult legal counsel to create appropriate policies and authorization to release PHI forms.

According to the HIPAA, the list of required elements that must be present for authorization of the PHI to be valid include:

- Description of the information to be used or disclosed
- Identification of the persons/class of persons authorized to use or disclose the PHI
- Identification of the persons/class of persons to whom the "covered entity" is authorized to make use or disclosure of PHI
- Description of the purpose of each use or disclosure
- Expiration date or event
- Individual's (patient or representative) signature and date
- Description of a personal representative's authority to act for the individual if not signed by the individual
- Statement that the individual may revoke the authorization in writing (including statement on right to revoke and instructions on how to do so or reference to NPP if that notice already includes this information)
- Statement that treatment, etc. is not conditioned on obtaining signed authorization where prohibited by the HIPAA privacy rule
- Statement that PHI may be redisclosed by the recipient

The individual must be provided with a copy of the signed authorization form.

It is important to note that the patient (athlete) is supposed to grant permission in advance for *each* disclosure or non-routine use of the PHI on a per incident (per injury) basis. This brings into question the validity of blanket, universal authorization forms. Also, in cases involving minors, state law supercedes the federal HIPAA so that sports medicine providers need to be aware of applicable state laws regarding disclosure of information about a minor to a parent. Even under the HIPAA, treating physicians of any patient (including athletes) can use and disclose whatever information they deem necessary for the treatment of the patient to other members of the healthcare treatment team (for example, releasing information about an injured athlete to an emergency room physician who will be assuming treatment of the patient). What is limited is disclosure to other parties, such as the media, athletic staff, or other team members.

This is not an all-inclusive discussion of the HIPAA, and more information can be obtained by accessing the websites www.hhs.gov/ocr/hipaa/ or www.aafp.org/hipaa.

Infectious Disease

Potential confidentiality issues can occur when athletes who possess infectious diseases such as hepatitis B, human immunodeficiency virus (HIV), herpes simplex, and fungal infections choose to play sports in which potential transmission to other athletes could occur. The physician should inform the athlete about the transmission risks of the infection and should discuss with the athlete precautionary steps to be taken to prevent transmission.[26] Of note, the American Academy of Pediatrics Committee on Sports Medicine Guidelines recognizes that the chance of transmitting the HIV virus during sports is extremely low and recommends that HIV-positive athletes be permitted to participate in all sports.[26] Thus, even though a nominal risk exists of blood exposure with HIV-infected athletes who choose to participate in contact sports, physicians must maintain the confidentiality of these athletes and not disclose their HIV status to others (including the athletic staff) without the express consent of the athletes.

Prescribing Medication

Prescribing medication also raises confidentiality issues. At times it is important to inform both the athlete and someone other than the athlete of the potential side effects that might occur from specific medications, but doing so may compromise an athlete's confidentiality. In those instances, the physician should explain why it would be helpful for someone other than the athlete to be alert for side effects and obtain the athlete's permission to give limited disclosure. Additionally, a physician can incur liability from prescribing a medication that will cause an athlete to be disqualified or restricted from participation if found on drug testing. The physician must be aware of all drug regulations and all medications that are on the banned list of athletic governing bodies such as those published by the National Collegiate Athletic Association,[27] the U.S. Olympic Committee, and other athletic organizations. For example, an unsuspecting physician may prescribe an over-the-counter cold preparation that contains ephedrine and violates the drug prohibitions of the sport in which the athlete competes. If a question arises about use of a medication that is not clear from any published list, the physician could contact the

sports organization without identifying the athlete to clarify if a specific medication may be used.

Drug Testing

Illicit drug use by athletes also creates confidentiality issues. Drug testing has been implemented in many collegiate and professional sports and some high school athletic programs with the goal of maintaining competitive fairness. However, when an athlete discloses to the team physician that he or she is using performance-enhancing or illicit drugs, the team physician cannot disclose that information without the specific consent of the athlete. Due to the potential conflict of interest and risk of compromising the physician–patient relationship, it is advised that the physician who is involved in an institution's drug testing compliance be someone other than the team physician. If an athlete admits to the use of such drugs as anabolic steroids or cocaine, the team physician should take advantage of that opportunity to have an honest, balanced discussion regarding the risks and benefits of the particular drug with the athlete in an attempt to discourage its use.[4]

RISK MANAGEMENT

Sports medicine litigation is on the increase. In order for the sports medicine physician to deter litigation or prevail if litigation occurs, it is important for sports medicine physicians to practice the "4C's" of good risk management: *compassion*, *communication*, *competence*, and *charting*.[1] *Compassion* is exhibited by those physicians who foster good relationships with the athletes and athletic staff and perhaps, not coincidentally, are less likely to be sued even if a bad outcome occurs. *Communication* focuses on the exchange of information between the patient and the physician. Physicians who communicate effectively are those who give the athletes and parents (if the athlete is a minor) clear informed consent about the risks of sports and the risks and treatment options for certain conditions or injuries. *Competence* is found in physicians who stay knowledgeable about sports medicine and deliver the required standard of care to the patient athlete. The fourth component, *charting*, concerns the physician's duty to maintain complete medical records. Meticulously prepared medical records can save a physician untold grief if litigation ensues. The team physician must ensure that an organized system for record retention and retrieval is in place. Each patient's file should, at a minimum, contain the preparticipation physical examination record.[4]

Although team physicians often practice medicine on the field instead of in an office, the need for specific medical records is not removed. If a physician renders sideline care without much time for detailed documentation, the physician (as well as any assisting athletic trainers) should use a notepad to contemporaneously document the essential medical information. Then, as soon as feasible, the physician should complete the notes, make a copy, and place the document in the patient's file. The record should be legibly written and signed by the physician and should contain the athlete's name, sport, date of event, assessment of injury or illness, immediate treatment, and further recommendations for treatment and rehabilitation.[1,4] The storage of that information must also be in compliance with HIPAA regulations regarding storage of identifiable patient information.[24]

Medical records should be maintained by the team physician for extended periods of time, often beyond the time an athlete is participating on a particular team or organization. Physicians who care for minor athletes who may be able to sue for medical malpractice after they reach the age of majority (depending on the laws of their state) should consult with an attorney before destroying any medical records.

While the "4C's" of good risk management are useful for all physicians, two additional "C's" can be added to the list for the team physician: *contract* and *confidentiality*. The *contract* should be between the team physician and the entity for which the physician is providing services. The team physician should insist upon a contract or letter of agreement that delineates what services he or she will provide the team. The contract should also specify who provides the insurance coverage for those services, including event coverage for games and practice sessions. If the team physician is a volunteer (i.e., provides services without remuneration), this should be made part of the contract or agreement. Such an inclusion could prove to be pivotal evidence if the team physician attempts to use a Good Samaritan defense for litigation resulting from providing medical care in an emergency situation. Finally, the contract or agreement should include a statement that the final decision on participation or return-to-play considerations is to be made by the team physician after providing informed consent to the athlete. Maintaining the *confidentiality* of the physician–athlete relationship and adhering to the HIPAA rules (as the legal standard evolves) are also paramount to good risk management.

SUMMARY

The practice of sports medicine can be very enjoyable if the fear of liability does not dampen one's enthusiasm. To diminish fear of liability and provide excellent care to athletes, the sports medicine physician needs to be aware of the legal aspects of sports medicine and the specific laws that apply to the practice of sports medicine.

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5 THE ROLE OF THE PHYSICIAN IN SPORTS: ETHICAL CONSIDERATIONS IN SPORTS MEDICINE*

Ralph Oriscello

INTRODUCTION

Ethics is the conforming to accepted professional standards of conduct. It would be extremely difficult if not impossible to write a prescription for ethical behavior. Almost instinctively, one knows if a thought or action is ethical or not. All physicians have anguished over conflicts between ethics and expediency. No one achieves ethical perfection but most physicians are good by nature and are guided by high ethical standards. The main reason for insisting upon the highest ethical standards by physicians involved in sports medicine is that sport is considered to reflect values generally considered to be important to society: character building, health promotion, the pursuit of competitive excellence, and enjoyment.

SPORTS PHYSICIAN RESPONSIBILITIES

General trust continues to exist between a physician and any private patient who is also an athlete, whether amateur or professional. The physician always makes a decision in conjunction with the desires of his or her patient that is in the patient's best interest. Whether the decision is regarding eligibility to participate in an event or to undergo diagnostic or therapeutic intervention, the end result is the maintenance of good health with the least risk to the patient/athlete. Conflict is minimal to absent. While we live in an era of informed consent, confidentiality, and truthfulness, patient autonomy reigns. Nevertheless, many patients still rely upon their physician to lead them in the decision-making process. In the majority of cases, this works very well.

Sports medicine is little different ethically from other aspects of medical practice. It does contain unique traps that can cause a physician to stumble if unaware. Included in this chapter will be suggestions to make decision-making by sports physicians less difficult while still recognizing that rarely does one solution fit all and that more rarely is the sports physician's decision the only one under the circumstances. Exactness and infallibility, while desirable, are not traits of even the finest physicians.[1]

The primary duty of the sports physician is to expend his or her best effort and judgment for the athlete-patient so as to maintain or restore health and functional ability.[2] The athlete's welfare must be the guide for all efforts. The physician must seek out and try to know the athlete's goals and motivations. Importantly, the physician must have a genuine appreciation for the importance of athletics in human life. Dr. O'Donoghue, [3] in his now classic text, lists five precepts for the sports physician:

* In the second edition, this chapter was authored by Warren B. Howe.

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- Accept athletics.
- Avoid expediency.
- Adopt the best method.
- Act promptly.
- Achieve perfection.

The ill or injured athlete must know the diagnosis, understand its implications, and participate in all therapeutic decisions. Despite the athlete's wishes, the physician cannot do less than seek the best possible outcome. While all recommendations or forms of therapy have risks as well as benefits, knowledge and judgment can come only from experience. A medical degree does not ensure perfect decision making. Harm can come to the athlete-patient from unnecessary or excessive restrictions as well as from failure to restrict activity when appropriate.

Periodically, the physician/patient relationship is strained by a difference of opinion. If negotiation between the two fails, even with accepted intermediaries (family, other consultants, financial or religious advisors), the relationship may be terminated. The physician involved in making sports-oriented medical decisions must be well versed in current recommendations for eligibility and continued participation and not depend upon his limited personal experience or unscientific reasoning.[4] For the most part, patients themselves end the relationship with their physicians if the answers they wish to hear are not forthcoming. Under this set of circumstances, ethical conduct is not breached with the patient acting unilaterally. Recognizing the wide range of opinions and individual fallibility, the patient can assert his right to another opinion.

A sports physician must develop a suitable level of skill and knowledge and then maintain it.[5] Primary care sports physicians generally know their level of competence. This group can definitively care for about 85 to 90% of athletic problems but must know when to refer for specialized consultation or therapy. The referring physician must know the consultant's ability, personality, and, empathy for athletes in order to make a competent referral.[6] The primary care physician should not abandon the care of the referred athlete and must maintain surveillance over the referral/therapeutic process. The consultant may gain insights from the referring physician, and the athlete is then afforded the continuing support of his or her physician. Referring physicians can always question the recommendations of consultants if they seem incongruent with their knowledge of the patient. The trust established between athlete and physician, more likely than not, allows for resolution and comfort with the decision-making process.

POTENTIAL FOR DIVIDED LOYALTIES

While rare at the high school level and uncommon at the college level, major distrust does exist among professional athletes and team physicians.[7] The athletes feel that too many times the quality of their treatment is secondary to the doctor's obligation to team owners and coaches. A salaried position can interfere with a traditional physician/patient relationship. To many, the role of the salaried physician leads to a conflict of interest. A conflict of interest for a physician exists when his or her objective professional duties are compromised by personal interests (e.g., the financial reward of being associated with a professional team, as well as the publicity and high visibility one gets from such a position). At any and all levels, it is an ethical breach for anything but the athlete's best health interest to be considered, recognizing that judgment errors in regard to too conservative or too liberal therapy can occur.

At times there may seem to be confusion as to where the loyalty of the sports physician lies. The ultimate welfare of the athlete may seem in conflict with the wishes of parents or spouse, coaches or team management. The fact that an entity other than the athlete pays the physician is immaterial. The loyalty of the physician is to the physician/patient relationship. Decisions must be based solely on sound medical judgment. Reasonable third parties will understand this. If any party insists otherwise, physicians should consider removing their services from that party. It is not infrequent for the wishes of the patient to conflict with what the physician believes is in the athlete's best interest. These situations are very delicate, require much effort in explanation, and may indicate the need for further consultation. If, after further consultation, the treating sports physician still feels uncomfortable with another recommendation, continued care of such an athlete-patient may be difficult if not impossible. Reassignment to another physician should be strongly considered.

At the highest level of sports, the unfavorable mix of high salaries and short careers makes for risky decision making by both the athlete and the physician. Coaches often encourage physicians to rush players back to the playing fields to win games. Players themselves often desire to rush back too quickly. Teammates are thrown into the

mix by suggesting non-playing team members are malingering while collecting a substantial income. Under these circumstances, many physicians play by the rules of the coaching staff. With a substantial number of complications from such behavior, as demonstrated by court-authorized awards to players in the millions of dollars and a current suit seeking $100,000,000 for a heat-related death, players are at the point of not trusting team physicians. Salaried physicians must make the effort to assure players that it is their utmost responsibility to protect each player, and any player who should not be on the playing field will not be there.

DRUG USE

Sports medicine and ethics have several unique aspects. A major one is drug use by athletes at all levels. Therapeutic medications are an integral part of sports medicine practice. Used appropriately, they control pain AND inflammation, speed recovery, and hasten return to function. The physician must understand each drug thoroughly, including its potential effects on the safety or effectiveness of an athlete's performance.

It is never appropriate to use narcotics or local anesthetics to permit activity that would otherwise be impossible. The use of medications that alter the sensorium or affect coordination must be restricted to times when performance and safety will not be affected. The use of agents that will artificially enhance performance, whether effective or not, is unethical.

The use of drugs, hormones, and blood transfusions invariably is in the public's mind when outstanding performances in any sport are recorded.[8] From baseball to cycling, any new record is suspect. The use of ergogenic substances to enhance performance is often aided by covert, if not overt, support and supply by unethical team physicians. Because of this, the International Olympic Committee has banned five classes of substances: stimulants (e.g., amphetamines), narcotics, anabolic agents (e.g., steroids), diuretics, and peptide hormones (e.g., erythropoietin, growth hormone). Sports physicians can neither condone the use nor participate in supplying any of these agents. The downfall of the individual, the program, and the physician is inevitable.

Some appropriately used medications are banned at certain serum levels or types of competition. The sports physician must be familiar with these so that any drugs appropriately prescribed do not expose the athlete to potential disqualification. Lists of banned drugs are published by regulating bodies (e.g., United States Olympic Committee). Usually, when use of a drug to permit athletic performance is appropriate, as in the prevention of exercise-induced asthma, an effective, acceptable medication can be found.

Current available testing makes it impossible to catch all participants whose behavior includes unethical use of banned substances. Because of that, some favor lifting the ban on hormones, for instance, feeling that it would be preferable to have a free-for-all and allow unrestricted use. The major argument against such a philosophy is the need not to condone cheating along with the essence of sport itself.

CONFIDENTIALITY

Confidentiality in medicine should not be graded, more or less, depending upon the individuals public persona. In sports medicine, confidentiality is of the utmost importance, an ethical principle that is inviolate. Athletes are very public persons. Society wants to know the most intimate details of their lives, including medical evaluations and treatments. By being an athlete, amateur or professional, the patient does not forfeit his or her right to medical privacy. Inquiries made of the sports physician by the press or other interested persons should not be answered without the specific permission of the athlete. Even with permission, the physician must be extraordinarily sensitive about details revealed. If any sense of doubt exists about what to tell or what not to tell, it is best to keep the athlete's status in confidence. Despite claims regarding the public's right to know, the right of privacy remains with the patient. It must be remembered that the press is very resourceful in obtaining information. If inaccurate information finds its way into print, the physician may, with the athlete's permission, attempt to correct it.

Information released to coaches, parents, and, spouses most often should be appropriate information about the athlete's care, especially when prohibition or limitation of competition or practice is necessary. The physician's explanation of the benefits of disclosure will usually result in permission from the athlete to do so. In the absence of such permission, the physician must consider the welfare of the athlete, the importance of the information, and the potential for harm or embarrassment to the athlete that might arise from disclosure (or nondisclosure), before sharing or withholding information. No greater breach of confidentiality can occur then if any health information is released to anyone remotely related to

the athlete's career without forewarning the individual.

When physicians are employed by or volunteer their services (highly unusual in this day) to a school, team or similar entity, the expectations of both parties should be agreed upon at the outset, preferably in writing. Frank and open communications will usually forestall misunderstanding and conflict. In all cases, physicians should strive to protect their autonomy in medical decision making so they will be able to maintain their position as advocate for the athletes' welfare.

RELATIONSHIP WITH COLLEAGUES

Many problems can arise when serving as a team physician. Because team members may be receiving care from other physicians, team physicians must be sensitive to their relationships with colleagues. A team physician must never criticize the actions of another physician directly to the patient. Concerns regarding therapy should be discussed with the primary physician in private. Sports physicians can often positively influence their colleagues' care of athletes by such positive input.

A fail-safe approach to handling playing restrictions imposed or removed by the athlete's primary physician is important. No playing restriction should ever be countermanded by the team physician, who should, however, always insist upon the final say in approving the athlete's return to play. Consultation between the team physician and the athlete's primary physician usually solves the problem and may provide an opportunity for education.

Sports medicine is a team effort involving physicians plus representatives of many paramedical disciplines. Sports physicians should be able to recognize where one or more of these can be helpful and should coordinate the services of all in the care of an athlete. In doing so, sports physicians must insist that such assistants adhere to the same high ethical standards they practice. Unfortunately, athletic medicine has proved a fertile ground for quackery and unproved practices employed in the guise of improving performance. The sports physician has an ethical responsibility to expose these practices and protect athletes from being victimized by them.

Except for the most basic training room treatment, an athlete is best referred back to his primary physician for definitive therapy of any illness or injury with a detailed note describing the team physician's concern.

Medical societies generally have rules about physician advertising, a troublesome area today even though it is permitted. Physicians promoting themselves as experts in sports medicine have an obligation to provide true expertise in that area, the same as experts in any other field.

FEAR OF LEGAL ENTANGLEMENT

Finally, a question remains regarding ethics and the law when the risk of a life-threatening situation or a potentially permanently disabling condition is uncertain. The physician should be most cautious and recommend against participation. It would not be unreasonable to recognize that the athlete may legally challenge such a recommendation. When operating at the highest ethical level with support from the medical literature and the medical community, such an event should never alter a physician's role in the future evaluation of other athletes. They would be best served by such a physician.

SUMMARY

Physicians involved in sports medicine soon realize the awesomeness of the responsibility and the magnitude of potential problems. They must be familiar with the many disease states that affect the ability of athletes to participate without endangering themselves and others. They must be familiar with the unethical means used to enhance performance. They must be aware of resources available to construct an authoritative opinion. They must be devoted to the principles of confidentiality, informed consent, and, truthfulness. They must be aware that occasional decisions may require legal enforcement. Most of all, they must realize that no table of contents exists to refer to for every decision. A backbone, on occasion, is more important than an ethics primer.

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PART II
PRINCIPLES OF HEALTHY EXERCISE

6 PRINCIPLES OF HEALTHY EXERCISE

Kevin D. Steele

Exercise physiology is the study of the functional changes that occur within an organism as a result of acute or chronic exposure to exertion. Exercise is a form of physiological stress, requiring a significant homeostatic adaptation in all organs if it is to be continued. In the absence of physical stress, functional capacity deteriorates. Almost all changes (e.g., heart rate, cardiac output) that occur during exercise are related to the increase in energy metabolism that occurs within contracting muscle.

BIOCHEMISTRY

The hydrolytic cleavage of adenosine triphosphate (ATP) to adenosine diphosphate (ADP) and inorganic phosphate (Pi) produces the energy that powers skeletal muscle contraction.[1–3] Because the muscle stores of ATP are small, these high-energy phosphates must be continually resynthesized at a very rapid rate to allow contractile activity to continue beyond a brief period of time. All cells have three basic pathways to produce additional ATP: (1) oxidative phosphorylation, (2) glycolysis, and (3) creatine phosphate (CP).

AEROBIC METABOLISM

The catabolism of energy substrates utilizing oxygen with the net production of ATP is referred to as *aerobic metabolism*. The site of oxidative phosphorylation is the muscle cell mitochondria, the cristae of which constitute respiratory chains (electron transport systems). The major metabolic substrate at rest and during low to moderate exercise is fatty acids, whereas glucose becomes

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more important at increasing exercise intensities. Both substrates produce hydrogen atoms during beta-oxidation and the Krebs cycle. While 60% of these processes are lost as heat, 40% are captured as ATP. Nonetheless, the complete oxidation of a molecule of glucose produces a net 38 molecules of ATP; similar oxidation of a molecule of fatty acid (e.g., palmitic) yields 129 molecules of ATP. Aerobic reactions provide the major supply of body energy.

ANAEROBIC METABOLISM

The catabolism of energy substrates without the utilization of oxygen is called *anaerobic metabolism*.[4,5] Such energy transfer occurs when CP donates its high-energy phosphate to ADP to resynthesize ATP (a process that occurs within the first 3 to 8 seconds of exercise). A second pathway is the generation of ATP from glycolysis with the production of lactic acid. Although the anaerobic breakdown of glycogen produces only three molecules of ATP, it yields a rapid, though limited (several minutes), energy source for muscular activity.

TYPES OF EXERCISE

Generally speaking, aerobic pathways are utilized during endurance training that incorporates dynamic, rhythmic movement of large muscle masses for prolonged periods of time (e.g., long-distance running, swimming, cross-country skiing, cycling). Anaerobic pathways are involved during brief periods of intense muscular activity such as sprinting, isometrics, lifting, and jumping.[2,3,6] Normally, anaerobic activity also occurs during the first several minutes of endurance exercise in order to make up for the oxygen deficit caused by the lag in blood flow to the muscle and the onset of aerobic metabolism. Blood lactate does not accumulate under such circumstances. During endurance training, aerobic metabolism provides most of the required energy up to the maximal aerobic capacity or oxygen consumption (VO_{2max}). VO_{2max} is the greatest rate of oxygen consumption measured in liters per minute (absolute) or milliliters per kilogram body weight per minute (relative). The relationship of VO_{2max} and heart rate is discussed in Chapter 14. Under moderate exercise conditions, a steady state of oxygen consumption is achieved within 4 to 5 minutes of training initiation (Figure 6.1). Because the VO_{2max} is an important factor that determines an individual's capacity to sustain high-intensity exercise for longer periods, it becomes clear why athletes can outperform non-athletes. The anaerobic

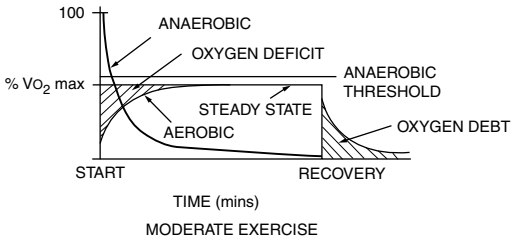

Figure 6.1 Contribution of aerobic vs. anaerobic metabolism during moderate exercise.

or blood lactate threshold occurs at about 50 to 55% of the VO_{2max}, a point where the generation of ATP via anaerobic glycolysis begins to exceed aerobic energy supplies from oxidative phosphorylation.[4,5]

This point is also known as the onset of blood lactate accumulation (OBLA). OBLA is a standard value, typically set at either 2.0 or 4.0 mmol lactate per liter of blood, and is a very accurate reference point. Trained individuals can tolerate and more efficiently metabolize elevated levels of lactate, which, in part, may explain their superior performances during competition. Specifically, experienced endurance athletes demonstrate an improved ability for lactate turnover during their events.[45–49] This enhanced capability is largely due to a mechanism called the lactate shuttle.[49] During lactate production in fast-twitch muscle fibers, lactate can circulate to other fast-twitch or slow-twitch fibers to convert via glycogenolysis for entry into the citric acid cycle for energy in aerobic metabolism.[45,46,50,51] Nevertheless, during exhaustive exercise (Figure 6.2), a steady state cannot be achieved; blood lactate accumulates due to the increased contribution of anaerobic pathways, fatigue and exhaustion occur, and the oxygen deficit and debt become large. Lactate shuttling can expedite recovery in highly trained

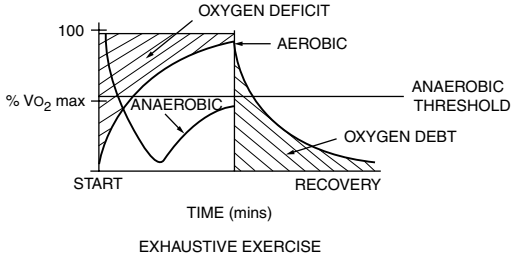

Figure 6.2 Contribution of aerobic vs. anaerobic metabolism during exhaustive exercise.

individuals; this occurs due to muscle tissues being not only responsible for lactate production but also primary locations for lactate removal via oxidation.[45,46,49,52] Submaximal aerobic exercise facilitates the recovery process; thus, a spectrum of activity is equally matched by differing metabolic pathways.

Anaerobic pathways predominate in short bursts of strenuous exercise (e.g., golf swing, 100-yard dash, volleyball spike), whereas training of long duration (e.g., marathon, triathlon) utilizes mainly aerobic mechanisms. Intense exercise of intermediate duration (e.g., middle-distance running, swimming, soccer, basketball) represents a blend of both anaerobic and aerobic energy inputs. The physiologic adjustment to exercise is dependent on several factors: (1) the age, sex, habits, health, and genetic endowment of the individual; (2) the type of exercise (the intensity, duration, and muscles involved in the movement); and (3) the environment in which the activity is performed (heat, cold, altitude, haze, etc.).[7] Adaptations in physiological function that aggregate as a result of regular training not only benefit the individual in the short term but may even enhance longevity.[7–9]

CARDIOVASCULAR RESPONSES TO EXERCISE

The major function of the cardiovascular system during exercise is to deliver blood to the active tissues. Circulating blood transports oxygen and fresh nutrients while removing metabolic waste products, dissipates heat by increasing blood flow to the skin, and transports and distributes regulatory substances (hormones, etc.) from sites where they are produced to target tissues. The average individual's cardiac output is 4 to 6 L/min with an extraction of 40 to 50 mL of oxygen per liter of blood and a total oxygen uptake of 0.2 to 0.3 L/min. A very fit person can increase oxygen uptake from 0.25 to 5.00 L/m or more when exercising; during maximal exercise, the cardiac output can go from 5.0 to 30.0 L/m, or 6 times the resting level.[6,10–12]

Heart Rate

Heart rate is perhaps the most significant factor increasing cardiac output during exercise. In many cases, the increase in heart rate is linear with the increase in rate of exercise. Heart rate is controlled by factors intrinsic to the heart and by extrinsic neural and hormonal factors. The sympathetic cardioaccelerator nerves release norepinephrine at their endings, causing an increased heart rate during exercise,[13,14] while the parasympathetic vagus nerve releases acetylcholine, leading to a reduction. Additional factors include the state of hydration, temperature, anxiety, altitude, and time between meals. The average resting heart rate in the supine position is 72 to 80 beats per minute (bpm). During submaximal exercise, heart rate is elevated and will gradually plateau when the oxygen requirements of the activity have been fulfilled. As the intensity of the activity increases, the ability of the heart rate to level off is much more difficult to achieve. In dynamic exercise, heart rate rises with increases in work load and oxygen consumption, leveling off at VO_{2max}.[5,15,16] In normal populations, heart rate will increase from a resting value of approximately 70 bpm to a maximum of 180 bpm or more. As the human heart ages, the chronotropic response is attenuated, accompanied by an increase in end systolic volume and a diminished ejection fraction.[8,10] VO_{2max} remains essentially the same in trained individuals and, depending on intensity and duration of activity, is capable of increasing with age.[14]

Cardiac Output

Cardiac output (Q), quantified as liters of blood pumped per minute, is a reflection of the overall functional activity of the heart and is a principal determinant of the rate of oxygen delivery to peripheral tissues such as active skeletal muscles.[2,16–18] Cardiac output is equal to the product of heart rate and stroke volume ($Q = HR \times SV$), the frequency of the heart's contraction (bpm), and the volume of blood pumped by the heart with each contraction (milliliters of blood × contraction).[17] From a resting value of approximately 5.0 L/min, the cardiac output increases as a direct function of the level or intensity of the exercise to a maximal value of 20 to 40 L/min or higher, with the absolute value reflecting the individual's size and state of conditioning.[11,12,17,19]

Stroke Volume

Stroke volume is the quantity of blood pumped by the heart with each contraction and is regulated primarily by factors intrinsic to the heart:

- Return of venous blood to the heart
- Ventricular distensibility
- Ventricular contractility
- Aortic or pulmonary artery pressure

Venous return is the principal constituent. Extrinsic factors to the heart can also affect stroke volume. Sympathetic neural stimulation of the myocardium, as well as epinephrine and norepinephrine released from the adrenal glands, can increase the contractile force of the heart.[11,16,20]

Stroke volume increases with increasing rate of work, up to exercise intensities between 40 and 60% of maximal capacity; it normally plateaus, remaining essentially unchanged up to and including the point of exhaustion.[12] Exercise in the vertical position vs. the horizontal or supine position increases stroke volume, due to a decrease in left ventricular end-systolic volume that results in a greater ejection fraction.[21,22] Resting stroke volume varies from 60 to 100 mL at rest. Maximal volume is about 125 mL for the sedentary individual.

Blood Pressure

Systolic pressure increases in direct proportion to an increase in exercise intensity. As a result of vasodilatation in the vascular bed of active muscles, the peripheral resistance to blood flow is reduced during exercise, but the elevation in cardiac output causes the blood pressure to rise.[3,12,14] Systolic blood pressure is indicative of the force generated by the heart during ventricular contraction. The normal value at rest is approximately 120 mmHg but can rise to 200 mmHg or greater at the time of exhaustion. Activities that involve high-resistance, forceful isometric, isotonic, or isokinetic muscle contractions cause marked increases in systolic blood pressure.[18,23] Diastolic blood pressure remains essentially unchanged during low-resistance exercise, but evidence indicates that, as the resistance increases, increases in diastolic pressure occur.[18] Normal resting diastolic blood pressure is approximately 80 mmHg.

Blood Flow

During exercise, blood is redistributed from inactive tissues to those directly involved in the exercise bout. Only about 15 to 20% of the resting cardiac output goes to muscle, while in exhaustive exercise the muscles receive 80 to 85% of the cardiac output.[13] This shift in flow is accomplished by shunting the blood from the kidneys, liver, stomach, and intestines. For instance, the blood flow to the kidneys and spleen during maximal exercise is reduced to approximately 500 mL/min from the normal resting blood flow about 2.8 L. Coronary blood flow increases with intensity of exercise, from about 260 mL/min at rest to 900 mL/min during maximal exercise.[12] During heavy exercise, blood flow to the skin is also significantly increased as a response of the thermoregulatory system to dissipate excessive heat production generated by working muscle and/or environmental temperatures. If the exercise continues for an extended period of time, the problem of reduction of blood volume due to a loss of water in the form of perspiration becomes significant. With total blood volume decreasing as the duration of exercise increases and continued shunting of blood to the periphery for cooling, the cardiac filling pressure is reduced, leading to a decreased venous return. The decreased return reduces the stroke volume and causes a compensatory increase in the heart rate.[3,16]

The oxygen content of the blood at rest varies from 20 mL of oxygen for every 100 mL of arterial blood to 14 mL of oxygen for every 100 mL of venous blood.[11,14] During exercise, the arteriovenous difference progressively increases, due to enhanced oxygen extraction by the exercising muscles.

CARDIOVASCULAR ADAPTATION TO AEROBIC EXERCISE

Endurance leads to the ability to perform prolonged bouts of work without experiencing fatigue or exhaustion and is comprised of two components. The muscular or local component refers to the ability of a single muscle or muscle group to sustain prolonged exercise, and the cardiovascular component refers to the body's total ability to sustain prolonged exercise.

Heart Rate

Heart rate decreases significantly with regular aerobic exercise. It is not unusual to see a resting heart rate of 40 to 50 bpm or less in a very well-conditioned endurance athlete. Although the range is usually between 55 and 65 bpm,[24] a decrease in heart rate response to submaximal exercise occurs. Work performed by the heart is a direct function of its rate of contraction, both at rest and during exercise, and the reduced submaximal heart rate following training indicates that the heart is working more efficiently. At maximal levels of exercise, little effect on heart rate is observed; the normal response is a decrease of approximately 3 bpm.[12,20] The major function of heart rate during exercise is to combine with stroke volume to provide the appropriate cardiac output for the rate of work being performed. Heart rate recovery time, however, significantly decreases after several weeks of cardiovascular training.

Stroke Volume

Stroke volume increases at rest and during submaximal and maximal exercise. There is a substantial increase at rest following regular conditioning. Left ventricular chamber size is increased

and absolute left mass (eccentric hypertrophy) is also increased with training.[25,26] These changes seem to be the result of a more complete filling of the heart during diastole (preload), resulting in a greater end-diastolic blood volume. Increased mass is accompanied by increased contractility, allowing for a decreased end-systolic volume and an increased ejection fraction.[17,24] Resting stroke volumes range from approximately 90 mL for trained endurance athletes to 130 mL for the highly trained athlete. Maximal stroke volumes vary from 150 mL for participants in regular exercise to 220 mL for the highly trained endurance athlete.[16]

Cardiac Output

Cardiac output at rest or during submaximal work bouts does not significantly change as a result of training.

Blood Pressure

Regular aerobic training tends to reduce resting and submaximal exercise systolic, diastolic, and mean arterial blood pressures.[22] Blood flow to the working muscle during maximal exercise appears to be enhanced by training due to an increase in the number of capillaries in the trained muscle. Diastolic and mean arterial pressures are reduced at maximal exercise.[22]

ADDITIONAL EFFECTS OF EXERCISE

Regular aerobic activity reduces LDL cholesterol and triglycerides but elevates HDL cholesterol for both sexes.[27] Resistance and strength training may also produce favorable changes. Lipid improvements have been confirmed in the 16- to 65-year-old age categories. Renal changes include an antidiuretic effect and preservation of most electrolytes (Na, Cl, Ca, P). Post-exercise proteinuria is common and is related to exercise intensity, not to the duration of the activity.[28] Increased erythrocytes and leukocytes in urine and cylindurea can occur following regular and vigorous training activity. Hemoglobinuria and myoglobinuria are abnormal and are due to the breakdown of red blood cells and muscle tissue, respectively, during prolonged strenuous exercise, rather than to renal dysfunction. The coagulation system, platelet function, and prostaglandin cascade are significantly impaired by acute exercise, particularly in untrained males. Platelet counts increase, platelet aggregation is accelerated, and thrombosis occurs.[29,30] Regular training blunts the effect by decreasing platelet activity and improving the homeostatic balance. Serum enzymes (e.g., aldolase, creatine kinase, lactate dehydrogenase, alanine, and aspartate transaminase), particularly those found in skeletal muscle, increase in proportion to the intensity and duration of the preceding exercise, peaking 24 hours after the event or training session.[31] The effect of duration is dominant; prolonged competitive exercise (e.g., triathlon) produces the highest enzyme activities. High levels are also observed after weight-bearing exercises incorporating eccentric muscular contractions (e.g., bench-stepping), and in males, blacks, and the untrained, although marked individual variation occurs. Elevated alkaline phosphatase levels have been documented in 9- to 15-year-olds who regularly exercise and compete in sports. Exercise, via the sympathoadrenal axis, elevates the hormones of gluconeogenesis, lipolysis, and glycolysis, while lipogenic and glycogenic levels decline or are unchanged.[32] Aldosterone and parahormone levels increase. The reproductive hormones (e.g., progesterone, estradiol, testosterone, prolactin) increase during training. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels remain stable, while the pulse frequency of LH and gonadotropin-releasing hormone decrease. The latter may account for menstrual dysfunction.[33]

Endogenous opioids (e.g., β-endorphin and lipotrophin) increase with significant levels of exercise (>75% VO_{2max}) and are linked to several physiological and psychological changes, including altered pain and fatigue perception, euphoric mood states, menstrual dysfunction, ventilatory depression, and reduced stress hormonal responses.[34] There is considerable intra- and inter-individual variation in response, with the effect lasting 15 to 60 minutes. Finally, acute exercise causes a brief leukocytosis, lymphocytosis, and stimulation of cytoxicity, immunoglobulins, complement, and cytokines. Prolonged moderate exercise appears to enhance the body's immune systems, whereas heavy training or overtraining may impair immunity.[35–37]

CHILDHOOD PHYSIOLOGY

Absolute aerobic power increases from childhood through the teenage years in males; however, it generally plateaus in females at about the 13th or 14th year. In females before the ages of 10 to 12 years, the average VO_{2max} reaches between 85 and 90% of the mean values for males. After sexual maturation in females, the average VO_{2max} reaches only about 70 to 80% of mean values for males.[38,39] The relative VO_{2max} declines linearly for

adolescent females but is relatively stable for males. Differential growth rates of aerobic power and body weight, changes in body composition, and the amount of regular activity are some of the factors thought to contribute to the decline. The changes in aerobic power are paralleled by similar changes in balance, strength, agility, endurance, speed, coordination, and power. Flexibility is greater at all ages for females. During childhood, flexibility is stable or declines slightly for females; it increases during adolescence and plateaus around 14 to 15 years of age. For males, flexibility systematically declines until late adolescence, when it increases.

Consistent training and conditioning of children of either gender produces higher VO_{2max} levels and motor fitness (strength, power, agility, etc.) levels than in sedentary individuals and narrows the percentage difference between the sexes. The mechanism for these improvements is presumably like that in adults.[39] No optimal age or program has been established for training; performance and VO_{2max} are not as strongly related in children as in adults.[38,39] While regular physical activity has no apparent effect on biological maturation or stature, body weight and composition are favorably modified (e.g., a decreased percentage of body fat and an increase in muscle and bone). The impact of training on blood pressure, blood lipids, and glucose metabolism is probably favorable but is unknown at the present time.[40]

EFFECTS ON AGING

Lifestyle, environmental factors, and genetics are all elements that can significantly influence the quality and quantity of life. Regular exercise is an important factor in maximizing and maintaining physiological function and longevity.[9] Regular exercise and physical activity produce significantly higher levels of work capacity in the older individual when compared to sedentary younger persons.[10] Nonetheless, VO_{2max} inevitably decreases approximately 30 to 40% between the ages of 20 and 80 years, owing primarily to declines in maximal heart rate and cardiac output.[41] The decrease is more pronounced in males. Stroke volume and arteriovenous difference may or may not decrease in elderly persons. A longer recovery rate is also characteristic of aging, with an impaired ability of the heart to pump blood to the periphery due to a gradual loss of contractile strength and delays in ventricular filling secondary to loss of muscle compliance.[11,17,27] The efficiency of breathing declines, and muscle strength decreases 20 to 30% by the age of 70 years, due to a reduction of fiber function and number.[38] Loss of bone and joint function also occurs. Regular exercise slows or reverses these trends, and promotes psychological well being and favorable metabolic changes (e.g., improved glucose handling, decreased LDL, and elevated HDL).

SEX DIFFERENCES IN EXERCISE PHYSIOLOGY

The absolute physical capacity for males is 5 to 50% higher than females for all age groups and training backgrounds.[42] The difference is in large part due to differences in body composition (e.g., women have increased body fat, less absolute muscle mass, and smaller muscle-fiber size). The remainder is accounted for by differences in work efficiency, VO_{2max} (e.g., stroke volume, hemoglobin levels), and genetic and behavioral traits. Under similar training conditions, relative increases in strength, muscle force output, and VO_{2max} are similar for both sexes; however, less muscle hypertrophy occurs in females.[43,44] Sex differences have little impact on training mode response, and menstrual cycle does not affect performance; however, increased levels of relaxin and estrogen during the third trimester of pregnancy account for increased flexibility.

SUMMARY

The impact of regular exercise and physical activity on the human organism is pronounced, multifactorial, physiologically predictable, and individually valuable. The quality of life is appreciably improved for all age groups, for both sexes, and for the healthy as well as the handicapped and chronically ill. Recent studies suggest that quantity of life (i.e., longevity) also may be increased.

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7 SPORTS MEDICINE APPROACH TO TRAINING COMPETITIVE ATHLETES*

Warren A. Scott

INTRODUCTION

The Olympic motto, *citius, altius, fortius* (swifter, higher, stronger) characterizes today's modern sports warrior. Athletes all over the world use training and conditioning methods to improve sports performance. Today's competitive athletes, however, may have limited access to quality information with regard to proper training methods, with the result being injury, an overtrained state, or poor performance. The paradox of the situation can be simply stated: training harder than normal will produce a combination of fitness and fatigue. Repeated bouts of training will improve fitness,[1] but eventually fatigue will accumulate and the athlete will begin to "go flat" and fail to respond to additional training bouts. The goal of athletes (and their coaches) is to maximize the athlete's performance *while avoiding injuries*. Successful training cannot occur without a fit, healthy athlete. An injury site is the weakest link in the chain and prevents proper application of training loads. Following a precise plan for training will greatly increase the probability of improving individual performance while preventing injuries and illnesses.

* In the second edition, this chapter was authored by Michael DeBevec.

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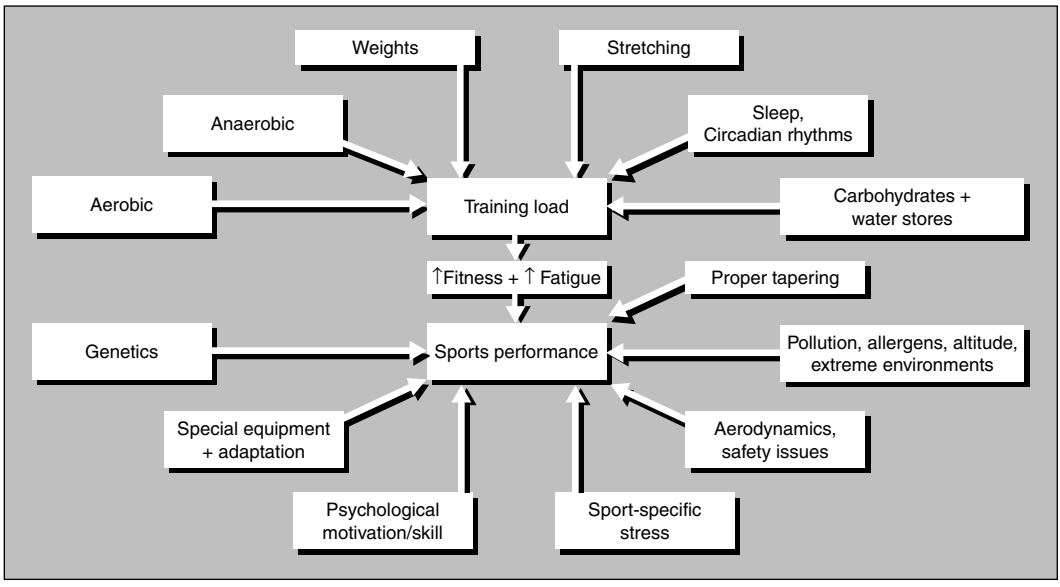

Figure 7.1 Determinants of sports performance. (From Scott, W., *Curr. Sci.,* 1(3): 184–190, 2002. With permission.)

GETTING STARTED

We develop a training program based on a specific goal. For example, consider two different athletes and their goals for exercising. Athlete "A" is 25 years old and wants to get into shape with a running and weightlifting regimen. His goal is to improve his fitness and lose 10 pounds of fat weight. After 9 months of a cross-training program that includes running, weights, and stretching, he is running 3 to 5 miles three to four times a week and lifting weights twice a week. His weight program consists of multiple upper and lower body exercises, and he performs three to four sets of 8 to 10 repetitions of each exercise. The work effort feels medium to hard, and he denies any pains while running or lifting weights. At this point, athlete "A" has reached his goals, and his exercise regimen has reached a steady state. Now his regimen has become a maintenance program, performed with similar effort on a regular basis. The athlete adapts to a specific level of exercise stress,[1–9] and eventually the exercise is no longer stressful. A maintenance program is very important from a public health perspective. A regular exercise routine such as that of athlete "A" can help prevent and/or eliminate major health problems such as diabetes, obesity, hypertension, and cardiac disease (see Chapter 14 on the exercise prescription).

Now compare Athlete "B." He is a 24-year-old former college runner wanting to make the Olympic team in 3 years. Athlete "B" will require a very detailed analysis of his current level of fitness, as well as a training plan sufficient to meet the goal for his desired sporting event. Based on his current level of fitness and previous performances, one can calculate the percentage improvement required to be competitive at the Olympic trials. Training athlete "B" for competition requires a comprehensive plan, including weight training, a stretching program, running, and cross-training, to stress the athlete sufficiently to improve. Research shows that both over-training and under-training produce suboptimal results (Level of Evidence B, systematic reviews).[2–9]

The remainder of this chapter will describe the necessary components of a comprehensive training program (see Figure 7.1) for today's competitive athletes. The sports medicine approach to training includes special emphasis on maximizing performance while also preventing injuries.

BASICS OF TRAINING

Warm Up/Cool Down

The human body, including muscles, tendons, and ligaments, functions best at a particular temperature. From a theoretical standpoint, an increase in blood flow and temperature of the extremities from a warm-up period has several

benefits: (1) better oxygen delivery to the muscles, (2) facilitation of oxygen release from the hemoglobin and myoglobin molecule, (3) enhanced nerve conduction, (4) increased speed of muscular contraction, and (5) decreased viscosity of the musculotendinous unit. Although evidence is limited that warming up prevents injuries or enhances the quality of the training session, it is generally accepted that to prevent injury and optimize physiologic function, warming up is essential.

Choice of activity for warm-up is an individual matter. It should include both general and activity-specific exercises in the broad categories of flexibility, strength, and coordination, such as walking, light jogging, stretching, low-level calisthenics, and other light-resistance exercises. Fifteen minutes of an aerobic sport such as jogging, cycling, rowing, or swimming should be followed by stretching all the major muscle groups. The athlete should then engage in the primary sport, beginning at an easy level and building the intensity of activity over the next 15 to 30 minutes. Consider a second round of stretching if the athlete will play at the 100% effort. Typical cool-down consists of 5 to 10 minutes of easy exercise in the primary sport or a similar cross-training sport. Emphasis is placed on monitoring muscle tension and completing sufficient stretching to combat the muscle tightness from strenuous exercise.

Aerobic Training for Endurance Sports

This type of training is best-suited for weight loss and for preseason or in-season training for court sports and aerobic sports. It has no specific place in the training program of a sprinter or a gymnast except to optimize body weight. The objective is to increase the athlete's maximal oxygen consumption (VO_{2max}), which can be achieved by either continuous exercise or interval training. For healthy individuals, the training heart rate (THR) is 70 to 85% of the maximum heart rate (MHR). Weight loss can be maximized at 60 to 70% MHR, because body fat is the principal fuel and the lower intensity allows a longer exercise duration. Higher THRs are recommended for better trained individuals who are interested in peak performance; this topic is discussed later in this chapter.

Interval Training for Power and Strength Sports

Interval training, defined as repeated short periods of higher intensity exercise punctuated by periods of rest, can also be used in a specific manner primarily to increase VO_{2max} and potentially performance.[10] A certain level of previous endurance conditioning is best before undertaking interval training, in order to avoid injury. The athlete whose sport requires a sprint or explosive-type efforts should emphasize anaerobic interval training. Either interval training (e.g., running, for a basketball player) or sport-specific activity (on-court basketball drills) can be used with similar beneficial effects (Figures 7.2 and 7.3).

Flexibility

The purpose of stretching is to correct muscle fatigue, repair injury, and improve joint motion and movement technique. The amount needed for each individual varies, but it must be commensurate with the amount of muscle tightness produced from training. The best stretching programs include a brief bout of cardiovascular activity to improve muscle elasticity via improved blood circulation and slightly elevated body temperature. To stretch the muscles, use a variety of similar exercises for each muscle group. Apply slow, steady pressure for 30 to 60 seconds, or until the muscle relaxes. A specialized technique called proprioceptive neuromuscular facilitation (PNF) can be helpful for an unusually tight or painful muscle. Begin stretching and hold the position in the usual manner (30 to 60 seconds), then contract the muscle being stretched for 6 to 7 seconds. Following the contraction pause for 1 to 2 seconds and then repeat the stretch. Repeat this process five times. This stretch–contract–stretch technique allows for improved flexibility, especially for muscles that cross over two joints. If a muscle is injured and rapid recovery is desired, stretch every hour during the day until relief is achieved.

Strength Training

Strength training or resistance training (free weights, calisthenics, machines, plyometrics) can be helpful for all athletes (Level of Evidence B, systematic review).[11] The amount of weight training must be matched to each athlete's specific goals. Weight training can also help to build up strength[12–16] and correct muscular imbalances.[14,16] The intensity of weight lifting is also governed by the goal. If a muscle is injured, it must be strengthened without further damaging it. Muscle-tendon injuries repair slowly in adults. Athletes must avoid pain at the injured site during all activities, including weight lifting and stretching. The work effort must be the most difficult possible without causing pain and further injury. If the

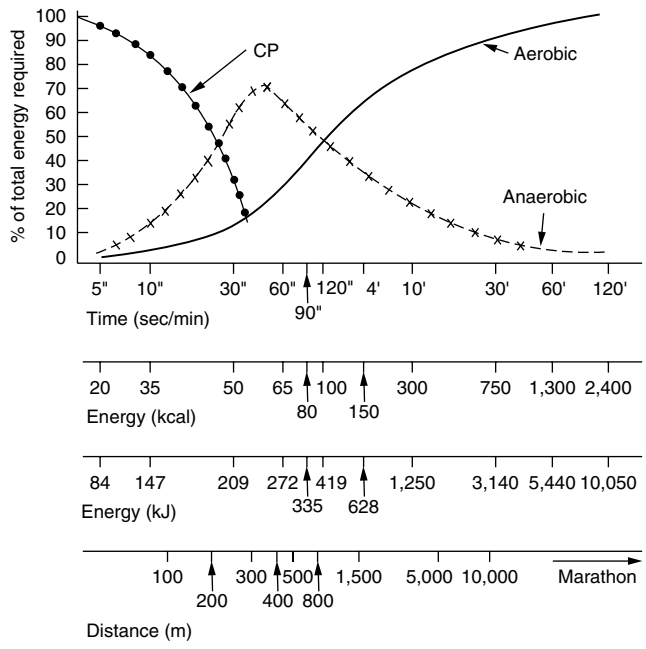

Figure 7.2 Energy systems for sports performance. (From Martin, D.E. and Coe, P.N., *Training Distance Runners,* Human Kinetics Publishers, Champaign, IL, 1991. With permission.)

site is painful, the athlete should perform 4 to 5 sets of 20 to 25 repetitions with very light weights. If this is painful, the athlete should change the arc of motion to avoid the painful range and still do high repetitions with low weights. An athlete who cannot perform this exercise should work against gravity and consider using resistive elastic exercise tubing. To maintain the current strength level, athletes should perform 4 sets of 10 repetitions 3 times a week. Building up strength with weightlifting in a power sport athlete is done differently. Such athletes should start with 4 sets of 10 repetitions at a work effort perceived as medium to hard, followed by increasing the weight by 5 to 10% every 1 to 2 week. Eventually, the athlete should perform 4 sets of 6 to 7 repetitions in the medium to hard range and progress when ready to 2 to 3 sets of 2 to 4 maximum repetitions. This workload will be perceived as hard to very hard and is necessary to improve the strength of a muscle.

Cross-Training

Cross-training is the substitution of one (or a series) of training exercises that provide similar physiologic results. Cross-training methods are especially useful for off-season training, injury prevention and recovery, and preventing burn-out in the primary sport. Essentially all athletes cross train; for example, baseball players lift weights, run, and bike to keep fit in the off season. Indoor equipment such as a bike, rower, cross-country skier, or the newer elliptical treadmills are very easy on the joints and are well tolerated in the early postoperative period for rehabilitating lower extremity injuries. Cross-training can be subcategorized into skill similar, strength, plyometric, aerobic, or anaerobic type training.

MEASURING FATIGUE AND FITNESS

Measuring the effects of fatigue and improvements in fitness is an essential component of coaching. Traditionally, the coach's stopwatch and VO_{2max} testing were the standards. Invasive testing with blood analysis for lactate can now be conducted field side utilizing portable lactate monitors costing less than $500. Extensive research reveals correlations among VO_{2max}, blood lactate, resting and exercise heart rate, body weight, and psychological state. Mathematical modeling for measuring fitness and fatigue and critical

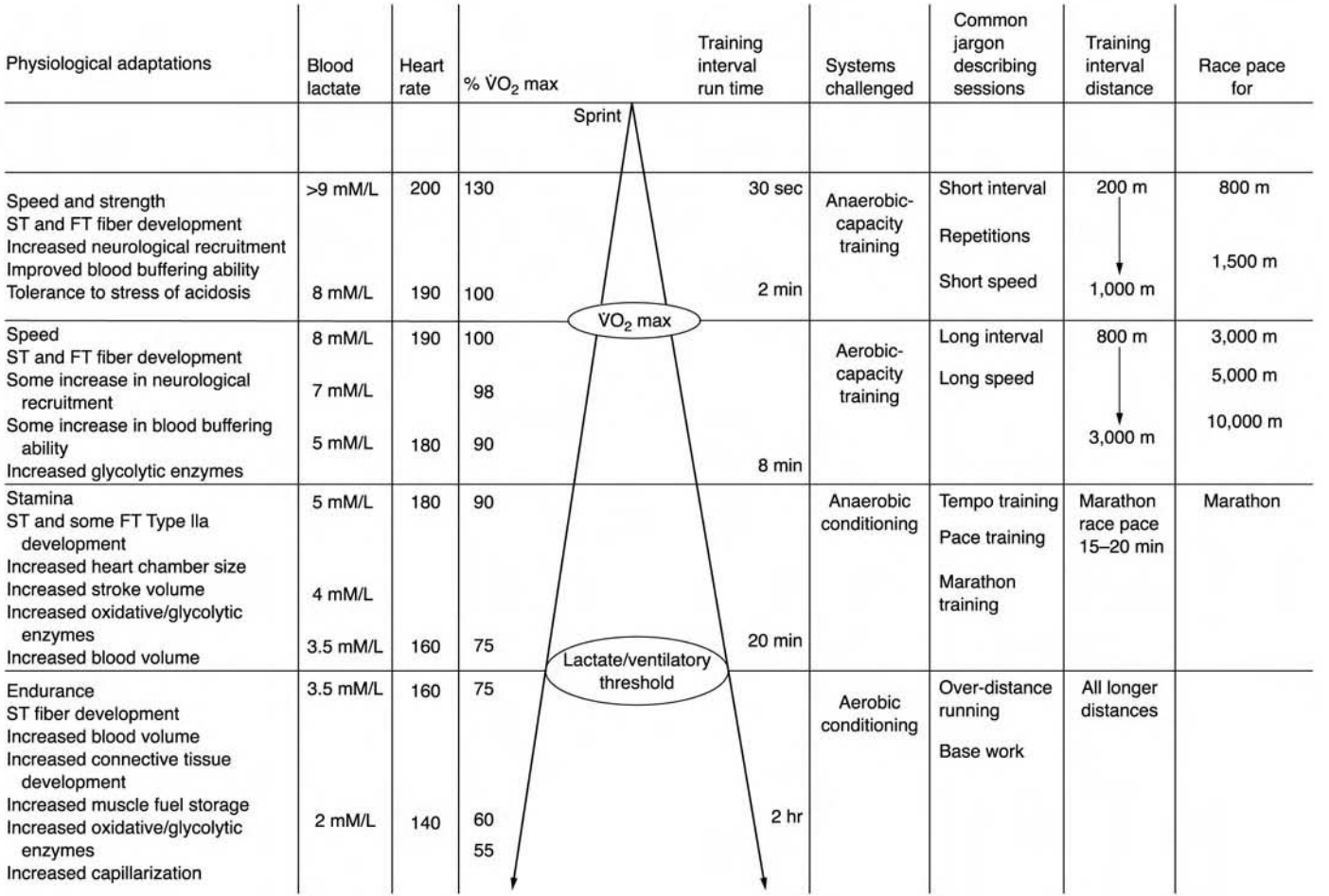

Figure 7.3 Primary training zones of performance during training. (From Martin, D.E. and Coe, P.N., *Training Distance Runners,* Human Kinetics Publishers, Champaign, IL, 1991. With permission.)

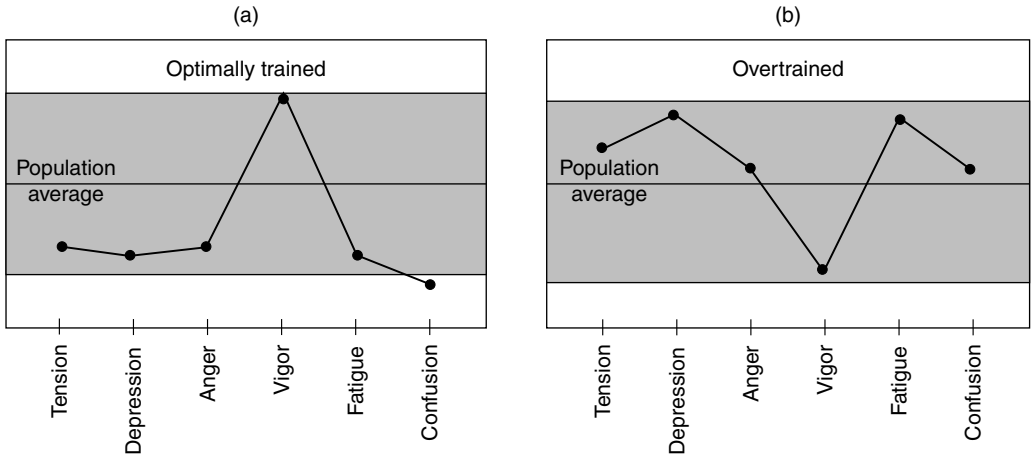

Figure 7.4 Profile of mood states. (From Martin, D.E. and Coe, P.N., *Training Distance Runners,* Human Kinetics Publishers, Champaign, IL, 1991. With permission.)

power function analysis for measuring aerobic and anaerobic components of exercise are two new scientifically based methods that should be utilized by coaches and athletes.

Applying High-Intensity Training Loads

Neuromuscular patterning is a key component of improving mechanical efficiency. Typically, athletes train using a combination of aerobic, anaerobic, cross-training, and sport-specific techniques. For example, a speed skater, cross-country skier, runner, biker, weightlifter, and swimmer may all engage in a combination of aerobic and anaerobic exercise, flexibility improvement, and weightlifting. The time spent training at high speeds is usually shortened by the fact that it produces great physiologic distress on the exercising athletes.[6–8] In other words, most world-class athletes need to be working out at approximately 95% of their peak power one to two times per week. This type of training requires that the athlete be well rested and well hydrated and have muscle glycogen stores that can provide caloric energy necessary to perform the exercise bout. When applying training loads, it is necessary to document the amount of fatigue that eventually sets in. Training too little or too much is ineffective. An athlete must train to strain the system but must allow the system to recover.[9] If athletes do not keep up vigilance and pay attention to the details of training,[17–21] they will suffer several consequences, such as overuse injuries or the over training syndrome. Research has shown that exercise stress to the body measured at the cellular level yields changes that can take 4 weeks to normalize. Many marathon runners will confirm that it is very difficult to run more than one or two successful marathons per year. Athletes can usually handle training loads of approximately 10% per week; however, if carried out over and over for several months, 10% increases can precipitate an overuse injury. Training stresses that are at a 50% level or greater can build up enough acute stress that an injury can prevail in only 1 week.

Psychology of Training Athletes

Athletes who train for sports competition are already approaching the theoretic limits of what is tolerable by the body.[22,23] Athletes like to know how far they can push themselves in training and competition.[24] Blood tests have proved too elusive and cumbersome for most athletes. Psychological interventions appear promising for athletes,[25–28] yet seem to be underutilized. Research on visual imagery,[29] calming,[30] motivational issues,[31,32] and mood awareness and control has been reported in the literature. The paper by Berglund and Safstrom[33] on using the Profile of Mood States for dosing proper training provides excellent evidence of this powerful, reliable, and simple intervention (Level of Evidence B, clinical cohort study) (Figure 7.4). Athletes and coaches need to incorporate psychological measures into their routine training programs.

Heart-Rate Monitoring

Heart-rate monitoring is very simple, highly reproducible, and sensitive to neuroendocrine and

extracellular fluid volume changes. Additionally, psychological anxiety can affect the neuroendocrine system and indirectly affect the heart-rate response to exercise; therefore, it is important to repeatedly measure the heart-rate response to an athlete's exercise sessions. This is very useful because it provides minute-to-minute feedback. One can typically expect a certain response; not seeing that expected heart-rate response can signal fluctuations in the athlete's fitness fatigue status. Athletes can record their heart rate in several ways. First, the athlete should record a morning resting heart rate, then stand up, wait 15 seconds, record the heart rate again, then wait another 2 minutes, and record the heart rate again. Also, the heart rate should be recorded during various exercise intensities, making note of the number of minutes for each exercise session. Abnormal elevations are indicators of dehydration and/or systemic fatigue build-up. Continued monitoring over a period of months, combined with body weight and criterion performances (time trials), will clarify the difference between fatigue and dehydration. This is essential in order to interpret heart-rate data, especially at or above critical power.

Body Weight

Body weight will yield information on water stores, sweat rate, and glycogen storage. When embarking on hard training, it is necessary that the carbohydrate stores of the body be adequate at every workout.[34] In order to learn the caloric intake necessary for an individual's particular training session, several months of repetitive weightings are necessary.[35–39] Athletes should weigh themselves four times each day for several months over the course of their training season. Weighing before and after exercise under a variety of conditions (heat, wind, sun) will yield information on sweat rate and how well the athlete compensates by drinking and eating during the day. Endurance athletes who train for 4 to 6 hours a day need to consume several thousand calories of liquid and/or solid carbohydrate during this time period. The only way to document proper intake and storage is by repetitively measuring the body weight as it fluctuates throughout the day, when water and muscle glycogen are in flux (Level of Evidence B, systematic review).[35]

Weight #1: Morning Weight

This weight should be recorded after the athlete has emptied the bladder and colon. This is necessary because a full bladder and fecal material can add 2 kg or more of weight.

Weight #2: Before Exercise

This weight should be recorded just prior to exercise and provides a baseline level.

Weight #3: After Exercise

This weight should be recorded immediately after exercise. Care should be taken to use the same scale and utilize similar technique (*e.g.,* minimal clothing). Subtracting weight #3 from #2 provides information on sweat rate.

Weight #4: Bedtime Weight

This weight is necessary to record how much replenishment took place between the last exercise session and bedtime, when the athlete stops eating and drinking. The bedtime-to-morning weight difference indicates weight loss during sleep. This is a compilation of respiratory losses from breathing and also calories consumed during sleep.

Analysis of Body Weights

Weighing the athlete four times per day for several months reveals the athlete's water utilization pattern, which allows proper interpretation of the heart rate data that are recorded during training. Knowing the amount of water lost and gained during exercise is important so athletes can properly prepare themselves for competition and calculate the necessary replenishment for their particular sport venue. Fluctuations in body weight reveal how well athletes replenish their carbohydrate and extracellular fluid stores. Athletes can achieve the best accuracy with a sample of four-times-per-day weighings carried out over 2 to 6 months as the training season progresses. Extracellular fluid volume will distort heart-rate data. Personal experience yields a pattern of 10 to 12 beats elevation of the heart rate at workloads above 75% when in a slightly dehydrated state. One consequence of not having adequate carbohydrate stores is that the exercise bout will be shortened, especially when the workload is above 75%. Proof that the athlete is eating sufficient quantities to match the calculated needs can be observed in weight fluctuations over several months. Once a proper pattern is established, less frequent weighings are required.

Mathematical Modeling

A clever approach to predicting training loads is the use of mathematical modeling.[40–44] The model (Figure 7.5) takes into account that athletes are highly individual in their responses to a given training load. For example, consider the current

$$\text{Training impulse (daily training load)} = \begin{pmatrix}\text{Duration of}\\ \text{exercise, } min\end{pmatrix} \times \begin{pmatrix}HR_{exer} - HR_{rest}\\ HR_{max} - HR_{rest}\end{pmatrix} \times (y)$$

$y = e^{bx}$ = weighting factor applied to calculation of training impulse to increase the magnitude of quantity of training nonlinearly at higher intensities

$e = 2.718$ $\quad b = 1.92$ men $\quad 1.67$ women

$$x = \frac{HR_{exer} - HR_{rest}}{HR_{max} - HR_{rest}}$$

Figure 7.5 Mathematical modeling. (From Scott, W., *Curr. Sci.,* 1(3): 184–190, 2002. With permission.)

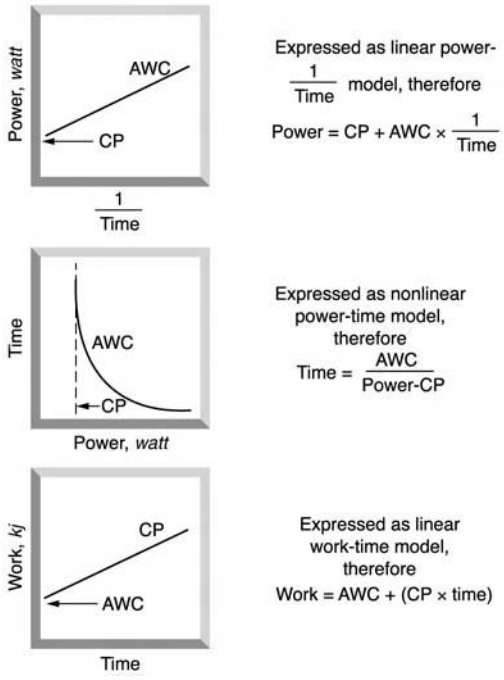

Figure 7.6 Critical power function. (From Scott, W., *Curr. Sci.,* 1(3): 184–190, 2002. With permission.)

world record in any given sport as a goal. By definition, this world record is the theoretical best a human can perform. Now consider an athlete's own personal best in the same event. Calculate the percentage difference between the two. How long will it take to make this much improvement? What is the physiologic possibility of the athlete achieving this goal? The ultimate outcome is going to be dependent on genetics, years of training, and psychological willpower.[45,46] Training impulses produce a small amount of fitness and a small amount of fatigue. The build-up of each can be measured indirectly by using mathematical models to increase the training until the athlete accumulates enough fatigue that his or her running economy suffers and the athlete eventually slows down over a period of time trials. After 3 to 4 weeks of intense training, a taper period begins while simultaneously the time trials repeat. The athlete should super-compensate and rebound back and show improvement in the time trials or actual competition (Figure 7.6).

Critical Power Function

The critical power function is a hyperbolic relationship between time and power output.[47–49] The power output vs. time can be plotted on a graph (Figure 7.7). Critical power is the highest intensity level that can be sustained for a long time. It correlates with lactate threshold, ventilatory threshold, and aerobic–anaerobic threshold.[50–57] Research shows that a 15-minute bike time trial, a 5-K run, and a 1-mile swim (world record, ~15 minutes) all are conducted at approximately 95% VO_{2max}. Accordingly, a 30-minute bike time trial and a 10-K run are carried out at 85% of VO_{2max}. In similar fashion, a 2.5-hour bike race and a 42-K marathon are both carried out at approximately 75% of VO_{2max}.[41,45] Coaches and athletes should incorporate this approach into their programs, as it allows them to precisely monitor fitness improvements week to week, without the use of invasive testing (VO_{2max}, lactate).

PERIODIZATION OF THE TRAINING SEASON

The training year can be subdivided into preseason, seasonal build-up, peaking and racing season, and postseason recovery (Figure 7.8). Each sport has a typical competitive season usually lasting about 6 months and frequently ending in a championship event. Two to three major competitions per season are selected. The 2 weeks immediately preceding each event are the

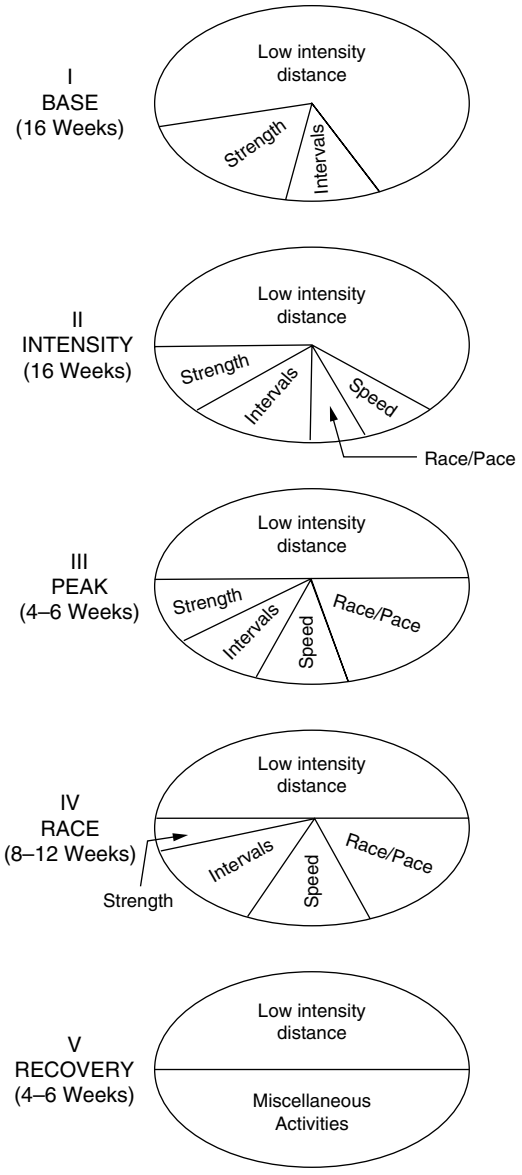

Figure 7.7 Training components. (From Sleamaker, R., *Serious Training for Serious Athletes,* Human Kinetics Publishers, Champaign, IL, 1989. With permission.)

designated tapering periods. The time prior to the tapering period is dedicated to progressive build-up (intensity and quantity) of workouts for several weeks. Over the course of the training season, the athlete will build up training for 2 to 4 weeks, followed by 1 to 2 weeks of reduced training, again depending on the time necessary for the athlete to adapt. This cycle allows the athlete's body to adapt to the training stress. Athletes may participate in minor competitions in this relatively fatigued state. Modern training programs include aerobic and anaerobic exercise, weight training, flexibility exercises, and sport-specific drills to improve technique. Additional sport-specific exercises can be added to the training, such as plyometric exercises. The entire regimen is followed throughout the year, but the percentages of maximal exertion for each component will vary throughout the year (Figure 7.9). The preseason training will focus on establishing a sound training base. The postseason period is also an essential part of the training, allowing for psychological and physiological recovery.

TAPERING

The tapering process can be highly variable from one athlete to the next and is often improperly performed. It is important to document the individual variation and determine the sensitivity of changes in your methods. Morton et al.[43] showed that the tapering period can be somewhere between 11 and 26 days. The tapering period needs to be highly specific.[58–66] When the tapering period is being monitored, it is essential to prove that weight did go up, illustrating supersaturation of water and carbohydrate stores. Also during the tapering period it should be noted whether a compensatory change in heart-rate response to exercise was observed. When an athlete is tapered, the heart rate at the critical power level is typically five beats lower than calculated, and the morning resting heart rate is at baseline level (Figure 7.6).

EXERCISE LOG

All athletes should be encouraged to maintain an exercise training log. As noted previously, athletes should record: (1) body weight, (2) morning supine and standing heart rates, (3) time spent training above the 95% level, and (4) heart rate at critical velocity during criterion performances, as well as Profile of Mood score and any unusual symptoms that occur during or after the training session. A training diary provides a psychological incentive to continue an exercise program, and facilitates adjustments to improve the overall quality of the program.

SLEEP

Sleep is very important for recovery.[67] Athletes who do not accumulate enough sleep hours will not properly recover from day-to-day exercise sessions. It is necessary to learn individual requirements. Athletes who train 4 to 6 hours a day frequently need 10 to 12 hours of sleep per

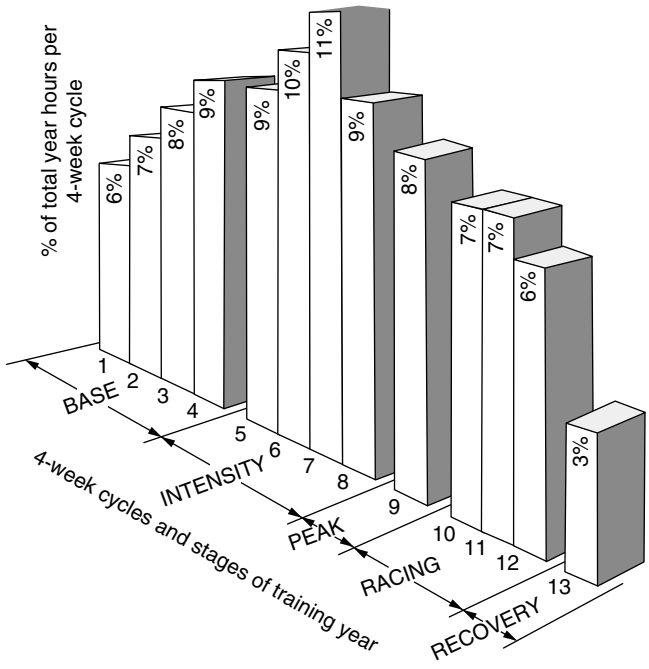

Figure 7.8 Periodization distribution between training cycles. (From Sleamaker, R., *Serious Training for Serious Athletes,* Human Kinetics Publishers, Champaign, IL, 1989. With permission.)

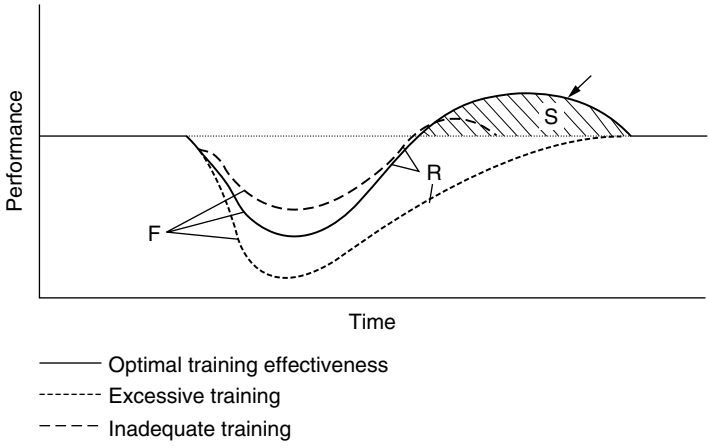

Figure 7.9 Model of supercompensation. (From Martin, D.E. and Coe, P.N., *Training Distance Runners,* Human Kinetics Publishers, Champaign, IL, 1991. With permission.)

night. Many athletes, due to busy lifestyles engaged in businesses and educational pursuits, will try to get by on 6 to 8 hours of sleeping when their bodies probably require much more. This can have a huge effect on the athlete's ability to recover from the rigors of training. A useful method for determining the required sleep pattern is to make sure that the athlete can sleep undisturbed over the weekend or on holidays. Trial and error will reveal how much sleep the athlete would naturally get if allowed to sleep undisturbed.

SPECIAL ADAPTATIONS

It is important for athletes to keep in mind that special equipment and the effects of altitude, circadian rhythms,[68–70] temperature, wind, humidity, arduous terrain, pollution, allergens,[71] and other sport-specific variables must be calculated into the overall training program. Failure to recognize the influence of such factors can surely alter an athlete's performance by 5 to 10%. It is important to understand that world-record differences between first and second place are frequently less than 0.5% at the Olympics.

SUMMARY

Training for competition is hard on the athlete. In order to improve performance, the athletes must stress themselves. Athletes must learn to tolerate fatigue and the ill feeling associated with stressful training. Supervising an athlete who is training is both an art and a science. The experienced coach can prescribe hard training in a manner that is psychologically pleasing and acceptable to the athlete. Given the rigors of proper training, it takes a tough constitution to endure the necessary workouts. In today's sports world, athletes must follow scientifically based, comprehensive training programs to remain competitive and injury free.

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8 PERFORMANCE VARIABLES IN SPORTS*

Sara M. Kass

Performance and achievement in sports are more complex than just hard work and dedication. Many variables impact on the level of skill achieved by an individual participant. Genetic, environmental, mental, and physical factors weave together into a complex matrix that defies a simplistic approach or a single overriding factor that can predict or result in maximal athletic performance. For the practicing physician, it is worthwhile to be knowledgeable of genetic and physiologic factors while addressing issues such as environment, nutrition, training, psychological factors, and injury prevention with the athlete.

HEREDITARY/GENETIC FACTORS

Genetic factors influence performance through several determinants: body composition, cardiopulmonary functions, muscle characteristics, biometabolism, and physiology. Considerable differences

* In the second edition, this chapter was authored by Jeffrey I. Tanji.

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exist in the individual level of endowment with regard to these factors.

Somatotype

It has long been recognized that success in a given sport is often associated with and epitomized by a particular body type. Somatotypes are often described according to the Heath–Carter method,[1] using the terms *ectomorph*, *endomorph*, and *mesomorph*. It is easy to conceptualize how particular body types may be more suited to particular sports, with most competitive basketball or volleyball players being characterized by higher ectomorphy,[2] while gymnasts and female pentathletes as a group tend toward a mesomorphic body type.[3,4] The tendency toward the ideal body type may be more significant as the level of competition is considered; Gualdi–Russo and Zaccagni found more homogeneity of the body type of male volleyball players at the maximum level of performance.[2] Numerous examples of very successful athletes, including National Basketball Association stars Shaquille O'Neal, a 338lb endomorphic center, and Speedy Claxton, a 166lb mesomorphic guard, demonstrate that a particular somatotype is not necessarily a requirement for success in a particular sport. It is clear that while anthropomorphic body types generally do correlate with success in certain sports, these correlations are not strong enough to predict performance on an individual basis.[3]

Muscle Fiber Type

Different muscle fiber types have been shown to correlate with different types of athletic performance. Type I fibers have superior oxidative capacity when compared to type II fibers, which have superior glycolytic capacity. Type I fibers are more abundant in athletes participating in endurance type activities, such as long distance running, swimming, and cycling. Type II fibers are associated with short-duration, high-intensity activities such as soccer and short and middle-distance running events.[5,6] Skeletal muscle properties may vary as a function of race. Samson and Yerles[7] reviewed performance in Olympic events and found that U.S.-trained black athletes dominated sprinting events, while U.S.-trained nonblack athletes excelled at long-distance events. Ama et al.[8] compared muscle fiber types between African-American and Caucasian men and found that type I fibers were less abundant in African-Americans than Caucasians. Difference in racial group athletic aspirations and success has long be attributed to sociocultural factors, but these findings suggest a possible genetic predisposition to success in various sports.[7] Further research is needed to examine the relationship between racial differences in skeletal muscle properties and the role they play in performance, as current studies are small and difficult to extrapolate to the population level.

Gene Mapping

Investigation into DNA-level differences and their effect on fitness and performance started in the 1990s. The most extensively studied gene associated with athletic performance has been the angiotensin-converting enzyme (ACE) gene. A greater frequency of the insertion (I) allele in the ACE locus has been reported in endurance athletes, specifically Australian rowers, British mountaineers, and runners.[9,10] Human gene mapping for physical performance is in its infancy but is an exciting new area of human performance research.

PHYSIOLOGIC FACTORS

Oxygen Consumption

Athletes who perform well in endurance events often possess a high VO_{2max}.[11] Physiologic adaptations that may occur as a result of training that challenges the oxidative system include increases in maximal cardiac output, blood volume, concentration of oxidative enzymes, capillary density, and oxidative capacity of muscles through increased mitochondrial volume and density.[11–14] Researchers agree on the existence of a physiological upper limit to the body's ability to consume oxygen.[14] It is debated whether this limit is set by pulmonary, cardiovascular, or skeletal muscle factors. Current opinion is that cardiac output is the principle limiting factor for VO_{2max} in endurance events such as running and cycling.[14,15] Exercise physiologists are engaged in ongoing research to further identify opportunities to improve VO_{2max} and possible limiting factors.

Lactate Threshold

Lactate threshold (LT) is another marker of endurance performance as it is the point at which blood lactate accumulates above resting values during increased exercise intensity. A runner with a high lactate threshold is able to run at a high percentage of VO_{2max} before the lactate production rate exceeds the lactate removal rate. Lactate production is related to multiple variables, including mitochondrial content in muscles and mitochondrial enzyme activity.[14] Endurance athletes have been found to have a higher LT, suggesting that

they can operate at a higher percent of VO_{2max} for a longer period of time before beginning lactate accumulation.[11, 14]

Running Economy

Running economy describes a relationship between VO_{2max} and velocity of running, or the oxygen required at a given absolute exercise intensity.[11,12] Running economy influences performance for distance runners by allowing the runner to run faster over a given distance or run longer at a constant speed because of a decrease in oxygen consumption for the work completed. Both endurance and resistance training have been demonstrated to improve running economy.[11,14] Specifically, resistance training is thought to improve running economy by improving efficiency of the muscles used, recruitment of muscle fibers, and muscle coordination which translates to decreased workload and decreased oxygen consumption.[11]

Anaerobic Power

Anaerobic power refers to the ability to convert energy to useful work in the absence of oxygen. This is accomplished through glycolysis. Training that improves glycolytic enzyme activity, force production, and storage of intracellular glycogen or causes shifts within major fiber types (more type II than type I) may increase anaerobic capacity. Improved anaerobic power has clearly been demonstrated to improve strength and when combined with anaerobic training may have a synergistic effect in improving endurance performance.[11] One potential benefit of improved anaerobic power in endurance performance may be in the realm of increased time to exhaustion, which appears to be related to improved efficiency of muscle work and recruitment of other muscle fibers.[11,14]

Jet Lag

It is not uncommon for elite athletes to compete in events many time zones from home. For this reason, much research has been done to evaluate the effect of such travel and interventions that may mitigate the effects of travel. The symptoms related to travel across numerous time zones include sleep disorders, difficulties with concentration, irritability, depression, fatigue, decreased appetite, disorientation, and gastrointestinal disturbances.[16] These effects seem to be at their worst within the first 24 hours but can persist for 6 to 7 days.[16-19] It seems prudent to advise the athlete about these findings if air travel is required for their competition. Numerous studies have looked at possible interventions that may decrease the effects of jet lag. These studies have evaluated the benefits of benzodiazepines, melatonin, and room light exposure.[20–23] All studies have been inconclusive in proving a benefit, and further research is indicated.

ENVIRONMENTAL FACTORS

Temperature and Humidity

The adverse effects of heat and humidity on performance, especially in endurance events, have been demonstrated in several studies. If ambient temperature exceeds body temperature, heat cannot be dissipated by radiation. If relative humidity is high, the potential to dissipate heat by evaporation of sweat is substantially reduced. Dehydration decreases performance in endurance events, and this effect is exacerbated by hyperthermia.[24] The practicing physician must emphasize the importance of proper hydration, acclimation, training, and clothing to minimize the negative impact of heat and humidity.[24–27] (Level of Evidence B, small randomized controlled trials). Dehydration can also occur in cool and cold weather. Factors that contribute to dehydration in these conditions include respiratory fluid losses, sweat losses that may be high due to the use of insulated clothing, and limited fluid intake.[26,27]

Equipment and Playing Surface

Manufacturers of athletic equipment are regularly promising improved performance; however, measurements of performance rarely address player–equipment interaction so the efficacy for enhancing performance on the individual level is difficult to predict.[28] Protective equipment has been shown to decrease injury with minimal impact on performance. Playing surfaces have been repeatedly studied in regard to correlation with injury. Shoe surface traction for the average player is the specific relevant variable that is most likely to correlate with injury in various forms of football, including soccer, rugby, and football.[29] Many studies have looked at natural turf vs. artificial turf and found that minor injuries are more common on artificial turf in football and on grass courts than hard courts for tennis.[29,30] While injury prevention is critical to athletic performance, none of these studies addresses differences in performance based on the playing surface alone.

NUTRITION/DIETARY FACTORS

Energy

Meeting energy needs is the first nutrition priority for athletes. Inadequate energy intake relative to energy expenditure compromises performance and the benefits of training. When energy intake is inadequate, muscle glycogen is depleted and eventually fat and lean muscle mass will be used as fuel for the body. Normally active people need to consume 37 to 41 kcal/kg body weight per day based on Recommended Dietary Allowances. Recommendations for athletes should take into account the intensity, duration, and frequency of the events performed, and that energy requirement should be added to the baseline requirement.[26] While these numeric guidelines are helpful, each athlete needs to consume enough energy to maintain appropriate weight and body composition while training for a sport.

The Dietary Guideline for Americans and Nutrition Recommendations for Canadians suggest that the energy in a healthy diet should come from 55 to 58% carbohydrates, 12 to 15% protein, and 25 to 30% fat (Level of Evidence C, consensus/expert opinion).[31,32] Data are not currently available that would recommend a diet substantially different from this for athletes.[26] Carbohydrates are important in maintaining blood glucose and replacing muscle glycogen. The amount required depends on the athlete's total daily energy expenditure, type of sport performed, sex of the athlete, and environmental conditions. Protein requirements are slightly increased for highly active people and more so for resistance exercise than endurance exercise.[26,33,34] Supplementation with branch-chain amino acids has been studied, but to date the results do not demonstrate a performance improvement. Their safety is not well established, and accordingly their use cannot be advocated.[26,35] Fat intake should not be restricted, no performance benefit is gained by consuming a diet with less than 20 to 25% of energy from fat.[26]

Timing of meals is another issue that should be addressed with athletes. Eating before exercise, rather than exercising in the fasting state, has been shown to improve performance.[26,36,37] The meal should leave the athlete neither hungry nor with undigested food in the stomach. Carbohydrates are easily broken down and quickly provide energy to the body. Ingestion of carbohydrates during exercise has been shown to improve performance in strength and endurance activities. The carbohydrate consumed should be primarily glucose, as fructose alone is not effective.[26,36,38] Post-exercise meals should include carbohydrates and protein to maximize glycogen synthesis and provide substrates for muscle protein repair, respectively.

Fluids

Dehydration refers to both hypohydration and exercise-induced dehydration. Hypohydration is intentional dehydration induced prior to exercise. It is commonly employed in sports that require certain weight standards for participation, such as wrestling, boxing, and judo. Studies demonstrating the adverse effects of dehydration on performance date back to the 1940s, yet athletes still often do not consume adequate volumes of fluid before, during, and after competition. The magnitude of performance deficit is related to the degree of exercise-induced dehydration, with even low levels of dehydration impairing performance. This effect is magnified in hot and humid conditions.[24] Hypohydration clearly impacts on aerobic endurance, but research into the effects of hypohydration on muscle strength and endurance has produced conflicting results.[24,39,40] The American College of Sports Medicine recommends consumption of 400 to 600 mL of fluid 2 to 3 hours before exercise and 150 to 350 mL at 15- to 20-minute intervals, beginning at the start of exercise. Beverages containing 4 to 8% carbohydrate are recommended for intense exercise events lasting longer than 1 hour (Level of Evidence C, expert opinion).[41] After exercise, athletes should consume fluid and sodium to reestablish fluid balance.

Vitamins and Minerals

Micronutrients play a valuable role in energy production, hemoglobin synthesis, bone health, immune function, repair of muscle following exercise, and protection of body tissues from oxidative damage. In general, no vitamin and mineral supplements should be required if an athlete is consuming adequate energy from a variety of foods to maintain their body weight. Supplementation should be based on specific micronutrient deficiencies.[26,45,46] Iron depletion is one of the most common nutrient deficiencies observed in athletes, especially female athletes. Iron deficiency anemia negatively affects exercise performance by decreasing VO_{2max} and aerobic performance. Recent studies have documented benefit in replenishing iron when ferritin levels are low even if hemoglobin levels remain in the low normal range (Level of Evidence B, small RCT).[26,42–44]

Ergogenic Aids

The marketing of ergogenic aids is an international, multimillion-dollar business that takes advantage of the desire of athletes to be the best and gain any competitive edge that may be available. In the United States, the manufacturers of supplements are able to make claims regarding the effects of a product on the structure or function of the body as long as they do not claim to "diagnose, mitigate, treat, cure, or prevent" a specific disease (Dietary Supplement Health and Education Act of 1994). The manufacture may make claims that may or may not be valid as long as the label indicates the active ingredient and all other ingredients. The practicing physician should counsel the athlete on the use of ergogenic aids, including aspects of product safety, efficacy, potency, and legality.[26]

PSYCHOLOGICAL FACTORS

The importance of sport psychology has become more evident over the last two decades. Many professional athletes work closely with their sport psychologist to optimize their performance. Techniques such as goal setting, imagery or mental rehearsal, relaxation training, stress management, self-efficacy or self-talk, and modeling interventions dominate the sport psychology literature. The use of imagery to enhance performance has been studied in numerous different types of athletic participation and at different levels of skill development. Imagery has repeatedly been shown to have a positive effect on performance.[47–55] Not only has the benefit of positive imagery been documented by numerous studies but the adverse effect of negative imagery has also been demonstrated.[55] Hypnosis appears to enhance the intensity and efficacy of imagery.[48] Relaxation exercises are also well studied in relation to athletic performance. In relation to free-throw shooting in basketball, some studies suggest that relaxation is more effective than imagery;[51,53] however, relaxation has been found to decrease strength performance.[56] "Psyching up" refers to self-directed cognitive strategies used prior to or during skill execution to enhance performance.

The most commonly employed techniques include focused attention, preparatory arousal, imagery, and self-efficacy statements, with results suggesting that preparatory arousal is the most effective technique for muscular force production.[56,57] Untrained athletes likely may have a greater benefit from psyching up, as they have a greater variation in performance than highly trained athletes.[56]

Self-confidence is also an important psychological factor in athletic performance. While no overall agreement has been reached with regard to what factors are most important for the occurrence of repeatable good performance in a sport, athletes often rank total self-confidence as one of the most important factors. Additionally, self-confidence has been found to correlate directly with performance in young athletes.[58,59]

An interesting area of research in the field of sport psychology is childhood traits that predict future athletic performance. Poulton and Milne[60] found that low levels of fear in childhood correlated with athletic achievement in adolescence and young adulthood. Clearly, numerous other factors are likely to have similar correlations and further research in needed.

TRAINING

Physiologic adaptations that occur with endurance training lead to increased delivery of oxygen to the exercising muscles (central adaptations) and increased utilization of the oxygen by the working muscle (peripheral adaptations). The central adaptations to training include decreased heart rate, increased blood and plasma volume, and increased cardiac output due to an increase in stroke volume. These changes may occur relatively rapidly (3 to 5 days) with training, but an improvement in VO_{2max} will require longer periods of training (12 to 38 days).[61] Peripheral adaptations take even longer to occur and include increased muscle capillary density, increased mitochondrial volume, and higher oxidative enzyme activity. In the untrained individual or recreational athlete, these changes are predictable and result in more efficient muscle contraction and increased physical work capacity, which in turn lead to enhanced performance. In the elite athlete, these adaptations have already occurred, and a further increase in performance is not related to increased volume of training so other methods of training are often employed.[11,61]

Resistance training, high-intensity interval training (HIT) and living and/or training at altitude are a few of the training strategies used by elite athletes to improve performance. Resistance training is designed to increase muscle strength, power, and muscular endurance. Resistance training leads to increased strength in trained and untrained athletes but typically does not improve VO_{2max}.[11] Circuit training (resistance training with limited rest between sets) in untrained individuals,

however, can lead to some improvement in VO_{2max} and lactate threshold (LT). These changes are felt to be due to the limited rest in circuit training and improved muscle efficiency. Resistance training does not seem to influence VO_{2max} or LT in either a positive or negative way in the trained athlete. Running economy does however appear to improve with resistance training, and this has been shown to improve running performance.[11,62] Increased strength as a result of resistance training in the elite athlete appears to improve mechanical efficiency, muscle coordination, and motor recruitment patterns, which results in decreased workload, improved economy, and increased time to exhaustion.[11]

High-intensity interval training is broadly defined as repeated bouts of short to moderate duration exercise completed at an intensity that is greater than the anaerobic threshold. Rest or low-intensity exercise occurs between bouts, allowing some, but not complete, recovery. In the untrained individual, the addition of HIT to endurance training improves performance to a greater extent than seen with endurance training alone. Adaptations in the untrained individual include upregulated contributions of both aerobic and anaerobic metabolism to the energy demand.[61,63] HIT can lead to significant improvements in endurance performance in the highly training athlete as well, but the mechanism for improved performance is unclear, and further research is required.[61,63]

Acclimatization to moderately high altitude while training at lower altitudes ("live high–train low") has also been evaluated for its effect on performance. A common theory as to how this combination works is through stimulation of erythropoiesis, leading to an increase in oxygen delivery to peripheral tissues.[64] Other researchers have suggested that the benefit is the result of changes at the muscle level and not erythropoiesis.[65] In collegiate runners, this method of training resulted in a 1.4% improvement in 5000-m performance at sea level, and elite athletes showed a 1.1% improvement in their 3000-m performance.[64] Studies looking at similar effect on elite cyclists have found similar levels of improvement.[65] While these improvements may not seem like a large effect, it is important to remember that races at these levels are won and lost by small fractions of a percent. Other studies have looked at living and training at altitude or living at sea level and training at altitude and have not demonstrated improvements in performance at sea level.[65,66]

BIOMECHANICS

Over the past three decades the role of biomechanics in sports science and sports medicine has grown exponentially. Biomechanics plays an active role in injury prevention, rehabilitation, and performance enhancement. Biomechanical analysis techniques provide a way to estimate forces and deformations experienced by various tissues during human movement.[67] These techniques can assist in identifying both anatomical as well as mechanical factors that put an athlete at risk for injury. Biomechanical analysis has been utilized to identify athletes at risk for various injuries, assist in development of protective gear that will decrease injury while not hindering performance, and identify movements and motions that should be avoided to decrease the risk of injury.[67–74]

Numerous biomechanical tools exist to directly or indirectly measure a range of mechanical variables that represent performance. A very common technique for assessing performance is motion analysis with two- or three-dimensional cinematography or videography. Other tools exist as well, but most are quite expensive, labor intensive, and typically only available to athletes performing at the highest levels of competition.[72–74] It is difficult to assess the effectiveness of biomechanical analysis on athletic performance due to the many variables between the biomechanical evaluation and successful execution of a recommended change in motor function.[72] Further research in the field of biomechanics and sports science will likely address this issue in the future.

INJURY RISK FACTORS

An athlete's performance is influenced by both the real and perceived risk of injury. Predisposing factors include both intrinsic or subject-related factors and extrinsic or environmental factors. Age, gender, body build, previous injury, physical fitness, and life stress are examples of subject-related risk factors. Environmental factors include sports-related factors, playing surface, equipment, and weather. Sports injury results from a complex interaction of these variables. The preparticipation sports examination gives the astute clinician the opportunity to identify possible risk factors and educate the participant in measures that can minimize the risk of injury.[75]

Exposure time, previous injury, and life stress are three factors that were found by Van Mechelen et al.[76] to be significantly correlated to injury. It is easy to see how increased exposure may lead to a greater risk of injury. It is not recommended that we encourage athletes to decrease exposure

to reduce risk, but emphasizing the importance of recognizing early signs of overuse injuries is reasonable.

Previous injury as a risk factor should be addressed by emphasizing the importance of full rehabilitation and implementation of measures to reduce re-injury. Tenacity is an attribute that is commonly associated with the successful athlete; however, this same tenacity can be detrimental when the athlete is insistent on returning to participation before full rehabilitation. Ankle sprains in basketball are one area that has been studied extensively. Recommendations to reduce the risk of re-injury include completion of supervised rehabilitation followed by use of an appropriate ankle-support orthosis for 6 to 12 months (Level of Evidence C, expert opinion).[77]

Psychological and psychosocial factors are important both in injury prevention and recovery. Sports psychologists can play an important role in sports injury rehabilitation by assisting the athlete in understanding the mind–body interactions and how they function regarding stress.[78] Life stress is an independent risk factor for injury and is influenced by the athlete's support system and coping skills.[76,79] The athlete who is experiencing stressful life events may suffer from decreased attention, decreased concentration, sleep problems, and fatigue and may be more likely to engage in risk-taking behavior.[76] In terms of injury prevention, it is advisable that the coach, trainer, and physician pay attention to athletes who have been exposed to high levels of stress and assist them in identifying healthy coping mechanisms and social support.[76,79]

GENDER

Female participation in sports has steadily increased throughout the 20th century. In the 1900 Olympic Summer Games, a few women were allowed to play golf and lawn tennis. World War II furthered the movement of women into traditionally male roles, and in 1972 Title IX was enacted, creating equal opportunity situations for women in sports. Today, we have numerous professional female athletic teams and individuals who enjoy a large and loyal fan base.

Rate of improvement based on gender difference has been evaluated in light of this increased popularity of sports participation by women. The rate of improvement for women in running, swimming, and skating events has been extraordinary, especially in longer distance events. Male athletes are more physiologically suited to anaerobic strength events than women, but it has been proposed that women could improve in endurance events such that they could be on par with male performances or possibly surpass them.[80,81] Sparling et al.[82] evaluated this by reviewing gender differences in world records for running and found that the gender difference for running the 1500-m and marathon distances has remained stable since the 1980s, indicating in his study that the gender difference has reached a plateau. Despite this finding, men and women continue to improve their running performance, and it is certainly possible that this gap may narrow again in the future.

Hormonal changes, either natural or due to contraceptive medications, have been studied as a variable in athletic performance for women. Cyclical endogenous hormonal variations throughout the menstrual cycle influence metabolic, thermoregulatory, cardiovascular, and respiratory parameters. While the effects on each of these systems are clearly established, their impact on athletic performance is less clear. The findings for exogenous hormones in the form of contraceptive pills has rendered similar results.[83–85] The use of oral contraceptive pills to control menstrual cycles, delay periods during sporting events, reduce dysmenorrhea, lessen premenstrual tension syndromes, reduce iron losses by controlling menses, and reduce the risk of pregnancy may exert a positive effect on the overall sports performance.[83–85] The impact of changes exerted by endogenous and exogenous hormones are not likely to be sufficient to impact the ability of most recreational athletes to participate in their sport. The elite athlete, however, may note a more significant impact, as even small variations in performance are noticeable. For this reason, it is important that further research in this field continue.

Female athletes are under intense pressure to have a low percentage of body fat both for body image and to enhance performance. This is even more notable in events such as gymnastics and ballet. Elite athletes tend to be goal oriented and perfectionists to begin with, and these tendencies put them at risk to succumb to disordered eating. This may subsequently lead to amenorrhea and bone loss or osteoporosis and is known as the female athlete triad.[85,86] It is important for the practicing physician to be aware of this risk and the early signs of disordered eating and to emphasize the importance of healthy body composition for athletic performance.

AGE

Cardiovascular endurance, muscle strength, balance, and flexibility are all influenced by age. Master athletes have been used to evaluate the ideal rate of physiologic decline with aging, based on the premise that older athletes may be able to reduce the rates of decline in functional loss. This premise is questionable. Wiswell et al.[87] found that VO_{2max}, strength, and performance for men and women declined significantly with age, and that these differences are not considerably different than rates previously reported for older sedentary adults. Importantly, this study did not take into account current level of athletic activity. In studies looking at prior athletic performance and development of postural imbalance, it has been shown that history of athletic activity did not protect an individual with regard to development of balance difficulties. Those individuals who are currently active, regardless of prior athletic involvement, perform better on balance testing.[88]

The rate of decline in athletic performance as a function of age has also been evaluated. Swimming performance has been demonstrated to decline linearly from peak levels at age 35 to 40 until age 70, at which time this decline becomes exponential. The age of peak level and age at exponential decline are slightly earlier for running, 25 to 30 years and 60 years, respectively.[89–91] These age difference are felt to be due to a slower decline in upper body strength when compared to lower body strength and fewer orthopedic injuries in swimming.[89,90] When comparing strength vs. endurance performance, it has been shown that strength declines at a faster rate than endurance, but the rate of decline of each can be slowed by continued training in both resistance and endurance exercises.[92,93]

It is also important to be aware of athletic performance in the very young, as well. With the fame and success of athletes such as Tiger Woods and Michelle Wie, both of whom started to play golf at an early age, many parents are enrolling their preschool-aged children in organized sports programs and athletic camps.

It is important to keep in mind the child's level of physical, neuromotor, cognitive, perceptual–motor, and psychological maturation when encouraging athletic participation. Teaching or expecting a child to learn a skill when they are not developmentally ready is more likely to lead to frustration than long-term success in a sport.[94] The preschool child should be encouraged to participate in a variety of different activities that allow the child to practice and refine skills and most importantly to have fun.[94,95] Participation in organized sports, when these goals are kept in mind, can help the child to develop confidence, overcome fear of being hit by a ball, and develop an enjoyment for a variety of athletic events.

SUMMARY

Understanding the various contributions of genetics, physiology, biomechanics, training, nutrition, stress, gender, age, and the environment on athletic performance will allow the physician to astutely assist the athlete in achieving optimal performance while avoiding injury. The multitude of variables that contribute to athletic performance is what makes participation in competition, either against others or against one's own best effort, exciting and unpredictable.

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9 PROMOTING PHYSICAL ACTIVITY, EXERCISE, AND SPORT BEHAVIOR: A BIOPSYCHOSOCIAL APPROACH*

Nicole L. Frazer

We have obtained this life as a human being. Whether we make it worthwhile or not depends on our mental attitude.

—The Dali Lama

INTRODUCTION

Obesity and overweight are major health problems in the United States.[1,2] The prevalence of overweight and obesity has exceeded 60%. Obesity is strongly associated with increased risk for type 2 diabetes, heart disease, osteoarthritis, stroke, cancer, and depression, resulting in increased suffering, disability, and medical cost.[1] In 1995, the combined direct and indirect medical costs for untreated obesity were estimated to be over $99.2 billion, and they rose to over $117 billion by 2001.[3] The rationale for treating obesity comes from evidence that even modest weight loss can have a favorable impact on the above conditions.[4,5] For example, in a recent study, as little as a 7% weight loss combined with mild to moderate physical activity reduced the relative risk of patients with glucose intolerance from progressing to diabetes by 58%, compared to medication alone (31%).[6]

One factor believed to play a role in our nation's rising weight concerns is the concomitant low levels of physical activity among almost all population groups in the United States.[7] Cross-sectional studies have supported an inverse relationship between activity level and weight, with more active people less likely to be obese (Evidence Level B, cross-sectional study).[8] In fact, national health surveys have revealed that children and adolescents are spending large amounts of time engaging in sedentary behaviors that are associated with increased risk of obesity.[9] In addition, only

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64% of adolescents report 20 minutes of vigorous activity at least 3 times per week.[10] Finally, minority youth are reportedly less active than Caucasian youth.[11]

Just as troubling are the prevalence rates for adults. Only 25% of the U.S. population over the age of 18 report engaging in light to moderate physical activity at least 5 days per week, 45% report insufficient activity, about 25% of adults report no leisure-time physical activity, and only 16 to 17% report engaging in vigorous physical activity.[7,12,13] With regard to ethnic differences, young Caucasian adult males with higher incomes report the highest amount of leisure-time physical activity.[8] The most sedentary population group is adults 50 years of age and older. Furthermore, one in two women and one in three men 75 years of age and older report engaging in no regular physical activity.[10] Finally, even our nation's military personnel appear to be struggling to be physically active. In a random sampling of military installations, Harrison et al.[14] found that only 63% of respondents reported exercising 3 or more times per week, 37% reported exercising less than 3 times per week, and 15% reported not exercising at all.

Given these low rates of physical activity and the cumulative data regarding the physical and psychological health benefits of physical activity, exercise, and sport behavior, public health agencies and national organizations have made recommendations regarding physical activity and exercise. New recommendations for physical activity and health recommend that every adult in the United States engage in 30 minutes or more of moderate-intensity physical activity daily, such as heavy cleaning in the home (e.g., lawn mowing with a power mower, painting, or home maintenance) or walking briskly (3 to 4 mph, or 4.8 to 6.4 km/hr) (Figure 9.1).[15] Those individuals able and willing to further increase the intensity of their activity levels should.[16] With respect to youth, the *Healthy People 2010*[10] recommends at least 20 minutes of vigorous activity three times per week; however, due to rising obesity concerns among youth, others recommend at least 60 minutes of physical activity per day for children and adolescents.[17] Finally, for older adults, the ACSM[18] highlighted the importance between maintaining appropriate activity and physical, psychological, and cognitive abilities.

Primary care physicians are in an ideal position to significantly impact these public health issues, as they are typically an access point for health care.[19] Not only do adults average about 3 medical visits per year,[20] but Nawaz et al.[21] reported that approximately 25% of the people that enter a primary care clinic for any type of medical treatment are moderately overweight or obese (body mass index ≥ 27.3). This means that the typical primary care physician who treats 25 people a day could intervene on behalf of 30 individuals in a typical week who are at an increased health risk because of excess weight. A recent study found that only 42% of physicians discussed weight loss with obese patients (body mass index ≥30) and 35% with overweight patients (body mass index 26 to <30).[22] Similarly, 31% indicated that they referred obese patients to a nutrition counselor and 27% reported they discussed exercise.[22] Physicians have cited lack of resources, time, and knowledge of effective interventions as among the barriers to effectively addressing these important health issues.[22-25] Therefore, while optimally poised, primary care providers would benefit from additional tools, information, and/or training to effectively target such important public health issues.

This chapter focuses on recent studies examining the importance of physical activity, exercise, and sport behavior for optimal health. Strategies for primary care physicians to use for case conceptualization and treatment will also be presented. Additionally, common issues pertinent to competitive sports will be highlighted.

HEALTH BENEFITS

The effects of physical activity, exercise, and sport on physical and psychological health were extensively reviewed in the 1996 Surgeon General's report on physical activity and health.[16] Highlighted is the consistent evidence supporting a dose–response relationship between moderate to high physical fitness and lower mortality. That is, sedentary individuals have a 1.2- to 2-fold increased risk of dying compared to more active individuals. Among other health benefits, most studies have found an inverse dose–response relationship between level of physical activity and risk of coronary heart disease, risk for hypertension, and colon cancer risk. Evidence has also suggested that physical activity may play a protective role on the development of non-insulin-dependent diabetes mellitus. Regular, moderate exercise has also been shown to reduce symptoms and improve function for individuals with rheumatoid arthritis or osteoarthritis. Additionally, exercise has been associated with a reduced risk of falls and fractures in the elderly.

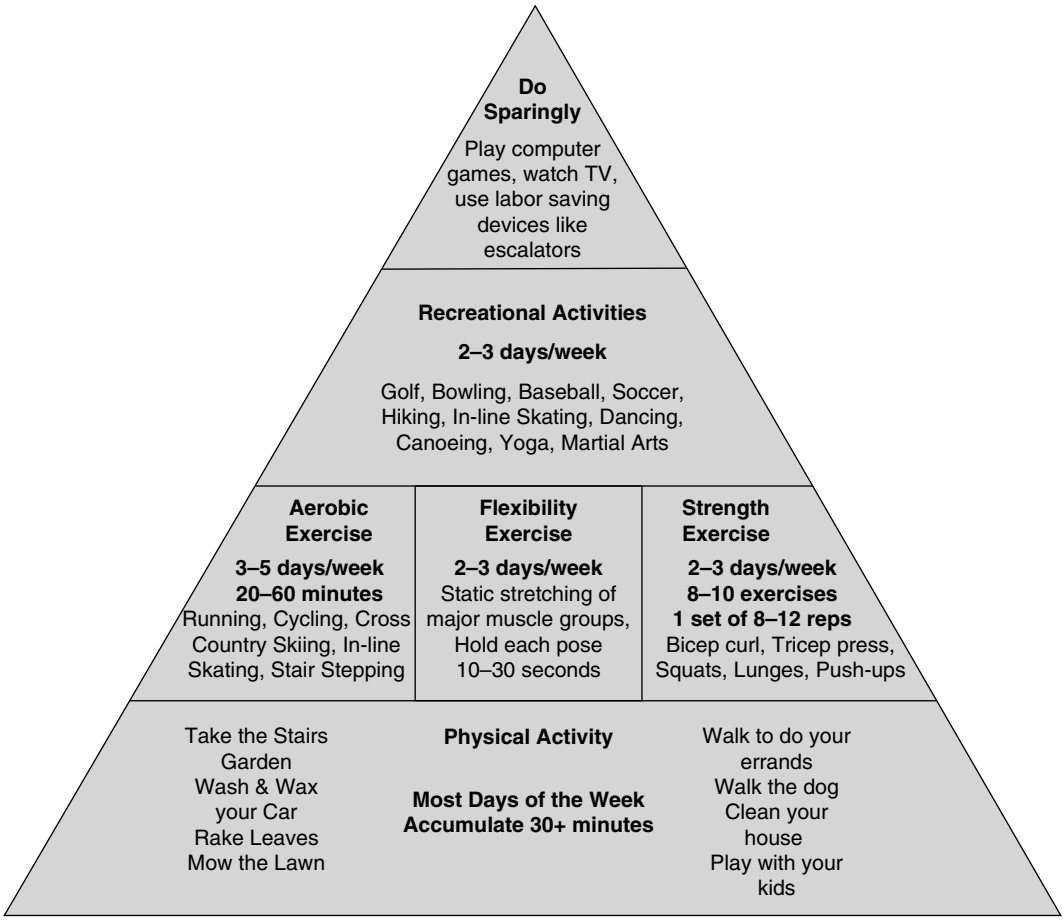

Figure 9.1 Physical activity pyramid. (From Georgia State University website http://www.gsu.edu/~wwwfit/physicalactivity.html. Copyright 1999 Board of Regents of the University System of Georgia. With permission.)

Exercise has been shown to be an effective intervention for depressed and anxious moods.[16] Worth noting is a randomized controlled trial involving older adult men and women who met *Diagnostic and Statistical Manual of Mental Disorders*, 4th ed., diagnostic criteria for major depressive disorder. They were assigned to 16 weeks of either exercise (30 minutes of walking or jogging three times a week) or antidepressant medication (sertraline), or a combination of exercise and medication. Across all three groups, participants had clinically significant improvements in their depressions scores at the 16-week follow-up. At the 10-month follow-up, those in the exercise-only group and the exercise-plus-medication group had a lower rate of relapse and a higher likelihood of being partially or fully recovered than those in the medication-alone group (Level of Evidence A, randomized controlled trial).[26] An association was also found between exercising on one's own during follow-up and reduced risk of relapse. Furthermore, studies have supported a positive impact on affect following exercise (Level of Evidence B, cohort studies),[27,28] and even improvements on some aspects of cognitive function (e.g. scheduling, planning, working memory) (Level of Evidence A, randomized controlled trial).[29] For adolescents, an inverse relationship between physical activity and depression has been supported (Level of Evidence B, nonquantitative review).[30] The aforementioned psychological health benefits are not exhaustive, but are among those with the strongest and most consistent findings.[16]

UNDERSTANDING AND CHANGING EXERCISE BEHAVIOR

Over the past decade, researchers have applied several different theoretical explanations to the study of physical activity, exercise, and sport behavior. These have included learning theory, social-cognitive theory, the trans-theoretical or stages of readiness to change model, and relapse-prevention models, to name a few.[31–34] Unfortunately, any one theory has been insufficient for successful understanding and promotion of physical activity and exercise behavior for health.[7,31] Suggested future clinical and research endeavors must include environmental (e.g., organizational and community) variables, in addition to intrapersonal and interpersonal factors.[11,16,35]

A BIOPSYCHOSOCIAL APPROACH

Through use of the biopsychosocial model, physicians and their multidisciplinary teams can conceptualize physical activity, exercise, and sport behavior across interpersonal, intrapersonal, and environmental dimensions.[36–38] The fundamental assumption of the biopsychosocial model is that health and illness are caused by multiple factors and produce multiple effects. These include physical, emotional, cognitive, behavioral, and environmental factors, which are detailed below.

Physical Factors

Identifying the patient's current health status is a critical part of any behavioral health assessment. Do they have any medical conditions that might limit their ability to exercise or participate in certain sports (e.g., an obese women with arthritic knees may indicate that she has trouble exercising due to the pain in her knees)? What is the patient's general level of risk for a cardiovascular event associated with physical activity or exercise? Can any other physical reasons be identified as to why the patient should not follow an exercise program? Is the patient over 35 years of age and unaccustomed to vigorous exercise? Does the patient have a permanent condition or injury that would preclude engaging in certain activities (e.g., cannot lift more than 20 pounds due to a degenerative joint disease)? Is evidence of deconditioning present? What is the patient's energy level? How is the patient's sleep/wake cycle? Is the patient overweight or obese? Understanding the patient's current health complaints will assist the physician in targeting the appropriate levels and types of physical activity and exercise goals.

Emotional Factors

Emotional factors can present on a continuum from normal mood fluctuation to clinically significant levels of distress. The physician should assess the patient's overall mood. Is the patient's current mood state a change from baseline and is it an acute or chronic change? Is the patient suffering from clinically elevated levels of depression or anxiety? It is important to identify whether the mood symptoms have preceded any decline in physical activity, exercise, and or sports participation. Intervention efforts would then be more appropriately aimed initially at understanding and improving the patient's mood symptoms; otherwise, attempts to focus solely on an exercise regimen might fail. Transitory or acute subclinical changes in mood status should be normalized and relapse prevention strategies discussed. For example, if the patient has recently stopped exercising or is exercising less due to increased feeling of stress, interventions should target ways to increase the likelihood that the patient will be successful at a return to the previous activity level. Have the patient set a goal to resume activity that day or the following day at the latest. Set an initial, small, realistic goal depending on the patient's health status and previous exercise level (e.g., walk 20 minutes). Suggest that the patient set out exercise clothes the night before and perhaps enlist the aid of a friend.

It is also important to assess the patient's perceived level of stress.[39] In one study during weeks of high perceived stress, women exercised less, reported less satisfaction with their exercise, and reported less confidence that they would achieve future exercise goals. Finally, additional concerns regarding fear of injury and engaging in exercise in public should be queried.

Cognitive Factors

Understanding an individual's mindset will have an influence on the success of intervention efforts.[7] For adults, having a higher self-efficacy for physical activity, believing in the value of physical activity, and perceiving fewer barriers are important predictors of physical activity.[40] In a national telephone survey of women over age 40 from a variety of ethnic groups, inactivity was found to be associated with lower levels of education, perceived poor health, and perceived lack of energy to exercise.[41] For children, a consistent positive relationship between physical activity and intent to be active and a preference for activity has been observed, and a consistent positive relationship for perceived activity competence and intent

to be active for adolescents has been observed.[30,42] Therefore, assessing patients' beliefs regarding the merit of physical activity, their ability to perform physical activity and/or exercise, their preference for activity, and their motivation for physical activity (exercise or sports participation) is essential.

Behavioral Factors

It is important to assess the patient's history of physical activity level, exercise, and sports participation. A positive relationship between previous physical activity and current physical activity, access to programs and/or facilities, and time spent outdoors has been observed for children.[30] Additionally, a relationship between after-school time for sports and physical activity has also been observed.[42] For adolescents, increased physical activity is associated with the availability of community sports, and decreased physical activity is associated with sedentary activity after school and on weekends.[30] Therefore, a comprehensive assessment of a patient's current time demands, work and/or school schedule, and previous levels of physical activity, exercise, and sports participation should be performed. Additional behavioral factors to assess include unhealthy behavioral habits, such as tobacco use, caffeine dependence or abuse, alcohol dependence or abuse, and abnormal eating patterns.

Environmental Factors

A person's social support network can have an important impact on the patient's physical activity level. Patients may have a lack of support from family or friends, or perhaps their family does not encourage physical activity, exercise, or sports participation. A strong and consistent relationship between children's physical activity and family support exists across age and gender subgroups.[42] A lack of resources or access to exercise facilities and financial limitations are other factors. Also, unsuitable weather conditions may play a role in inactivity. For example, the patient may typically walk outdoors but has stopped during the winter months due to the cold and snow. Environmental factors, such as exercising at home vs. in a class, are also important to consider for older adults.[43] Finally, women have cited a lack of others exercising in the neighborhood and a lack of hills in the neighborhood,[41] as well as concerns with neighborhood safety[44] as among barriers to exercise. Comprehensive assessment and understanding of mitigating, exacerbating, and maintenance factors within each domain will aid in case conceptualization and provide for a more appropriate treatment plan (Figure 9.2).

PROMOTING PHYSICAL ACTIVITY, EXERCISE, AND SPORT BEHAVIOR

Attention to intrapersonal, interpersonal, and environmental variables through use of the biopsychosocial model allows physicians to increase the fruits of their labors. For example, use of this model in the treatment of injured athletes has shown increased adherence to treatment and improved rehabilitation.[45] Furthermore, provider-delivered interventions that have targeted at least some aspects of the biopsychosocial model (e.g., psychological) have already been successful at changing physical activity and exercise behavior, such as Project PACE (Physician-Based Assessment and Counseling for Exercise) (Level of Evidence A, randomized controlled trial).[46] Project PACE, which is based on the trans-theoretical model (also known as the stages of readiness to change model)[47] and social cognitive theory, encourages physicians to tailor physical activity and exercise programs to the patient's current stage of readiness for change.[48] The stages of readiness to change model has been commonly used in the study of physical activity and exercise promotion. The model suggests that individuals move through a series of stages of readiness for change which include precontemplation, contemplation, preparation, action, and maintenance. For example, if a patient is in the precontemplative stage of behavior change, which means that they have no intention of starting an exercise program in the next 6 months, the physician would not want to devote clinic time to designing a program for this person; rather, the physician would want to begin talking with the patient about the benefits of exercise. In the contemplation stage, the patient is not currently engaging in physical activity but does intend to start. For patients in this stage of readiness for change, a discussion of barriers to beginning an exercise program and assistance with goal setting would be appropriate. Providers can emphasize benefits and strategies to increase the success of patients' initial efforts (e.g., assist patients with setting realistic, attainable goals). A patient in the preparation stage does participate in some physical activity or exercise but not regularly as defined by the ACSM guidelines; therefore, reinforcing a patient's current level of activity is important and finding ways to increase the patient's level of activity should be the focus of interventions. Suggesting ways to

Physical: The patient is a 35-year-old single Caucasian female who has been experiencing abdominal pain for 3 years. After multiple tests, she was diagnosed with endometriosis and is currently prescribed Motrin for this pain condition. Additionally, she has been diagnosed with IBS since the mid 1980s and is currently prescribed Prilosec. She reportedly has gained 35 lbs since the onset of the pain condition, appears deconditioned, but has no other significant medical conditions. She has no cardiac contraindications for starting an exercise regimen. The patient has reported significant disruptions in sleep due to pain and reports significant daytime fatigue.

Emotional: The patient reported that her mood varies in response to the pain, but is clearly tied to her pain condition. She stated that when her pain is worse, that she feels no "motivation" to engage in any activities. She reported experiencing several physiological symptoms consistent with IBS, but ascribes these symptoms to medication side effects rather than anxiety. The patient's mood symptoms do not meet criteria for a major mood disorder.

Cognitive: The patient tends to engage in more alarming vs. reassuring thinking with regard to her pain condition. She stated that one of her stressors is the thought that she has to live with her pain the rest of her life. However, she does engage in some reassuring thinking in that she stated she "knows she can deal with the pain." She reported that she has been focused on the pain and reducing the pain. Understanding how her cognitions impact her behavioral response to pain, and refocusing her attention elsewhere will likely assist her in better managing the pain. The patient reports she does not feel she can increase her activity level because when she does she "hurts."

Behavioral: The patient has evidenced a significant decrease in her activity level in response to her pain. She used to go bowling, go shopping or to other places with her daughter, and exercise consistently 3-4 times per week with either aerobics and/or strength training. She was also a member of a bowling team. She reported she used to enjoy exercising. She reported that when she is in pain, she has an emotional response such that she has no desire to be active or social. She also does not engage in any pleasurable activities or hobbies. The patient denied current or past use of tobacco, alcohol, or caffeinated substances.

Environmental: The patient's social support system is limited. She reported that she has no family or friends in the area with whom she engages in social activities. She used to be on a bowling team which allowed her to have some social contacts outside of work, but has discontinued her participation on the team in response to her pain. She is also not currently dating. Her daughter, who is 10 years old, is her only source of support. The patient stated that she often "feels bad" because her daughter often asks her "Mom are you ever going to get better?" The patient reported that she and her daughter used to be more active together before she began experiencing pain, but that has since decreased.

Treatment Recommendations:

The following list of treatment goals was established in collaboration with the patient:

Goal #1: The patient indicated that she would like to increase her activity level to improve her quality of life.

Intervention Strategy: Increase time spent walking.

Tactic: After establishing a baseline of current time spent walking, patient and provider will monitor graduated increases in activity level. Educate patient on the concept of hurt vs. harm and the process of reconditioning so that the patient understands that increases in pain with activity do not necessarily indicate bodily damage.

Outcome Measure: Exercise record.

Goal #2: Reduce overall physical tension levels to assist her in coping more effectively with her pain condition.

Intervention Strategy A: Cognitive Restructuring

Tactic A: Teach and work with patient on the model of thinking to decrease alarming thoughts which may be exacerbating current physical conditions.

Outcome Measure A: Patient's ability to recognize alarming vs. reassuring thinking, and evidence an increase in the latter (measured via self-report log).

Intervention Strategy B: Relaxation Training

Tactic B: Teach and work with patient on behavioral posturing, diaphragmatic breathing, and progressive muscle relaxation that may assist patient with relaxing. Refer for a biofeedback assessment and utilize biofeedback-assisted relaxation if applicable.

Outcome Measure B: Patient's self-report log of relaxation and ability to recognize tension vs. relaxation.

Figure 9.2 Biopsychosocial impressions.

add variety to the patient's activities or exercise regimen might be beneficial. Similar to those in the preparation stage, patients in the action stage do engage in physical activity, but they do so on a more regular basis. The emphasis at this stage should be on providing patients with praise for their efforts but specifically focusing on relapse prevention; therefore, discussions with the patient regarding inevitable potential barriers (e.g., a vacation) and developing solutions are necessary. Additionally, discussions about coping with injury rehabilitation may be appropriate for individuals in this stage. The difference between patients in the action vs. maintenance stage of readiness for change is time; in the latter stage, patients have been engaging in regular physical activity, exercise, and/or sport behavior for 6 months or more.

Long et al.[48] examined a multisite field test of the acceptability of PACE and found that 100% of providers rated the program as "valuable" to "very valuable" and 100 of providers reported feeling "somewhat" to "very confident" in their ability to use PACE. In a controlled trial of the PACE program focusing on those in the contemplation stage of readiness for change, Calfas et al.[46] (1996) found that a greater percentage of PACE patients (52%) adopted regular activity than those in the control group (12%). PACE patients reported more walking (37 minutes more per week above baseline) than control patients (7 minutes more per week above baseline) at a 6-week follow-up. While these results are indeed promising, as mentioned earlier, interventions developed solely with respect to the individual's stage of readiness for change (i.e., intrapersonal variables) may not yield as promising results as an intervention that comprehensively considers the biopsychosocial model.[7,31]

The most important first step, then, to promoting physical activity, exercise, or sport behavior is to assess the patient's current level of activity.[34,49] Then, through a comprehensive understanding of physical, emotional, cognitive, behavioral, and environmental factors, physicians and patients will be able to target their efforts more appropriately and effectively. For example, a common belief among patients with chronic pain due to degenerative joint disease is that when they increase their activity level they might somehow harm themselves. Without addressing or clarifying such cognitive factors, efforts to increase patients' activity levels may be unsuccessful. Simply telling patients that they need to be more active will be ineffective. When specific physical activity and exercise recommendations are indicated, the physician should assist the patient with goal setting (e.g., writing an exercise prescription). Finally, physicians should be a role model for physical activity and exercise.

ISSUES RELEVANT TO COMPETITIVE SPORTS

More than 360,000 collegiate athletes and almost 6.5 million high school athletes participated in sports during the 1998–1999 school year.[50] Participating in athletics encourages the development of leadership skills, self-esteem, muscle development, and overall physical health. For children and adolescents, play and sport can enhance physical, psychological, and social development.[51] In addition to providing interventions for increasing physical activity and exercise among their patients, the primary care physician is also likely to encounter individuals, both children and adults, who present with issues relevant to participation in competitive sports. Among the most common issues they will face are performance issues and sports injuries.

Performance Issues

According to Howe and Ogilvie, "Optimal performance comes with a certain level of tension and mental activation."[52] For athletes struggling with arousal control, educationally based psychological interventions have yielded significant improvements in performance.[53] For the primary care physician, this might mean instructing patients in the use of performance imagery, such as mental rehearsal of their performance,[54] or encouraging an athlete to replace any sabotaging, negative thoughts (e.g., "I will never make this shot") with reassuring, more positive thoughts (e.g., "I have made this shot before and will try my best to make it again"). Educating and instructing patients in these techniques can help them develop a greater sense of arousal control and, more importantly, improve performance.

Injured Athlete

An injured athlete may present to the primary care physician with a concern about the injury itself but also with concern over the impact of the injury on present and future performance and concerns regarding the nature of the rehabilitation process.[55] In fact, Thompson et al.[56] suggest that "rehabilitation is 75% psychological and 25% physical." Further, successful rehabilitation should begin with a comprehensive case conceptualization using the biopsychosocial model, which then allows effective identification of target areas for treatment (Level of Evidence C, expert opinion).[55]

Effective treatment must then begin with a thorough understanding of the component parts of the rehabilitation process and a collaborative agreement on specific goals for treatment.[55] The provider may then choose from among several effective interventions to tailor a rehabilitation program to meet the athlete's needs. This might include the use of imagery and other mental devices, increasing social support, pain management, and/or other cognitive–behavioral techniques such as self-management training.[55]

Specialty Consultation for Performance Issues or Injury Rehabilitation Concerns

It is important that primary care physicians be vigilant of symptoms that may warrant referral for more extensive psychological assessment and treatment.[55] For example, Brewer et al.[57] reported that in a sample of orthopedic patients 33% of injured football players were regarded as depressed. A psychological referral may be indicated for refractory cases or when patient present with co-morbid mood symptoms (e.g., anxiety, depression, grief) that significantly impact personal, social, and/or occupational functioning.[58,59]

SUMMARY AND CONCLUSIONS

Given the physical and psychological health benefits of physical activity, exercise, and sport behavior, it is not surprising that national organizations and public health agencies have focused on targeting these health behaviors in our population. Unfortunately, targeting important public health issues, such as the low levels of physical activity and increasing rates of obesity, is not always done by primary care physicians. Physicians have cited lack of knowledge, limited resources, and limited time as among the barriers to addressing these issues effectively.[22–25] Through adoption of the biopsychosocial model, these barriers can be overcome and more effective interventions can be delivered.

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10 THE ATHLETE AND THE OUTDOORS: ENVIRONMENTAL INFLUENCES ON SPORTS*

David K. Lisle and Michael Kernan

INTRODUCTION

The environment in which we compete and recreate poses numerous challenges to the athlete and outdoorsmen. Awareness of potential dangers is crucial not only for the participants but also for the physicians who oversee these activities and provide care for the competitors. This chapter focuses on the influences of the environment on sports and outdoor recreation and the possible illnesses and injuries faced by those who participate in adverse conditions.

HEAT-RELATED ILLNESS

Heat-related illnesses present a danger to all athletes competing in high-temperature and high-humidity environments. Preseason football, endurance races, and any hot-weather athletic events may lead to heat stress ranging in severity from mild cramping to fatal heat stroke. Heat-related illnesses are preventable, and their declining numbers represent an increase in awareness of environmental risks and knowledge of appropriate preventive measures.

Heat Stress and Thermoregulation

The human body produces heat from basal metabolism ranging from 60 to 70 kcal/hr; without a mechanism to dissipate heat, body temperature would rise 1°C/hr.[1,2] Moderate physical activity increases heat production to approximately 100 to 250 kcal/hr, and with heavy exercise heat accumulation may reach 300 to 600 kcal/hr.[2] Furthermore, for each 1°C rise in body temperature, metabolic demand for oxygen increases between 6 and 13%.[2,3] In addition to heat gain from physical work, an athlete may accumulate heat from the environment, either from radiation from the sun or stored heat on the surface of the Earth. For all these reasons, an athlete's ability to dissipate heat is a crucial factor in preventing excessive rise in body temperature.

The body's mechanisms for heat dissipation primarily rely on conduction, convection, radiation, and evaporation. Conduction is the heat exchange between two surfaces in contact and is the least important for an athlete attempting to cool down. Convection occurs when heat

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transfers from the body surface to surrounding air or fluid. As air surrounding the body is warmed, heated air molecules move away and cooler ones replace them.[1] When a body vasodilates in response to heat, it loses heat by convection. Radiation, the transferring of body heat to a cooler environment, may account for 65% of heat loss provided the air temperature is lower than body temperature.[2] Evaporation of sweat normally accounts for 30% of the cooling of a body. About 1 kcal of heat is lost for each 1.7 mL of sweat evaporated from the skin.[2] Put another way, for every 100 mL of sweat evaporated, a 1°C decrease in core body temperature occurs. However, when ambient temperature reaches 95°F, radiant heat loss from the body to the environment ceases and evaporation becomes the only way for the body to dissipate heat. The evaporative mechanism decreases in proportion to humidity, as the moisture gradient from the skin to air is decreased under humid conditions.[2,4] If humidity level exceeds 75%, evaporative heat loss is less efficient and sweat that drips from the skin only worsens dehydration but has no cooling benefit. The combination of high ambient temperature and high humidity blocks the two main mechanisms the body has to dissipate heat, thus creating ideal conditions for heat-related illness.

In response to heat stress, the body's thermoregulatory mechanism increases blood flow to the skin through vasodilation and increases sweat production. Increased skin blood flow raises skin temperature, and heat is transferred from the body core to the skin surface and is lost by convection, as well as radiation.[1,4] During exercise, sweat production accounts for about 80% of total heat removed from the body.[3] The combination of increased sweating and vasodilation results in a decline in circulatory volume as well as a decrease in peripheral vascular resistance. To maintain blood pressure, cardiac output may double or quadruple, placing a strenuous load on the heart.[2] With only moderate heat exposure, the increased blood flow to the skin occurs at the expense of flow to other organs. The athlete's body may reach a critical point at which a higher rate may not be sustainable.[2,3,5]

Risk Factors for Heat-Related Illness

Predisposing factors for heat-related illness are listed in Table 10.1.[1] For the athlete, factors such as poor physical fitness, lack of adequate acclimatization, obesity, poor sleep habits, and dehydration increase the likelihood of heat-related illnesses. Taking certain medications (such as anticholinergics or sympathomimetics) or herbal supplements (such as ephedra) may lead to an increased basal metabolic rate (Level of Evidence B, systematic reviews).[6] When athletes consume ephedra (e.g., ma huang, an herbal supplement containing ephedra alkaloids), whether as an appetite suppressant or performance enhancer, they are at greater risk of suffering heat stroke. While ephedra may increase the contractile strength of muscle fiber and allows for quicker consumption of fat into energy, it also leads to greater peripheral vasoconstriction; in combination with caffeine, it increases the thermogenic effect of exercise by 20% and therefore leads to a heightened risk for heat stroke (Level of Evidence B, systematic reviews).[6,7]

Elderly people who lack the ability to increase cardiac output have impaired heat dissipation mechanisms, as do those taking medication that hinder perspiration and skin blood flow (e.g., anticholinergics). Preexisting heart disease or dehydration from alcohol or diuretic use can leave individuals at risk for heat-related illness. In infants and children, heat dissipation is more readily overwhelmed due to their greater ratio of surface area to body mass; therefore, their absorption of heat is more rapid. In addition, young individuals may not be capable of removing themselves from a hot environment, which, in combination with their relative inability to

TABLE 10.1
Risk Factors for Heat-Related Illness

Poor physical fitness
Lack of acclimatization
Obesity
Prolonged exertion
Lack of sleep
Dehydration
Use of heavy equipment or clothing
No air conditioning
Unable to care for self
Alcoholism
Drug use (e.g., anticholinergics, sympathomimetics, herbal supplements containing ephedra alkaloids)
Lack of shade
History of heat injury
Chronic diseases

Source: Adapted from Khosla, R. and Guntupalli, K.K., *Crit. Care Clin.*, 15, 251–263, 1999.

sweat compared to adults, results in an elevated predisposition for heat stress.

Minor Heat-Related Illnesses

Heat cramps represent the mildest form of heat-related illness and present as severely painful muscle contractions, usually in the large muscle groups of the legs.[5] Risk factors include poor conditioning, lack of acclimatization, dehydration, and an imbalance of sodium and water in the muscles, a negative balance that occurs when a large amount of sweat is replaced with hypotonic fluids.[1,3,8] The diagnosis is made from a history of cramps in muscles, usually quadriceps, hamstrings, or soleus muscles, but muscle contractions may occur in abdominal and intercostal muscles, as well. The cramps typically follow heavy physical activity and often occur in the evening after muscle cooling has taken place. Treatment consists of rest, passive stretching, and massage of affected muscle groups. Oral rehydration with a 0.1 to 0.2% salt solution (1/4 to 1/2 teaspoon table salt dissolved in one quart of water) is sufficient for mild cases.[8] In more severe cases, intravenous hydration may be required using isotonic fluids (0.9% NaCl). Adequate hydration with electrolyte solutions before and during exercise is usually adequate prevention. Heat cramps may be recurrent, and return to play is allowable only after hydration and electrolyte balances are normalized.[3,5]

Heat syncope occurs with prolonged standing after vigorous exercise. Vasodilation of peripheral blood vessels and resultant dependent pooling of blood in the lower extremities of a standing individual are typical precursors to heat syncope. Reduced venous return and decreased cardiac output lead to a drop in cerebral blood flow, causing severe lightheadedness or a transient fainting episode.[5] The loss of consciousness is self-limited and responds to placement in a recumbent position. Further treatment includes rest in a cool environment and oral rehydration, if necessary. Prevention of heat syncope involves avoiding prolonged standing, increasing venous return by flexing of leg muscles while standing, and lying down immediately upon feeling symptoms. Typically, core body temperature is normal and complete recovery is rapid following adequate rest in recumbent position.

Heat exhaustion is the most common heat-related illness in athletes and is characterized by profuse sweating, nausea, vomiting, weakness, fatigue, and lightheadedness. Usually core temperature rises above 100.5°F (38.0°C) but is below 104.9°F (40.5°C) in heat exhaustion, which is believed to be a continuum of signs and symptoms that lead to heat stroke.[2,4] A major factor differentiating the two diagnoses is the lack of profound central nervous system impairment in heat exhaustion; however, the diagnosis of heat exhaustion is one of exclusion, and initiation of treatment for possible heat stroke should not be delayed if any doubt exists.

Treatment of heat exhaustion includes lying down in a cool environment, removal of excess and restrictive clothing, and fluid replacement. Rehydration can be either oral or intravenous depending on the patient's overall condition. If oral hydration fails to provide improvement, intravenous rehydration should be started with either normal saline or Ringers lactate. Severe nausea or vomiting, absence of gag reflex, or minor mental impairment necessitates intravenous hydration. Most healthy young individuals with mild heat exhaustion improve after receiving intravenous fluids in the emergency department and do not require admission. These patients should avoid heat stress for 24 to 48 hours and continue to hydrate themselves even after discharge. The athlete with moderate to severe heat exhaustion should be monitored for electrolyte imbalance and should not return to competitive play until normal body weight is achieved.

Heat Stroke

Under extreme conditions, the amount of heat generated overwhelms the body's thermoregulatory system and it is no longer capable of adequate heat dissipation. In these instances, core body temperature may rise to dangerous levels, and if these temperatures reach critical levels heat stroke may result. Heat stroke is characterized by core body temperatures 105°F (40.5°C) or greater that cause acute mental status changes and produce multiorgan dysfunction.[1,8] Central nervous system alterations range from minor mental status alterations to delirium, seizures, and coma.[5] Taking a rectal temperature as soon as possible typically gives the most reliable and accurate maximum temperature and is crucial in evaluating any sick athlete.[9]

Heat stroke is a medical emergency, and anhidrosis is not mandatory to make the diagnosis. In a study of military recruits, half of the patients with heat stroke maintained their ability to sweat.[2,8] Hot, dry skin may be a late manifestation as a result of profound dehydration or necrotic plugging of sweat glands.[2] The diagnosis of heat stroke should always be considered in

TABLE 10.2
Exertional vs. Classic Heat Stroke

Exertional	Classic
Usually men (15–45 years)	Elderly age group
Previously healthy	Chronically ill
Strenuous exercise	Sedentary
No medications	Several medications
Sweating often present	Sweating absent
Lactic acidosis common	Lactic acidosis absent
Severe rhabdomyolysis	Rare rhabdomyolysis
Severe hyperuricemia	Mild hyperuricemia
Acute renal failure (ARF) in 25–30%	ARF in 5%
Marked disseminated intravascular coagulation (DIC)	Mild DIC
Creatine kinase (CK) markedly elevated	CK mildly elevated
Excessive endogenous heat production	Poor dissipation of environmental heat

Source: Adapted from Moran, D.S. and Gaffin, S.L., in *Wilderness Medicine*, Auerbach, P.S., Ed., Mosby, St. Louis, MO, 2001, pp. 290–316.

any patient exposed to heat stress with altered mental status.[1] The athlete who becomes irritable, aggressive, apathetic, or disoriented is showing warning signs for heat stroke. Almost all patients manifest signs of tachypnea, hypotension, and sinus tachycardia. Because the cerebellum is most sensitive to heat, ataxia may be an early sign.[2,8] Rapid diagnosis and treatment are crucial, as the more severe and prolonged the episode, the worse the predicted outcome, especially if cooling measures are delayed (Level of Evidence B, systematic reviews).[4,5,8]

Heat stroke is typically divided into two categories: classic and exertional. For the purpose of this text, the focus is primarily on the exertional type. The differences between the two are highlighted in Table 10.2.[8] Classic heat stroke occurs during maximal environmental heat stress and usually involves the elderly, poor, or indigent population. Onset of classic heat stroke is slow and is most commonly seen in urban settings during summer months. As would be expected, exertional heat stroke patients are a younger and healthier population engaging in vigorous exercise and the onset is relatively sudden.[1,4,8]

For all patients with elevated core body temperatures, an accurate diagnosis of underlying cause is of utmost importance. Although clinical suspicion is usually sufficient to diagnose heat stroke secondary to strenuous physical activity, an awareness of the differential diagnosis may help rule out clinical states that also cause elevated core temperature. A complete differential diagnosis divides hyperpyrexia into three categories: heat gain from the environment, increased heat production, and decreased heat dissipation.[5,8] Classic heat stroke from environmental factors is usually easily diagnosed by history. The remaining two mechanisms are highlighted in Table 10.3, which illustrates alternate etiologies for elevated core temperature and resultant heat stroke.[5]

TABLE 10.3
Differential Diagnosis for Heat Stroke

Increased exogenous heat load
High temperature
Sun exposure
Increased heat production
Overexertion
Infection (sepsis, meningitis)
Metabolic (thyroid storm, pheochromocytoma)
Drugs (sympathomimetics, antidepressants)
Decreased heat dissipation
Disorder of skin or sweat glands
Central nervous system disorders (subarachnoid hemorrhage, CNS trauma)
Drugs (anticholinergics, psychotropic agents)

Source: Adapted from Iseke, R.J., in *Textbook of Primary Care Medicine,* Noble, J., Ed., Mosby, St. Louis, MO, 1996, 63–66.

Management of Heat Stroke

Cooling of the heat stroke patient must be initiated as soon as the diagnosis is made. Morbidity and mortality are directly related to the duration and severity of hyperthermia. The athlete should be placed in a cool, shady area and all clothing should be removed. An ambulance should be called and basic life support (BLS) protocols should be followed, as necessary. If indicated and readily available, intubation, oxygen administration, and intravenous placement should be performed.

For rapid temperature reduction in the field, spraying of any available liquids on the athlete's skin will promote evaporative cooling. Any means of increasing the air flow around the patient is beneficial and potentiates the evaporative process. Evaporative cooling is the most available method in the field, and it is critical to keep the patient's skin wet. This cooling method does not interfere with resuscitation methods, and vasoconstriction and shivering are kept to a minimum. Evaporative cooling, however, has limited benefit in high humidity.[9] Wrapping the athlete in wet towels or sheets also may have cooling benefit; however, they must be changed frequently, as they warm and their cooling properties decline. While awaiting transport, nothing should be given orally, and anti-pyretics should be avoided. There is no role for aspirin or acetaminophen in heat stroke, and aspirin is contraindicated due to its effects on platelets and coagulation.[2]

Other methods of cooling in the field include placement of ice bags, chemical ice packs, or cold compresses in areas where large blood vessels come near the surface of the skin such as the neck, axilla, groin, and scalp. This technique has documented cooling rates of 0.1°C/min and avoids the deleterious effects of shivering and vasoconstriction.[2] Cool water immersion is quite effective; however, an immersion tub may not be readily available in the field, making this technique less practical.[9]

Once in an ambulance or in an emergency department setting, continuous core temperatures should be monitored using a rectal, bladder, or esophageal probe. The appropriate laboratory studies for a heat stroke victim are detailed in Table 10.4.[5] The optimal cooling method once the patient is in the emergency department remains controversial. Some centers will continue with more aggressive evaporative cooling, while others switch to immersion cooling techniques. Immersion in an ice water bath has documented cooling rates of 0.1 to 0.25°C/min and has been shown to lower body temperature to 102°F (39°C) in 10 to 40 minutes.[2,8] Although ice baths may help increase blood pressure by hypothermic vasoconstriction, they have the disadvantage of retarding heat loss by diminished peripheral circulation and generation of body heat through shivering.[1] Furthermore, ice water immersion makes monitoring body temperature with a rectal probe, protection of airway, and resuscitation efforts extremely difficult; also, defibrillation may be delayed until the patient is removed from the water. Whether by using aggressive evaporative techniques or immersion cooling, the target body temperature is 101 to 102°F (38 to 39°C). At these body temperatures, cooling should be stopped to prevent hypothermia.

TABLE 10.4
Laboratory Studies for Heat Stroke

CBC	CK, CKMB, troponin
Electrolytes	Lactate
BUN, creatinine	Fibrinogen and D-dimer
Platelets	ABG
PT, PTT	Urinalysis
Liver function tests	EKG

Source: Adapted from Barrow, M.W. and Clark, K.A., *Am. Family Physician*, 58, 749–756, 1998.

During the management process, administration of intravenous fluids consisting of D5 1/2 normal saline or normal saline should be guided by urine output, blood pressure, and laboratory values.[2,9] In the field, the athlete with heat stroke is not always volume depleted; in fact, a small number of symptomatic athletes presenting for care are overhydrated. A healthy competitor who sweats 1 L/hr and who is active for roughly 1 hour is not likely to be profoundly dehydrated; therefore, use of intravenous fluids in the field should be relegated to athletes who are unconscious or hypotensive.[9]

Heat stroke is a life-threatening emergency, and mortality rates in the general population range from 30 to 80%.[2,3] Survival rates in the athletic setting have greatly increased from 90 to 100% due to the improved rapid medical response in the field, early initial management, and aggressive cooling techniques.[9] Unfavorable prognostic indicators include a delay in cooling, core temperature greater than 105.8°F (41°C), and loss of consciousness over 2 hours. An elevated aspartate aminotransferase (AST) over 1000 U/L in the first 24 hours and acute renal failure are also correlated with a more unfavorable outcome.[4,8]

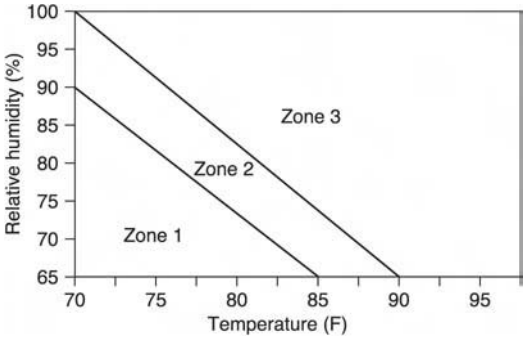

Figure 10.1 Heat stress danger chart. Zone 1, low-risk conditions; zone 2, moderate-risk conditions; zone 3, high-risk conditions. (Adapted from Barrow, M.W. and Clark, K.A., *Am. Family Physician*, 58, 749–756, 1998.)

Prevention of Heat Stroke

A key adjunct to proper treatment of heat stroke is vigilant anticipatory guidance and preventive measures. The American College of Sports Medicine has written guidelines of their recommendations stating specific precautions for outdoor athletic events. These include number of water stations, organization of medical facility, adequacy of medical equipment, and heat index guidelines. The heat index takes into account current conditions and helps athletic event staff determine the safety of holding a particular competition.

The number of participants in athletic activities has increased, as has the intensity and duration of events. Proper steps for prevention of heat-related illness can be divided into three categories: environmental awareness, acclimatization of athletes, and adequate hydration. Of course, athletes with predisposing conditions should be identified to avoid potential heat-related problems.

Most heat-related illnesses are directly related to the environmental conditions in which the athlete has been competing. Organizers of sporting events and the medical personnel should be aware of possible hazards. A knowledge of heat stress danger charts (Figure 10.1) is extremely useful in determining whether an event should take place.[4] These charts are based on ambient temperature and relative humidity. The wet-bulb-globe temperature (WBGT) uses a formula to create an index (Table 10.5) that guides event physicians.[10] The WBGT is based on several measurements such as ambient temperature, humidity, and radiant heat load. WBGT measurement requires a sling psychrometer or heat index thermometer. Both heat stress danger charts and the WBGT index offer objective measurements for event staff on which to base decisions. Coaches should adjust practice schedules during hot, humid weather in order to avoid the most intense heat at midday. Early season football games should be held at night and equipment should be breathable, lightweight, and light in color. Sports physicians have no control over the weather; however, if adjustments in exercise duration and intensity are insufficient, they should be prepared to cancel an athletic event if the conditions are deemed too dangerous.

TABLE 10.5
Wet-Bulb-Globe Temperature (WBGT) [a] and Risk of Heat Illness

Range (°F)	Range (°C)	Risk Level
<64	< 18	Low
64 < T ≤ 73	18 < T ≤ 23	Moderate
73 < T ≤ 82	23 < T ≤ 28	High
>82	>28	Hazardous

[a] WBGT = 0.7 (wet bulb) + 0.2 (globe temperature) + 0.1 (dry bulb).
Source: Adapted from Sparling, P.B., *Clin. J. Sports Med.*, 5:220–222, 1995.

As the athlete exercises in high temperatures and humidity, the body adapts to heat stress and has an increased tolerance for work under these conditions, a process called acclimatization.[3,4] The body becomes more efficient under heat stress and generates less internal heat.[3,8] Proper acclimatization takes between 7 days and several weeks with steady increases in duration and intensity of exercise. Repeated daily exercise of lengthening duration should occur during the hottest part of the day. Initial workouts should last only 30 minutes with gradual increases to 2 hours. Care should be taken to replace wet clothing and provide adequate hydration. Done properly, acclimatization results in an improved level of physical conditioning and lowers resting body temperature, lessens the increase in heart rate and respiratory rate, and causes the body to sweat earlier during exercise.[3,8]

Proper hydration is another key to preventing heat-related illnesses. The thirst mechanism is a late indicator of dehydration; therefore, fluids must be consumed before the urge arises. Frequent rest periods for rehydration should occur roughly every half hour; an approximate guideline for replacement is 10 oz every 30 minutes of strenuous exercise in extreme heat (Level of Evidence C, consensus/expert opinion).[3] Pre- and

post-exercise weights can also guide rehydration efforts, with about 16 oz of fluid replacement being required for every pound of weight lost.[4] Prehydration can provide a "fluid cushion" and delays the onset of dehydration. The goal for the athlete is to avoid experiencing thirst and to void light-yellow urine four times a day. Water is still the best means of hydration, but flavored sports drinks are acceptable if their glucose concentration does not exceed 5 to 8%.[3] It is recommended to dilute all flavored drinks 1:1 with water. Any drink with 10% glucose or more may cause stomach cramps or diarrhea during athletic activity.

Exertional heat stroke is a preventable medical emergency. Athletes should refrain from training during extreme heat, but if training is unavoidable then frequent periods of rest and hydration should be scheduled. Coaches, participants, and supervising physicians must communicate regarding potential dangers, and all involved must understand the impact of environmental factors, acclimatization, and hydration.

HYPOTHERMIA AND COLD INJURY

All outdoor enthusiasts at one time or another are faced with extreme cold, leaving them at risk for hypothermia and/or cold injury. Cold-induced injuries occur in a wide array of outdoor activities and may endanger the most active athlete as well as the stationary sports fan or hunter. Nearly all cold-induced injuries result from the inability to protect oneself from the environment. These injuries may be systemic (hypothermia) or local (frostbite), depending on the outdoor temperature and the body's exposure time, and the extent of injury is directly proportional to the degree of cold encountered.[11]

Hypothermia

Hypothermia is defined as an abnormally low core body temperature due to exposure to a cold environment. When heat loss exceeds heat production, hypothermia occurs in which body core temperature falls below 95°F (35°C).[12] The body's thermoregulatory response to extreme cold is to increase heat production through shivering, while core temperature is maintained by a decrease in peripheral blood flow.[13] Heat loss is influenced by both wind chill and the presence of wet skin or clothing. Wind chill may increase heat loss through both convection and evaporation, while moisture on either the skin or clothing may increase conduction of heat away from the skin, leading to more severe hypothermia. Water conducts heat 23 to 30 times faster than air, which explains the potentially dangerous hypothermic states caused by profound heat loss when bodies are partially or completely submerged in water.[14,15]

Although most cases of hypothermia occur during the winter, a large number of cases are seen during the summer and in relatively warm climates. Conditions that may lead to hypothermia are prolonged exposure to cold air, exposure to moderately cold and wet conditions in combination with exercise exhaustion, immersion in cold water, and drugs.[13] Alcohol and barbiturates, both of which alter the body's ability to regulate temperature and deteriorate mental status, lead to poor decisions and dangerous behavior. Activities that carry the highest risk for hypothermia are mountain climbing, hiking, skiing, hunting, and snowmobiling, but scuba diving, swimming, sailing, and fishing are also risky activities.

The hypothermic patient may present in many ways based on the degree of exposure; however, only three categories are used to describe hypothermia: mild, moderate, and severe.[12] Core temperature between 90°F (32°C) and 95°F (35°C) is considered mild hypothermia, while temperatures below 90°F but above 82°F (28°C) indicate moderate hypothermia. Severe hypothermia is marked by any core temperatures below 82°F (28°C).

The diagnosis of hypothermia is established when decreased core temperature is found, and an exact core reading is crucial to determine both the severity of hypothermia and appropriate treatment of the patient.[16] Core temperature is often inaccurately measured using traditional thermometers. The most precise measurements are taken with rectal or esophageal probes.

Depending on core temperature, hypothermia has a continuum of signs and symptoms as outlined in Table 10.6.[12] In mild hypothermia, the patient is cool and pale, and the peripheral vasculature is maximally constricted. Shivering reaches its maximal level and may be uncontrollable. A constellation of disorientation, impaired judgment, and dysarthria occurs in conjunction with an ataxic gait and an inability to perform fine coordinated movements with the hands.[12] Even at these levels of neurologic and musculoskeletal depression, the patient is tachypneic and tachycardic and has a resultant increased cardiac output and renal perfusion (cold diuresis).[12,17]

Symptoms of moderate hypothermia include more severely impaired judgment, dilated pupils, muscle rigidity, and hyporeflexia. Blood pressure, heart rate, and respiratory rate begin to decrease, and the shivering reflex ceases altogether. At 82°F,

TABLE 10.6
Signs and Symptoms of Hypothermia

Severity of Hypothermia	Temperature Range	Body Temperature	Characteristics
Mild	95°F (35°C) to 90°F (32.2°C)	95°F (35°C)	Maximum shivering
		93°F (34°C)	Dysarthria, poor judgment
		91.5°F (33°C)	Ataxia, apathy
Moderate	90°F (32.2°C) to 82.4°F (28°C)	88°F (31°C)	Shivering stops, stupor
		86°F (30°C)	Cardiac dysrhythmias, dilated pupils, paradoxical undressing
		82.4°F (28°C)	Ventricular fibrillation likely, 50% decrease in heart rate
Severe	<82.4°F (28°C)	80°F (26.7°C)	Voluntary motion ceases, pupils unreactive to light
		77°F (25°C)	Risk of cardiac arrest
		66°F (19°C)	Flat electroencephalogram
		64°F (18°C)	Asystole
		60.8°F (16°C)	Lowest temperature for adult accidental survivor
		59.2°F (15.2°C)	Lowest temperature for infant accidental survivor
		48.2°F (9.0°C)	Lowest temperature for therapeutic hypothermia survivor

Source: Adapted from Danzl, D.F. and Pozos, R.S., *N. Engl. J. Med.*, 331, 1756–1760, 1994, and Weiss, E.A., in *Proc. of the Sixteenth Annual National Conference on Wilderness Medicine*, Big Sky Resort, MT, July 31–August 4, 2002, pp. 243–247.

the heart becomes irritable and prone to developing arrhythmias, such as atrial and ventricular fibrillation.[18] In about 80% of hypothermic patients, an electrocardiogram may show a distinctive J-point elevation (a J wave or Osborn wave).[15] Because the patient's ability to generate heat is absent, if warming is not initiated the patient quickly cools to ambient temperature and dies.

In severe hypothermia, blood pressure is almost imperceptible due to the progressive decrease in heart rate and respiratory rate. The patient's muscles are areflexic, and if the cardiac rhythm is not asystole it is likely atrial fibrillation. Ventricular fibrillation may be induced easily, even by minimal movement of the patient.[18]

The practitioner in the field should estimate the severity of hypothermia by gauging level of consciousness and the presence of shivering. As a general guideline, in the absence of shivering or if level of consciousness is severely impaired, the patient's core temperature is likely below 90°F and therefore falls in the category of moderate hypothermia.

Basic life support should be instituted when necessary, but the use of endotracheal intubation or cardiopulmonary resuscitation (CPR) must be done cautiously. CPR is only indicated in patients with a nonperfusing cardiac rhythm. Because pulses are often difficult to palpate due to severe peripheral vasoconstriction and, moreover, because CPR may cause lethal cardiac arrhythmias, attempts to feel a pulse should last a full minute in order to avoid inappropriate chest compressions.[11,18] If core temperatures are below 86°F (30°C), advanced life support (ALS) protocols are mostly ineffective. Attempts at defibrillation should occur only after core temperature is raised above 86°F (Level of Evidence C, consensus/expert opinion).[17]

In all cases of hypothermia, therapy focuses on the most efficient rewarming strategies. When considering rewarming, a decision is made whether to use passive or active techniques. In patients who are otherwise healthy and suffer from only mild hypothermia, passive rewarming is usually sufficient (Level of Evidence C, consensus/expert opinion).[12] Passive rewarming involves

moving the patient to a warm environment and replacing wet clothes with dry insulated garments.[18] Insulating patients in a dry environment prevents further heat loss and should be considered in all hypothermic patients, regardless of severity.

Active rewarming is appropriate if the patient's core temperature is below 90°F (32°C), if signs of shock are present, or if passive rewarming techniques failed or were inadequate. Active rewarming is divided into two categories: active external rewarming and active core rewarming. Active external rewarming methods include the use of exogenous heat sources, such as hot packs, electric blankets, or heat lamps, or immersion in warm water. Care should be taken when using these methods to look for a phenomenon known as afterdrop. Afterdrop occurs after active rewarming when peripheral vasoconstriction is reversed and cold blood returns to the core from the previously cold extremities.[11,12] Also, rewarming shock may occur with the resultant venous pooling from peripheral vasodilation.

Active core rewarming uses the direct delivery of heat internally, and the most common of these techniques involves usage of warmed intravenous fluids to 104°F (40°C). Heated oxygen or humidified air may also be administered. All patients should receive warmed oxygen and intravenous fluids. Normal saline warmed to 104°F will not only aid in rewarming but will also help prevent hypotension from rewarming shock in those more severely hypothermic.[11]

Peritoneal dialysis and hemodialysis using heated crystalloid are examples of more invasive techniques for active core rewarming. The gold standard for active core techniques remains extracorporeal blood rewarming. Using arterial and venous catheters, this cardiopulmonary bypass is three to four times faster at rewarming hypothermic patients than any other methods (9 to 10°C/hour) (Level of Evidence B, clinical cohort study).[12,18]

Due to decreased metabolic requirements, severe bradycardia in the profoundly hypothermic patient may be sufficient for systemic perfusion. Efforts to revive the hypothermic patient should be initiated despite the absence of vital signs. Establishing the presence of a pulse by palpation of the carotid artery should last for one full minute. Because severe hypothermia mimics death, only after a patient has been rewarmed to 95°F (35°C) or greater without return of vital signs should consideration be given to termination of resuscitation efforts (Level of Evidence C, consensus/expert opinion).[15]

Cold Injury

Cold injuries are divided into two categories: nonfreezing injuries and freezing injuries (or frostbite). Both are potentially dangerous outcomes in most outdoor sports, especially those taking place in a cold environment. Joggers are most often affected by frostbite injury, but Alpine and Nordic skiing, mountaineering, and most other winter sports leave the participants at risk.

Nonfreezing injuries include chilblain, trench foot, and immersion foot. Chilblain occurs in individuals at higher risk due to predisposing vascular conditions, such as Raynaud's disease. In these susceptible individuals, chilblain is a peripheral vasoconstriction to cold exposure that is prolonged past the body's normal response, sometimes even beyond the actual cold exposure.[13] Tissue ischemia and edema occur most often in distal extremities, as well as in regions exposed to the cold. Treatment is usually supportive, so prevention is key.

Trench foot and immersion foot result in tissue injuries when moist and cold feet undergo prolonged exposure (days or weeks) to temperatures between 32°F and 50°F (0 to 10°C).[13] Trench foot occurs after wearing cold and wet footwear, while immersion foot describes injury to feet actually immersed in cold water. The initial stage of healing is prehyperemic, where feet are swollen and discolored and pulses and sensation are decreased. Next, hyperemia occurs where pulses return to normal or are exaggerated and the feet are hot, blistered, and painful. The last stage is that of recovery, where tissue and pulses return to normal.[11] Warming the injured feet and exposing them to air will usually resolve symptoms; however, cold sensitivity may persist. Prevention is most important and management is purely supportive.

Frostbite is an ischemic injury occurring after the actual freezing of tissue. Pathophysiology involves both ice crystal formation and vascular stasis. When enough heat is lost in an extremity or exposed anatomy, ice crystals form that not only disrupt the cell walls but also may change cellular osmolarity. When crystals form in extracellular fluid, osmolarity is increased accompanied by a resultant shift in fluid drawing water out of cells, raising intracellular concentrations to toxic levels. Vascular stasis also occurs with frostbite after vasoconstriction and increased blood viscosity cause a decrease in blood supply to tissue.[19]

Frostbite occurs when temperatures are below 32°F (0°C) and usually involves the distal extremities and areas of facial exposure. Risk factors for

frostbite include low temperatures, prolonged exposure, wind chill, and high altitude.[11,19] Frostbite has been described by degree of severity (i.e., first, second, third); however, a more clinically useful and simpler classification system categorizes frostbite as frostnip, superficial, or deep.[13] It should be noted that the severity of frostbite symptoms is directly related to underlying damage, and it is often difficult to determine severity until after thawing.

Frostnip is the earliest stage of freezing injury, and symptoms consist of cold sensation, pain, and numbness in the affected region. Typically, total loss of sensation does not occur. With rewarming in the field, the affected tissue returns to normal with care taken to prevent further injury.[13]

In superficial frostbite, the tissue freezing remains localized to skin and subcutaneous tissue. While the affected area is frozen, no pain or sensation is felt and the skin appears waxy with no capillary refilling. Pain begins on rewarming and progressively increases. As the area thaws, the skin is mottled and hyperemic, and the tissue becomes edematous, often with raised blisters. Blisters are often replaced with areas of eschar that appear similar to gangrene; however, tissue loss is rare in superficial frostbite.[19,20]

Deep frostbite is marked by freezing below the subcutaneous tissue layer. Before thawing, no pain is felt and typically sensation does not return, even after rewarming. With thawing, the skin remains at room temperature with no edema or blister formation, and the color is grayish and cyanotic appearing.[13,20]

Treatment of frostbite involves rapid rewarming of the affected extremities and should only occur after other medical emergencies, such as hypothermia, have been addressed. Once thawing begins, refreezing must be prevented because the resultant morbidity is more severe than extended freezing (Level of Evidence C, consensus/expert opinion).[11,13,20] In the field, padding and splinting of the extremity should occur, being careful not to rupture blisters. Once in an emergency department or more controlled environment, rapid rewarming should begin in a whirlpool bath or equivalent with water temperatures in the 100° to 110°F (38° to 43°C) range (Level of Evidence C, consensus/expert opinion).[19] The thawing tissue should be treated gently, and, while initial conditions may be nonsterile, post-thaw handling should use sterile techniques. Thawing should take around 30 minutes for superficial freezing and up to 60 minutes with deeper involvement.[13] Intravenous hydration is crucial before and during the thawing process, and intense pain is expected and should be adequately addressed.

Prevention of Hypothermia and Cold Injury

The keys to prevention of cold-induced injury are weather anticipation and awareness, thorough trip planning, and usage of proper clothing.[20] Heat production should be maximized through exercise and good nutrition, and heat loss should be minimized by wearing layers of loose-fitting insulation. Layering allows for adding or subtracting insulation as conditions change. Overheating and resultant perspiration may lead to dehydration and wetting of insulation; therefore, removal of layers prior to perspiration is recommended. Cotton clothing should be avoided due to its long drying time and loss of insulating properties when wet. More quality insulating materials include wool and polypropylene, both of which remain effective even when wet. Breathable foul weather gear, such as Gore-Tex™, allows vapor from perspiration to escape, thus reducing the risk of wetting deeper insulating layers. Depending on the severity of cold and length of exposure, toes and hands should be adequately protected, and ski hats should be used to protect the ears and minimize heat loss from the head. Under no circumstances should gloves be worn instead of mittens when temperatures are extreme; when insulating measures prove insufficient, external heat sources should be introduced, such as pocket warmers, warm liquids, or fire. Most importantly, when severe cold conditions present risks to athletes or outdoor enthusiasts, physicians or trip planners may prudently choose to postpone or cancel events. Therefore, the sports medicine or wilderness physician should be aware of each organization's cold weather safety regulations.

HIGH-ALTITUDE ILLNESS

High-altitude illness is a common form of morbidity and sometimes mortality for travelers to elevations above 8000 feet (2440 m).[21] The main altitude illnesses are acute mountain sickness (AMS), high-altitude cerebral edema (HACE), and high-altitude pulmonary edema (HAPE). AMS and HACE represent the neurologic pathology of altitude while HAPE refers to the pulmonary abnormalities.

Acute Mountain Sickness and High Altitude Cer ebral Edema

The incidence of acute mountain sickness is variable depending on the altitude reached and rate of ascent to that altitude.[22] For those visiting lower altitude Colorado ski resorts, the incidence is only about 15%, while among those flying directly to 14,000 feet (4367 m) the incidence of AMS reaches 70 to 100%.[21,22] AMS is the most common of the altitude-related illnesses and is typically a constellation of symptoms including headache, nausea, anorexia, dyspnea, insomnia, and lassitude associated with hypobaric hypoxia at high elevations.[22,23] High-altitude cerebral edema is defined as the progression of more severe neurologic symptoms such as ataxia and changes in consciousness in a person with AMS; therefore, HACE appears to be a more severe form of AMS. The incidence of HACE is 2 to 3% in hikers at 18,000 feet (5,500 m).[23] Specific clinical manifestations of HACE are severe headache, confusion, stupor, and coma. Although it is most frequently associated with very high elevations, HACE may occur at any altitude greater than 8200 feet (2500 m).[21] Progression from mild AMS to coma may occur within 12 hours.

The most critical factor in developing AMS is the sleeping altitude. AMS rarely occurs in those sleeping below 8000 feet (2440 m); between 8000 and 9000 feet (2700 m), the incidence is only 10 to 15%.[21,24] A dramatic increase in the incidence of illness occurs with sleeping above 9000 feet (2700 m), with greater than 20% affected.[24]

Susceptibility to AMS and HACE does not appear to be related to physical fitness or gender. Low vital capacity, low hypoxic ventilatory response (HVR), and severe pulmonary hypertension in response to hypoxia are three key contributing factors to AMS.[22] The most important risk factor and best predictor appears to be a previous history of altitude-related illness.[22,24,25] Other risk factors include having a primary residence below 3000 feet (900 m) and past medical history of cardiopulmonary disease.[25]

A high index of suspicion for AMS is necessary for any person at altitude with malaise and neurologic symptoms. The setting is typically an unacclimatized hiker with a rapid ascent to altitude. Early symptoms of AMS have been described as feeling exactly like a hangover, and often physical finding during the early stages are absent. When progressing from AMS to HACE, the finding are those of cerebral edema, with ataxia, change in mental status, and cyanosis being the most useful indicators of serious illness. Early diagnosis is key for successful management, and the differential diagnosis for AMS should include dehydration, exhaustion, carbon monoxide poisoning, hangover, viral syndromes, transient ischemic attack (TIA), meningitis, hypothermia, drug intoxication, and psychiatric problems.

The pathophysiology of advanced stages of AMS and HACE has been linked to brain swelling. Factors contributing to brain swelling include the rate of onset of atmospheric hypoxemia, hypoventilation due to low hypoxic ventilatory response (HVR), and impaired gas exchange in the lungs from interstitial edema.[22,24] The exact mechanism leading to brain swelling is not well understood; however, an individual's intracranial dynamics does appear to have an effect. Those with a higher ratio of intracranial cerebrospinal fluid (CSF) to brain volume seem to have an improved ability to compensate for brain swelling through increased ability to displace CSF. By having more intracranial space to cope with cerebral edema, these persons experienced fewer symptoms of AMS.[21,24]

The treatment of AMS and HACE is centered on reversing the offending causes and reducing brain swelling. The first step is always increasing oxygenation and/or reducing hypoxia. Usually, descent of as little as 1600 feet (500 m) is sufficient, but descent should be as far as necessary to obtain results (Level of Evidence C, consensus/expert opinion).[21] If oxygen is available, it should be used and has been shown to be especially effective in reducing headaches (Level of Evidence C, consensus/expert opinion).[22] In extreme cases, hyperbaric therapy in conjunction with oxygen is required. If patients are unacclimatized to high altitude, this process can be expedited pharmacologically through the use of acetazolamide 125 to 250 mg every 12 hours. In children, 5 mg/kg/day in two divided doses will work. Acetazolamide has been found to decrease CSF formation, promote diuresis, and stimulate ventilation (Level of Evidence B, clinical cohort study).[21,22] Symptomatic therapy is helpful for headaches and nausea. Analgesics such as ibuprofen or acetaminophen and antiemetics in the form of prochlorperazine are often used. Climbing to higher altitude after the onset of symptoms is contraindicated. After descent and adequate treatment, the patient may try to ascend once more using staged acclimatization.

In cases of HACE, it is necessary to reduce both brain edema and brain capillary leak by using renal diuretics (e.g., furosemide), osmotic diuretics (e.g., mannitol), or dexamethasone. Four

milligrams of dexamethasone by mouth (PO), intramuscular (IM), or intravenous (IV) every 6 hours has been shown to be as effective as or superior to acetazolamide; however, dexamethasone does not improve acclimatization and may need to be repeated until patient is evacuated to lower altitude due to the risk of symptom rebound upon termination (Level of Evidence A, randomized controlled trial).[21,24]

High-Altitude Pulmonary Edema

High-altitude pulmonary edema is the most frequent cause of death at high elevation. Fortunately, it is easily treated, but prompt diagnosis is key. AMS and HACE may precede HAPE, or HAPE may occur on its own, but often the symptoms overlap. HAPE occurs in about 1 to 2% of individuals at elevations exceeding 12,000 feet (3660 m) but has been described in rare instances at 8000 feet (2440 m).[23] It is most commonly experienced on the second night of sleeping at altitude and is linked to rate of ascent, degree of physical exertion, cold temperatures, and use of sleeping medications.[22,24]

Dry cough, dyspnea on exertion, and increased exercise recovery time are all early symptoms of HAPE. As the disease progresses, symptoms change to dyspnea at rest and a productive cough. The physician may note bilateral rales, pink-tinged frothy sputum, tachycardia, and tachypnea; however, these tend to be late findings.[26]

The pathophysiology of HAPE is non-cardiogenic as hemodynamic monitoring shows normal wedge pressures and echocardiogram indicates normal left ventricular function. The key appears to be high pulmonary artery pressure (PAP) secondary to elevated microvascular pressures in the lungs from uneven pulmonary arteriolar constriction.[21,22] This response is referred to as the hypoxic pulmonary vasoconstrictor response (HPVR) and at high altitude may lead to failure of the capillary membrane, causing interstitial edema.

To best manage HAPE, the patient should rest and be kept warm because both exercise and cold cause an increase in PAP. Oxygen is critical in these circumstances and administration of 4 to 6 liters may be required in severe cases of HAPE.[23] If oxygen and medical personnel are unavailable, immediate evacuation to a lower altitude is indicated with minimal exertion by the affected individual. Oral nifedipine (10 mg taken every 4 hours or 30 mg slow release every 12 to 24 hours) has been shown to reduce PAP by 30 to 50% and has become the drug of choice if oxygen administration or immediate descent is not possible (Level of Evidence B, nonrandomized clinical trial).[23,24] Once evacuated, hospitalization may be warranted in severe cases, and rapid recovery is seen with bed rest and oxygen (keeping $S_aO_2\% > 90\%$).[21] When HAPE has resolved, the hiker may ascend again; however, adequate acclimatization and preventive acetazolamide are recommended.

Prevention of High-Altitude Illness

Ascent to altitude in a slow, gradual manner is the best means of preventing altitude illness.[24] Often hikers will climb to higher altitude during the day and return to a lower altitude for sleeping. Recent recommendations suggest avoidance of sleeping 2000 feet (600 m) higher than the previous night once above 8000 feet (2440 m) and spending an extra night for acclimatization for every 2000 to 3000 feet (600 to 900 m) attained. Also, it is advisable to spend 2 to 3 nights at 8000 to 10,000 feet (2440 m to 3000 m) before climbing higher (Level of Evidence C, consensus/expert opinion).[21] Although a high-carbohydrate diet appears to reduce AMS by about 30%, strong conclusive evidence is lacking. Avoidance of respiratory depressants such as sleeping pills and drinking alcohol in very small amounts have been shown to decrease symptoms. Pharmacologic prophylaxis using acetazolamide or dexamethasone is indicated for forced rapid ascent or prior history of high-altitude illness. Acetazolamide is the drug of choice, and dosage is 5 mg/kg/day divided in 2 or 3 doses for 1 day prior and 1 day after ascent (Level of Evidence C, consensus/expert opinion).[22] For most hikers, acetazolamide 125 mg twice a day starting 1 day prior to ascent has been sufficient.[23,26] Dexamethasone has also been used to prevent high-altitude illness at dosages of 4 mg twice a day. This is useful for those allergic to acetazolamide or for insertion to extreme altitude with no chance for acclimatization.[22] Dexamethasone does not speed acclimatization like acetazolamide, and it must be continued for 3 to 4 days to avoid rebound symptoms. For most hikers, dexamethasone is reserved for summit day.[21,26] One study has shown the combination of acetazolamide and dexamethasone to be superior to dexamethasone alone (Level of Evidence A, randomized controlled trial).[27] Adequate hydration, avoiding overexertion, and exercising gently on acclimatization days are also key in preventing altitude-related illnesses.

SUMMARY

With an increasing number of people taking up exercise and traveling into the wilderness, a proportional rise in environment-related injuries and illnesses has occurred. Sports physicians, base camp doctors, and wilderness trip leaders must be cognizant of potential environmental hazards that might afflict their patient population. Extremes of heat, cold, and altitude offer challenges to athletes, and physicians must understand and treat the resultant pathologic changes that alter physical performance. To minimize morbidity and possible mortality, recognition, diagnosis, and management must be accomplished without delay. Preventive measures are key and if enacted properly may help avoid environmental injury or illness altogether.

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11 NUTRITION IN SPORTS*

Kim Edward LeBlanc

INTRODUCTION

Although many individuals perform little or no physical activity on a regular basis, many do live a very active lifestyle. These individuals are athletes in one phase of training or another, each at their own level of desire and ability. These athletes vary from young to old, and while none of them will become world-class athletes through dietary means, those with poor nutritional habits will never reach their full potential nor achieve their goals.

Athletic endeavors require high metabolic rates for extended periods of time both in training and competition. Dietary manipulation may be attempted at virtually every level of competition to enhance athletic performance. This is often done without adequate nutritional knowledge or sound nutritional advice. Dietary habits that are learned at a relatively young age may very well be carried forth into adult life. It is therefore imperative that any dietary manipulations be done with accurate and reliable information, particularly when instructing younger athletes or while serving as role models for them. Relying on myths and misconceptions is hazardous and frequently results in negative impacts on athletic performance. No known food or supplement will allow an athlete of mediocre ability to become an Olympic champion. Sound nutritional practices must be in place to allow athletes to perform at their very best and make the most of their potential by providing adequate nutrients for tissue maintenance, growth, and repair while avoiding intake that is excessive and unnecessary.

CALORIC REQUIREMENTS

It is difficult to provide an exact estimation of daily caloric requirements of any particular individual due to varying physical activity levels, genetics, medical illnesses, lifestyles, and other issues; however, it is quite apparent that physically active individuals will have higher caloric requirements to maintain body function and ensure optimal athletic performance than inactive, sedentary persons. Athletes trying to maintain a certain level of fitness may require a greater intake

* In the second edition, this chapter was authored by Randolph L. Pearson.

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TABLE 11.1
Formulas for Calculating Caloric Requirements

Method 1

Weight (lb) × 10 = basal energy needs + active calories; calculate active calories as follows:

Weight (lb) × 3 for sedentary

Weight (lb) × 5 for moderately active

Weight (lb) × 10 for very active

Method 2

Weight (kg) × 25 for sedentary

Weight (kg) × 30 for moderately active

Weight (kg) × 40 for very active

Weight (kg) × 45 for underweight

of nutrients than others who may be exercising to reduce weight. One pound will be gained or lost by the addition or reduction of 3500 calories from the diet; for example, an athlete who is losing 1 pound per week could correct the situation by adding 500 calories to his daily diet or by reducing his workouts an appropriate amount. When calculating caloric requirements, it is important to remember that aerobic activity involves the repetitive use of large muscle groups over time and burns more calories than anaerobic activity, which involves short bursts of activity that result in little caloric expenditure (Table 11.1). Keeping track of the athlete's weight and maintaining a food diary is perhaps the best method of ensuring optimal caloric intake.

MACRONUTRIENTS

Macronutrients are those substances that are necessary in large quantities and have large molecular structures. The most important of these are carbohydrates, proteins, and fats. These three substances make up the bulk of the required nutrients for optimal athletic performance. These three nutrients provide the necessary energy to maintain bodily functions, both at rest and during physical activity. In addition, these same nutrients help maintain the structural and functional integrity of the human body. The basic requirements are the same for both sedentary and physically active individuals; however, those who are physically active will require a greater amount above the basic needs, which are usually obtained through the normal increases in dietary intake that accompany such activity.

Carbohydrates

Carbohydrates may be thought of as the most important fuel necessary for muscle training. The requirement for this macronutrient increases as the intensity of the exercise increases. Carbohydrates contribute most of the energy utilized to support the muscle activity. These carbohydrates are stored in the liver and muscles in the form of glycogen. Prolonged exercise will result in glycogen depletion and subsequent muscle fatigue, with an inability to continue the exercise. Consequently, it is imperative that the athlete consume adequate quantities of carbohydrates not only to maintain glycogen stores in the liver and muscles but also to provide the fuel reserves necessary for continued athletic participation.

Dietary carbohydrates have a major role in a physically active lifestyle. In a typical American diet, carbohydrates make up approximately 40 to 50% of the total calories consumed. Due to increased fuel needs, the diet of an athlete should include at least 50 to 60% carbohydrate calories. During periods of heavy workouts and training, these requirements should be increased to 70% of total calories ingested. This would amount to about 400 to 600 g/day for those who exercise regularly. This compares to 300 g/day required by a typical sedentary male weighing 70 kg. These carbohydrates should be made up of complex carbohydrates, such as fruits, vegetables, and unprocessed, high-fiber grains. Simple carbohydrates such as table sugar should be ingested in limited quantities. This recommendation assumes that energy expenditure is balanced by adequate intake. More specifically, athletes engaging in heavy endurance exercise should consume approximately 10 g of carbohydrates per kilogram of body weight. This will provide adequate fuel for activity and maintain glycogen stores within the muscle and liver.

Proteins

Proteins have many functional roles within the body. Proteins are important for synthesizing tissue, enzymes, coenzymes, blood components, vitamins, neurotransmitters, and hormones, among their many functions. Collagenous proteins make up the structural components of tendons, ligaments, bone, hair, and nails while proteins such as actin and myosin provide the structure of muscle tissue. The current recommended daily allowance (RDA) of protein is 0.8 g/kg body weight per day. This is usually a sound recommendation for active individuals, and proteins should account for approximately 15% of

the total daily caloric intake. The daily diets of exercising adults will usually contain this amount of protein with ease; however, it has been suggested that athletes engaging in heavy training, particularly strength training, should ingest between 1.2 and 1.8 g (up to 2.0 g) of protein per kilogram body weight.[1–3] In actuality, this amount of daily protein intake is usually normal for this type of athlete, who thus has no need for additional protein supplementation. Supplementation might be considered for the unusual strength-training athlete who is unable or unwilling to eat a proper diet that would include the necessary amount of protein.

Fats

Fats have received a considerable amount of negative attention, most of which is justified; however, it must be remembered that fats do serve a beneficial purpose. Fats provide a form of energy storage; in fact, they are the only form of energy storage in the body. Additionally, fats serve to transport fat-soluble vitamins as well as to provide thermal insulation and protection to vital organs. Fats provide a tremendous store of energy that is in a very concentrated form and relatively water free, as opposed to glycogen, which is hydrated and heavy. Stored fat is the ideal fuel for maintaining the ability to continue a prolonged physical event, such as a marathon or 100-mile cycling event. Fat is the preferentially used substrate during prolonged activity. The longer the event, the more fat that is consumed by the exercising body. The current recommendation of fat intake is 20 to 25% of the total daily caloric intake, well below the usual daily intake of 34 to 38% in a typical American diet. Furthermore, of these calories, the vast majority should consist of unsaturated fatty acids, with saturated fats making up less than 10% of the total amount consumed. Finally, it should be recalled that fats burn in a carbohydrate flame. This means that the primer for fat metabolism is carbohydrate. Insufficient carbohydrate intake results in a greater rate of fat mobilization than oxidation which, in turn, allows the formation of ketone bodies. Excess ketone formation results in an increased acidity in the body that is certainly detrimental to exercise performance.

MICRONUTRIENTS

Micronutrients are present in very small quantities. In spite of these small quantities, these micronutrients play a major role in efficient and effective regulation of many metabolic processes. The vitamins and minerals that make up these micronutrients play very specific roles in facilitating energy transfer and the synthesis of tissue. Most athletes will naturally obtain much more of these nutrients than is required for their normal daily level of activity; regardless, some athletes still supplement their diets with them, which could pose a significant health risk.

Vitamins

The diet must contain sufficient amounts of vitamins to fulfill the body's requirements because the only vitamin that the human body is able to produce is vitamin D. If the diet lacks some of them, they must be supplemented. The 13 known vitamins may be classified as either water soluble or fat soluble. B-complex vitamins are water soluble, while vitamins A, D, E, and K are fat soluble. This distinction is important and should be kept in mind considering the frequency with which athletes ingest vitamin supplements. The ingestion of water-soluble vitamins is usually without consequence because they are eventually excreted in the urine. On the other hand, fat-soluble vitamins will accumulate in the body when taken in excessive quantities, potentially leading to toxic levels and resultant adverse consequences.

Many athletes take antioxidants in the hopes of forestalling damage or enhancing athletic performance. During normal daily respiration, free radicals are formed. These are molecules that lack one electron and exist in various forms. These free radicals have been shown to be a cause of oxidative stress and damage to cellular elements from lipid peroxidation. It has been suggested in the literature that antioxidants may protect the body from these harmful oxidative effects as well as from heart disease and cancer. Definitive data on this subject are still not available, and it deserves further study.

With few exceptions, too few data support the notion that exercise performance will be enhanced or improved by the supplementation of vitamins beyond the recommended daily allowances. Various studies have shown conflicting reports, and few have actually evaluated specific athletic events. One study showed statistically significant improvement in 5-km race performance with the use of β-carotene.[4]

Minerals

This group of micronutrients consists of 22 elements, most of which are metallic in nature. They are involved in many essential areas of bodily functions; for example, iron is essential in the blood,

and bone requires calcium. Similar to vitamins, few data support the idea that supplementation of any of these minerals can improve athletic performance unless the athlete is in a state of deficiency.

The most common mineral deficiency in the United States is the result of inadequate dietary intake of iron resulting in anemia, which may dramatically and negatively affect athletic performance. Iron is obtained from animal sources such as meat and poultry as well as plant sources such as peas, beans, and leafy green vegetables. However, in spite of the ease with which iron may be consumed, for a variety of reasons (particularly inadequate diet and menstruation) many female athletes suffer from anemia. One of the first complaints that an athlete may have is simply a decreased ability to perform at a previous level. This topic is discussed elsewhere in the text; suffice it to say here that the RDA for females between 11 and 50 years of age is 15 mg/day (18 mg if menstruating, 10 mg for men). Long-term ingestion of lesser quantities will result in anemia and negative impacts on athletic ability and performance.

The most abundant mineral in the body is calcium. It may be combined with phosphorus to form bones and teeth or may exist in its ionized form, where it plays a valuable role in nerve conduction, muscle function, and blood clotting. Although calcium plays an important role in athletics because it helps optimize muscle performance, it is most frequently discussed in its relation to osteoporosis, or thinning of bone. The condition may be seen in athletes, particularly females who maintain poor eating habits — most notably, gymnasts and ballet dancers. Ingesting 1200 to 1500 mg/day is strongly recommended. The diet should be modified to ensure that this amount of calcium is ingested; otherwise, supplementation will be necessary.

FLUID REQUIREMENTS/ REPLACEMENT

As exercise begins, muscles begin to work, increasing bodily fluid requirements. Fluid loss from sweating also greatly increases the fluid needs of the exercising athlete. Larger athletes may lose 2 liters per hour or more depending on the time of year and climate. Any amount of dehydration is to be avoided if at all possible because any degree impairs performance. Performance is negatively altered due to the effects on physiologic function and thermoregulation. The thermal and metabolic demands of exercise will not be met, resulting in significant reductions in temperature regulation and circulatory capacity. A 1-pound loss of weight translates into a 1-pound loss of fluid. It should be noted that the negative effects of dehydration begin to occur with the loss of as little as 2% of body weight.

All athletes should be hydrating at least 24 hours prior to competition. Some athletes may be in a minor stage of dehydration due to training. Pre-event rehydration is the best time to play catch-up and bring fluids to their optimal level before the athletic event begins. In addition, it is recommended that approximately 20 minutes before the event, the athlete consume 400 to 600 mL of cool water (particularly if exercising in the heat). This has the additional benefit of increasing stomach volume, which increases gastric emptying. This minimizes the time fluids spend in the stomach and speeds their delivery to the working muscle. Once the exercise has begun, drinking 8 ounces or so every 15 to 20 minutes will aid in replacing lost fluids. The actual amount required to maintain a positive fluid balance may be determined by monitoring body weight during practice or training.

Age Considerations

Nutritional requirements for athletes vary according to sport, activity level, and age. Young and preadolescent athletes require 60 kcal/kg of ideal body weight per day for normal growth and development; therefore, these athletes will require additional caloric intake to maintain growth and development and to compensate for the increased energy demands. Simply increasing the diet in these athletes should be sufficient; however, the diet should be monitored so that it has an appropriate distribution of carbohydrates, proteins, and fats. Excess fat should be avoided.

Adolescent athletes also require additional calories to compensate for normal growth spurts, which can account for 5 to 20 pounds of fat-free weight in a year. This is also a time when dietary habits typically deteriorate (e.g., eating disorders) in spite of athletes indicating that they are eating well. Iron and calcium intake, particularly in females, should be emphasized. Dietary intake should be discussed thoroughly with these athletes, and any dietary misinformation or misconceptions should be corrected. Misconceptions abound during this time of life.

As our population ages, so does the number of individuals maintaining active lifestyles. Those who were exercising in their 30s and 40s are now continuing to do so well into their 60s and 70s and beyond. As a person ages, the calories

necessary to maintain adequate nutrition decreases. For example, athletes require approximately 150 calories per day less from ages 35 to 55 than when they were teenagers or in their early 20s. Athletes between the ages of 25 to 35 years old require about 50 calories per day less than when they were younger.

CARBOHYDRATE LOADING

As noted earlier, the most important fuel for muscle activity is carbohydrates. It may be recalled that carbohydrates are stored in the form of glycogen within the muscles and liver. A technique was developed in the late 1960s that has become known as carbohydrate loading. The premise is to increase the ingestion of carbohydrate prior to the targeted activity (e.g., long endurance event) so that the muscles and liver become "superconcentrated" with glycogen. This increase in glycogen stores would provide more fuel for activity for a longer period of time than without carbohydrate loading. This has been repeatedly shown to be an effective method for enhancing performance for events lasting longer than an hour, such as marathons and/or triathlons. It has provided little, if any, benefit for shorter events.

The downside of this classic method is the depletion phase, which calls for little or no carbohydrate intake while exercising to exhaustion or near exhaustion. This causes the athlete to have feelings of glycogen depletion such as fatigue, extreme lassitude, and inability to complete a workout satisfactorily. In addition, during the loading phase, along with the increased storage of glycogen comes additional storage of water. Although increased water may be beneficial from a physiological standpoint, the athlete may feel "too heavy" and, at times, uncomfortable. This extra weight also increases the energy required to perform an event such as running, a problem that may not pose any difficulty to some athletes.

It has been determined, however, that the beneficial effects of carbohydrate loading may be accomplished without a strict depletion phase. This modified 6-day loading technique begins with the athlete exercising for approximately 1.5 hours each day at a 75 to 85% effort and then gradually tapering on the remaining days, a process beginning on the 6th day prior to the event. During the first 3 days of this process, total caloric intake should consist of 50% carbohydrates followed by an increase to 70% of calories on the final 3 days before the competition. This may be as much as 500 to 800 g of carbohydrates, depending on the normal dietary intake. This modified carbohydrate loading procedure has been shown to allow glycogen reserves to accumulate to the same level as the more traditional technique.[5]

PRE-GAME MEAL

The importance of a pre-game meal is of particular significance for events that take place in the morning after an 8- to 12-hour fast that normally occurs during sleep. Without replacing the glycogen that was used during the evening and night, the athlete will enter the event with less than optimal fuel reserves. This occurs even if the athlete has been strictly following dietary recommendations in the days and weeks prior to the event. Attention should also be focused on proper pre-event fluid intake, as the overnight fast will have a negative effect here as well. Fluid intake should be generous and amount to at least 16 to 32 ounces.

Foods to avoid for the pre-game meal are those high in protein and fat. These foods should be virtually eliminated the day of the competition. These foods digest slowly and will remain in the gastrointestinal tract for too long a period of time. In addition, prolonged digestion shunts blood away from the working skeletal muscle (where it is needed most during exercise) and into the working intestine. Other food items to avoid consist of simple carbohydrates and sugars, which impair glycogenolysis and free-fatty acid mobilization; caffeine, which acts as both a stimulant and diuretic; and alcohol, which acts as a diuretic and impairs central nervous system function.

The pre-game meal should be carbohydrate rich to maximize glycogen storage in the liver and muscles. The athlete should consume a liquid or solid meal that has between 150 and 300 g of carbohydrates (3 to 5 g/kg of body weight) within 3 to 4 hours of the competition. Some athletes may not be able to consume this amount of carbohydrates in solid form for a variety of reasons (e.g., pre-race anxiety, superstition, travel time, food unavailability, etc.). Liquid meals will provide a high carbohydrate content as well as additional fluids. Additionally, liquid meals may be used during a prolonged event or competition such as all-day track or swimming events. Whatever is chosen, never introduce anything new or strange into the athlete's diet at this time. The food that is consumed prior to the event must be accepted by the athlete both mentally and physically. Just before an event is not the time to

realize that what you just ate does not agree with you!

There has been some debate as to whether or not fructose is a beneficial source of energy prior to prolonged exercise. It is absorbed more slowly than glucose and causes only very minimal insulin response or decline in blood glucose; however, before fructose can be utilized by the muscle it must be converted to glucose. This occurs after it has been transported to the liver for conversion, which delays its eventual entry into the bloodstream for effect. Furthermore, consuming a beverage that has a high fructose content may produce vomiting and diarrhea, both of which are certainly undesirable prior to any event of any sort.

POST-GAME MEAL

The optimal time to initiate glycogen replacement is immediately upon completion of the event, and it can be in solid or liquid form. This will allow glycogen resynthesis to occur as rapidly as possible. Although glycogen levels will gradually be restored over time, the best time to replenish is within the first 2 hours following the exercise.[6] Post-exercise carbohydrate replacement that begins immediately has been shown to restore muscle glycogen nearly three times faster than waiting for 2 hours.[7] This is important for athletes who will begin training again within a short period of time. Recovery time will be shorter and optimal with proper nourishment. This is not to say that an athlete cannot restore liver and skeletal muscle glycogen concentration to pre-exercise levels without immediate carbohydrate consumption. Glycogen stores are replenished at a rate of 5 to 7% per hour with optimal carbohydrate ingestion. Even if the best replacement recommendations are followed, after a glycogen-depleting event it will take up to 20 hours to fully replace the body's glycogen stores.[8] The same foods that were chosen as a pre-race meal and for carbohydrate loading would be good choices here. These foods should be high in carbohydrate content, such as potatoes, bread, pastas, brown rice, and bananas. Legumes and milk products should be avoided during rapid replenishment only because they are slowly absorbed by the intestine.

ENERGY SUPPLEMENTS

While it is true that all of the required nutrients and calories are available in a normal diet, high-performance athletes may have some difficulty consuming adequate quantities of them, potentially resulting in impaired performance. Today, energy bars and sports drinks are readily available to supplement the added needs of athletes.[9] These supplements represent an energy-dense food that is quite convenient to utilize. Athletes need to read the labels on these supplements. As always, they should assist these competitors in maintaining the optimal intake of carbohydrates, proteins, fats, and fluids.

SUMMARY

Many myths and misconceptions regarding nutrition pervade our society. It is incumbent on all physicians, particularly those who regularly treat athletes, to be familiar with the basics of sports nutrition. Athletes are often confused by all the information available in magazines and newspapers, at gymnasiums, and on the Internet. In spite of this explosion of information, the basic advice remains the same. It is imperative that all athletes maintain and replace the fluids and fuel necessary for activity, most notably carbohydrates. Vitamins and minerals do play an important role in the health of athletes, but most athletes naturally consume larger quantities of food and receive more than their recommended daily allowance. Scientific data to support the need for further supplementation are lacking. The one exception may be strength-training athletes. Sports nutrition is often viewed as a minor player in the overall health and ability of an athlete, but it is here that training begins. If an athlete's nutritional needs are not met, however, it is here that the training ends.

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12 PERFORMANCE-ENHANCING SUBSTANCES: THEIR USE IN SPORT*

Mark B. Stephens and Richard B. Birrer

HISTORY

Since ancient times, athletes have ingested substances designed to impart speed, strength, or agility or to improve personal appearance.[1] Greek and Roman athletes used mushrooms, opiates, and other "performance-enhancing" products to achieve a competitive edge. Cyclists in the 19th century used heroine, cocaine, and ether to improve performance. The winner of the 1904 Olympic marathon used a concoction of strychnine and brandy to augment performance, and the winner of the 1920 Olympic 100-meter dash used a mixture of sherry and raw eggs to enhance speed.[2] Modern athletes are no different. Recent high-profile incidents involving anabolic steroid use disqualified the 1998 Olympic 100-meter dash winner Ben Johnson and banned the 1996 shot-put champion Randy Barnes from international competition. The pressure to succeed is intense for modern athletes. One informal survey of Olympic-caliber athletes indicated that a vast majority would use a banned product if they were certain that use would go undetected and guarantee victory.[3] The same survey also suggested that these athletes would continue to use the same substance for several years to maintain the competitive edge, even if use resulted in premature death (Level of Evidence C, survey). Table 12.1 presents a table of common performance-enhancing substances.

DEFINITIONS

When discussing the use of performance-enhancing substances (PESs) in sport, three general categories

* In the second edition, this chapter was authored by Bruce H. Woolley and Richard B. Birrer.

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TABLE 12.1
Current Performance-Enhancing Products

Ergogenic Aid of Interest	Reported Action(s)	Research on Ergogenic Effects	Reported Side Effects	Product Legality
Alcohol	Decreases anxiety	No benefits	Significant	Banned for shooting events
Amphetamines	Improves concentration, decreases fatigue and appetite	Mixed, some positive	Significant, dangerous	Illegal
Anabolic steroids	Increases strength, lean muscle mass, and motivation	Positive	Significant, dangerous	Illegal
Androstenedione	Same as anabolic steroids	Refutes, no benefits	Significant	Banned by IOC, NCAA
Beta blockers	Decreases anxiety	Positive effect on fine motor control, negative effect on aerobic capacity	Significant	Banned by IOC
$Beta_2$ agonists	Increases lean muscle mass	Mixed, no benefit from inhaled formulations	Mild	Banned by IOC, legal when prescribed
Blood doping	Increases aerobic capacity	Supports	Significant, dangerous	Illegal
Caffeine	Increases muscle contractility and aerobic endurance, enhances fat metabolism	Supports	Mild	Legal to urine level of 12 to 15 μ g per mL
Cocaine	Stimulates CNS, delays fatigue	Mixed	Significant, dangerous	Illegal
Creatine	Increases muscle energy, endurance, strength, and lean muscle mass	Supports, insufficient data on long-term use	Mild	Legal
Diuretics	Decreases body mass	Limited benefit	Potentially dangerous	Banned by IOC
Ephedrine and other sympathomimetics	Stimulates central nervous system, increases energy, delays fatigue, stimulates weight loss	No benefit	Potentially dangerous	Banned by IOC, some other organizations
Ephedrine plus caffeine	Increases energy, stimulates weight loss	Supports	Potentially dangerous, fatal at high doses	Banned by IOC, some other organizations
Erythropoietin	Increases aerobic capacity	Supports	Significant, dangerous	Illegal
Gamma-hydroxybutyrate	Stimulates growth hormone release and muscle growth	Limited, refutes	Significant, dose-related; abuse potential	Illegal
Human growth hormone	Has anabolic effect on muscle growth, increases fat metabolism	Refutes, limited ergogenic benefits	Significant, dangerous	Illegal

TABLE 12.1 (CONTINUED)
Current Performance-Enhancing Products

Ergogenic Aid of Interest	Reported Action(s)	Research on Ergogenic Effects	Reported Side Effects	Product Legality
Marijuana	Decreases anxiety	Refutes, negative effect	Significant, dangerous	Illegal
Narcotics	Increases endurance by suppressing pain, decrease anxiety	Mixed, negative	Significant, dangerous	Illegal

Source: Adapted from Ahrendt, D.M., *Am. Family Phys.*, 63, 913–922, 2001.

of products are used by athletes to enhance performance: prescription medications, nutritional supplements, and illegal/banned substances. The use of multiple drugs and a regimen intended to maximize desired effects, minimize negative side effects, and/or avoid detection in drug-testing scenarios is termed *stacking*. The use of multiple drugs in a sequence to maximize desired effects, minimize negative side effects, and/or avoid detection in drug-testing scenarios is termed *cycling*.

Prescription Medications

According to the Food and Drug Administration (FDA), a drug is any product intended to diagnose, cure, mitigate, treat, or prevent disease. Any product that is marketed as a drug must undergo clinic trials under strict FDA regulation to prove effectiveness, safety, potential drug interactions, and appropriate dosage. Prescription medications are available only under the supervision of a licensed medical provider. Some prescription medications (such as anabolic steroids, human growth hormone, and erythropoietin) have been diverted from legitimate medical use for illicit athletic use.

Nutritional Supplements

In contrast to prescription medications, dietary supplements are defined as any products (other than tobacco) that are solely intended to supplement a regular diet. Dietary supplements must contain one or more of the following as a primary ingredient: vitamin, mineral, herb, or amino acid concentrate, metabolite, or "extract." This nebulous definition has led to the explosion of an international supplement market with annual sales approaching $30 billion.[2] Much of the increase in supplement sales is the result of the Dietary Supplement Health Education Act passed by Congress in 1994. This act was intended to support an increase in popular demand for "natural" products. While the FDA does have the authority to establish good manufacturing procedure regulations governing the preparation, packing, and storage of dietary supplements, the FDA does not authorize or test dietary supplements. When a product is marketed, the FDA has the responsibility to show that a supplement is unsafe before it can take action to restrict use of that product.

Illicit Drugs

In addition to prescription medications and nutritional supplements, illicit or recreational drugs are used by athletes to enhance competitive performance. These products have no proven medical indication and are illegal. Table 12.2 presents a list of common prescription medications, nutritional supplements, and illegal drugs encountered in the athletic arena.

EPIDEMIOLOGY

Current estimates suggest that close to half of the population of the United States uses some form of nutritional supplement.[4] The reasons individuals cite for supplement use are varied and include ensuring good nutrition, preventing illness, improving performance, warding off fatigue, and enhancing personal appearance.[4] The prevalence of creatine use among young athletes varies from 6 to 45%, depending on age and sport.[5] The self-reported incidence of anabolic steroid use has been reported to be between 4 and 12% of the adolescent male population and between 0.5 and 2% of the adolescent female population (Level of Evidence C, survey).[6] In a random survey of health clubs and weight-lifting facilities, 18% of men reported androstenedione use, 25% reported using ephedra alkaloid products, and fully 5% reported use of anabolic–androgenic steroids.[7] Based on these data, it is estimated that 1.5 million individuals have used either androstenedione or anabolic steroids, and close to 3 million individuals

TABLE 12.2
Classification of Drugs and Supplements Used for Enhanced Sports Performance

Prescription Drugs	Dietary Supplements	Illicit Drugs
Anabolic steroids[a]	Androstenedione[a]	Amphetamines[a]
Beta blockers	Caffeine	Cocaine[a]
Beta agonists	Creatine	Gamma-hydroxybutyrate[a]
Diuretics	Ephedra alkaloids[a]	Marijuana[a]
Human growth hormone[a]	Vitamin supplements	
Corticotropin	Mineral supplements	
Theophylline	Amino acid supplements	
Narcotics[a]		

[a] Banned by most sports governing bodies. Other products listed may also affect an athlete's eligibility depending upon type of competition, medical indication, and rules of competition.

Source: Adapted from Koch, J.J., *Ped. Rev.*, 23(9), 200–212.

have used ephedra alkaloids in the past 3 years (Level of Evidence C, survey).[7]

PREVENTIVE COUNSELING

When counseling patients about PES use, it is helpful to use the four-question mnemonic "SOLE" (safety, outcomes, legal, ethical).[8,9] In other words, is the product safe? Are outcomes improved by use of the supplement? Is the product legal? Is the product ethical? Clinicians can readily adapt these simple questions to discussions with athletes about the use of ergogenic products.

NUTRITIONAL SUPPLEMENTS

Creatine

Creatine is a naturally occurring compound that is synthesized *in vivo* from the amino acids arginine, glycine, and methionine by the liver, pancreas, and kidney, respectively. Creatine is actively transported to skeletal muscle, where 95% of the body's creatine pool resides. The majority of muscle creatine exists as phosphocreatine. Endogenously, 1 to 2 g of creatine are synthesized daily and an additional 1 to 2 g are ingested in the diet. Excess creatine is metabolized to creatinine and excreted in the urine. Inter-individual differences in ambient muscle phosphocreatine levels and creatine transport capabilities influence if and how a particular individual will respond to creatine supplementation.

The interest in creatine as an ergogenic is based upon the following biochemical equilibrium:

Phosphocreatine + Adenosine diphosphate (ADP) Adenosine triphosphate (ATP) + Cr

Theoretically, flooding the system with creatine will drive this reaction to the left, thus increasing phosphocreatine stores and short-term energy supplies for exercise.

Many ergogenic claims have been associated with creatine use, including increased short-term energy during intense exercise, increased muscular mass, increased muscular strength, and increased lean body mass. The scientific evidence surrounding the effectiveness of creatine, however, is debatable. It is generally accepted that creatine is most useful as an ergogenic to increase energy availability for repeated bouts of short-term, high-intensity exercise.[10] Additionally, when combined with an appropriate resistance training program, creatine supplementation increases the maximal amount of weight that young men are able to lift (Level of Evidence A, systematic review).[11] Claims of decreased muscle lactate accumulation and increased muscle mass are unsubstantiated. Additionally, most of the work with creatine has been done in the laboratory. How this equates to performance on the athletic field is not known. Finally, creatine has been potentially associated with abnormalities in renal function.[12] Therefore, athletes with underlying renal disease should be counseled to avoid creatine supplementation.

Androstenedione

Androstenedione is a steroid precursor to testosterone. By itself, it is weakly androgenic. The ergogenic effects of androstenedione are due to its conversion to testosterone. It is metabolized to either testosterone or estrone. The popularity of androstenedione as a PES soared after Major League Baseball's homerun derby of 1998. Slugger Mark

McGwire admitted to oral supplementation with androstenedione, and sales skyrocketed thereafter. Some of the reported claims associated with androstenedione include an increase in testosterone levels, increased energy levels, enhanced recovery from exercise, and an increase in muscle mass and strength.

The original scientific evidence supporting an ergogenic effect for androstenedione comes from a very small study of 2 women in 1962 (Level of Evidence C).[13] In this study, supplementation with 100 mg of androstenedione per day led to a 4- to 7-fold increase in circulating testosterone. Other studies, however, have been less convincing. The largest clinical trial comparing androstenedione to placebo showed no change in free or total testosterone in young males. Supplementation with 300 mg/day did not lead to superior gains in strength when compared with a regular strength training program (Level of Evidence B).[14] Other studies have shown that higher doses of androstenedione can raise serum testosterone, but it is accompanied by a predictable rise in serum estradiol levels as well (Level of Evidence B).[15,16] Despite the scientific evidence to the contrary, anecdotal support for androstenedione use remains high, and clinicians should be prepared to deal with questions regarding its use. Conversion of androstenedione to estrone is normal, and users should be made aware of potential side effects including gynecomastia. Other androgenic side effects include male pattern baldness, testicular atrophy, and (for females) hirsutism and clitoromegaly. Androstenedione has been banned by the International Olympic Committee (IOC), the National Collegiate Athletic Association (NCAA), the National Basketball Association (NBA), and the National Football League (NFL).

Ephedrine

Ephedra alkaloids (pseudoephedrine, ephedrine, phenylephrine, tetrahydrozoline, and phenylpropanolamine) are sympathomimetic vasoconstrictor agents and, therefore, substances banned by the IOC and NCAA. Ephedrine stimulates the release of norepinephrine, resulting in an enhanced sympathetic response, including increased muscle contractility and enhanced cardiac output. Norepinephrine also increases metabolic rate and fat mobilization. Ephedra alkaloids, therefore, have been primarily promoted as medicinal alternatives to dieting. Ephedrine analogs are found in commonly used over-the-counter medications such as decongestants, antihistamines, and weight-loss products such as ma huang. Ephedrine has been relatively well studied, particularly in combination with caffeine. As with other PESs, the scientific data are mixed. Ephedrine supplementation has been shown to increase performance in both the laboratory[17] and field[18] setting (Level of Evidence B). Other studies show no benefit from ephedrine supplementation (Level of Evidence B).[19,20] Due to repeated reports of adverse outcomes, the FDA has proposed to limit ephedrine supplements to a total daily intake of 24 mg or more of ephedrine or less and to restrict use to 7 days or less. Most major sports governing bodies have banned ephedra products.

PRESCRIPTION MEDICATIONS

Anabolic–Androgenic Steroids

Anabolic–androgenic steroids (AASs) have both anabolic (protein-sparing, tissue-building) and androgenic (masculinizing) effects. Testosterone is the primary AAS. First isolated in 1935, testosterone use for improving athletic performance has been reported as early as the 1940s.[21] Early scientific studies on the effects of AAS were equivocal; therefore, the scientific community initially claimed that steroids were ineffective as performance-enhancing agents. Experienced athletes, however, insisted that AASs were effective in promoting muscle strength and muscle mass and continued to promote their underground use within the competitive athletic culture. This created a credibility gap between the scientific community and the athletic community that persists to the present day. Athletes are often mistrustful of physicians when seeking advice about performance-enhancing products, and many get their information either from other athletes or via the Internet.

At supraphysiologic doses, AASs exert their effects by acting on intracellular receptors to promote protein synthesis. In addition, AASs have an anticatabolic effect, allowing athletes to maintain a positive nitrogen balance during periods of heavy training. AAS are available in oral, injectable, and transdermal forms. Legitimate medical uses include testosterone replacement for congenital hypogonadism and treatment of refractory anemia. Typical medical replacement doses for hypogonadism are from 6 to 10 milligrams per day.

Many anabolic steroids, however, are diverted for illicit use. Users typically take anabolic steroids in 4- to 12-week cycles in an effort to minimize side effects. Anabolic steroid abusers use up to 50 to 100 times the physiologic dose during a cycle. *Stacking* refers to the process of combining

TABLE 12.3
Common Androgenic–Anabolic Steroids

Generic Name	Trade Name
Methandrostenolone	Dianabol®
Nandrolene decanoate	Decadurabolin
Stanozol	Winstrol®
Methyltestosterone	Testred®
Testosterone propionate	Testex®
Testosterone cypionate	Duratest®
Nandrolone phenpropionate	Durabolin®

oral and intramuscular steroids. *Pyramiding* refers to the practice of varying the AAS dose by starting with high doses and tapering toward the end of the cycle. Table 12.3 is a list of common androgenic–anabolic steroids.

The side effects of androgenic–anabolic steroids have been well described.[22] In young athletes, steroid use may accelerate the growth process with resultant premature closure of the epiphyses. Increased rates of tendon injuries have been reported among steroid users, as have complications involving the endocrine, reproductive, hepatic, and cardiovascular systems. Table 12.4 presents some of the more commonly reported adverse effects associated with anabolic–androgenic steroid use. Additionally, AASs have been reported as gateway drugs, because steroid users are more likely to use other illicit recreational drugs, including intravenous drugs, than are nonusers.[2] All anabolic steroids are banned by major sports governing authorities. A 6:1 ratio of testosterone to epitestosterone is considered an "offense" at Olympic events.

The quest for athletes to stay ahead of the doping process continues to evolve. Recently, a designer steroid, tetrahydrogestinone (THG), has been uncovered. While previously undetectable, new technology has evolved and testing of banked samples has implicated many high profile athletes, several of whom have already been banned from competition.

Human Growth Hormone

Human growth hormone (HGH) is a polypeptide hormone that is secreted by the anterior lobe of the pituitary. HGH increases translation of messenger ribonucleic acid to increase protein synthesis and promote lipolysis. HGH is used as a performance-enhancing substance to increase muscle mass and decrease body fat. For patients with endogenous deficiency, HGH is given in a therapeutic dose of 0.06 mg/kg 3 times a week. Athletes, however, have been reported to use 10 to 30 times this amount at a cost of up to $5000 per month.[2] A systematic review of the medical literature indicates that there is no evidence that HGH use promoted muscle strength in previously trained athletes (Level of Evidence A).[23]

TABLE 12.4
Side Effects of Anabolic Androgenic Steroids

System	Side Effect
Endocrine–reproductive	Testicular atrophy
	Irregular spermatogenesis
	Gynecomastia
	Menstrual irregularities
	Male pattern baldness
	Clitoromegaly
	Hirsutism
Cardiovascular	Altered lipoprotein levels (decreased HDL, increased LDL)
	Hypertension
	Left ventricular hypertrophy
Hepatic	Cholestasis
	Hepatic tumors (rare)
Musculoskeletal	Collagen dysplasia (decreased tensile strength with increased risk for musculoskeletal strain injury)
Psychological	Increased aggression
	Altered libido
	Affective lability
	Dependence

ILLICIT DRUGS

Marijuana

Marijuana has been used for centuries for both medicinal and mind-altering purposes. Marijuana is the most frequently used illicit drug in the United States; 40 to 45 million Americans have tried marijuana, and 15 to 20 million are regular users. Among athletes, 36% have used marijuana, 3% on a daily basis.[24] The principal active ingredient of marijuana is delta-9-tetrahydrocannabinol (delta-9-THC). THC primarily affects the central nervous system via diverse neurochemical pathways, including increased catecholamine synthesis,

decrease in hippocampal acetylcholine synthesis and release, and an alteration of dopamine, gamma-aminobutyric acid (GABA), serotonin, and norepinephrine uptake at the synaptic level of the cortex and striatum. Resulting acute behavioral changes include decreased short-term memory, vigilance, concentration, and work and psychomotor performance; euphoria; excitement; dissociation of ideas; anxiety; relaxation; and distortion of time and visual perception. Long-term usage leads to tolerance, psychological dependence, and, in some cases, physical dependence.

Marijuana produces effects that begin within several minutes of inhalation, peak within 30 minutes, and last several hours. Oral ingestion delays these effects severalfold. The terminal half-life of delta-9-THC is about 20 hours, but the half-life of its metabolites may be 50 hours or more. Due to high lipid solubility, 20% of these metabolites remain in the body for 1 week, making detection through urine drug screening possible for up to several weeks after use.

Marijuana use is deleterious to athletic performance, as it leads to a decrease in tracking ability, motor coordination, perceptual accuracy, reaction time, muscle strength, and maximal exercise capacity. Heart rate, metabolic rate, supine systolic blood pressure, and serum carboxyhemoglobin levels are increased. Decreased sweating ability can lead to an increased core body temperature. In susceptible individuals, chronic usage leads to the amotivational syndrome — apathy, impaired judgment, loss of ambition, and inability to carry out long-term plans.

Cocaine

Cocaine is a crystalline alkaloid that is obtained from the leaf of the coca plant. The only approved medical use for cocaine is as a topical anesthetic in combination with epinephrine. It is currently estimated that 30% of adults have tried cocaine, 5 million are regular users, and 2 million are addicted. Of high school athletes, 16 to 18% have used cocaine at least once. Additionally, 2 to 3% of Olympic-caliber female athletes report using cocaine before or during competition.[24] Cocaine's primary mechanism of action is a synaptic blockade that prevents reuptake of dopamine and norepinephrine. This results in characteristic feelings of euphoria, decreased fatigue, mood elevation, enhanced vigor, and psychic arousal. Cocaine's effects depend on route of administration. The peak effect achieved by snorting is achieved within 3 to 5 minutes. By comparison, intense effects begin 30 seconds after intravenous injection. Inhalation of vaporized cocaine base (crack), also known as free basing, produces an even more intense rush of sensations within seconds. The half-life of cocaine is 2 to 6 hours, and cocaine metabolites can be detected in the urine for 1 to 3 days by reliable screening tests. Cocaine use is banned by most sporting authorities.

MISCELLANEOUS AGENTS

Beta-hydroxy-beta-methylbutyrate (HMB), a branched-chain amino acid metabolite of leucine, decreases proteolysis and increases protein synthesis. Various studies have shown an ergogenic effect at doses of up to 6 g/day for 8 weeks without side effect. Chromium picolinate, a trace mineral required for carbohydrate and lipid metabolism, potentiates the effects of insulin, resulting in muscle growth and increased intracellular availability of glucose. While it may increase lean body mass, the general scientific community is in agreement that at levels above 50 to 200 μg per day no ergogenic benefits are gained. Glutamine is a nonessential amino acid affecting many of the regulatory functions of the body. Although adequate research does not exist in healthy men and women, recent data suggest that during recovery oral glutamine promotes storage of muscle glycogen. High doses (>5 g) are required for anticatabolism and enhanced protein synthesis. Conjugated linoleic acid, a natural substance in foods high in saturated fat such as meat and dairy products, has shown conflicting results or no benefit regarding weight loss or strength gains in a few, incomplete human studies.

The use of amino acid supplements in healthy human subjects does not provide convincing evidence with respect to improved training responsiveness or exercise performance. Guarana is a stimulant produced from Brazilian herb that contains multiple substances, the most significant of which is caffeine (2.5 to 5% by weight). Khat is an herbal product derived from *Catha edulis* that contains cathine and cathinone, which are amphetamine-like stimulants. Products containing these chemicals are banned substances at the elite level of competition. Plasma expanders such as heta starch are most commonly seen in athletes competing in endurance events and are banned substances at the elite levels of competition. Propanolol and other beta blockers are banned from use in such sports as archery, biathalon, and shooting where fine motor tremulousness would be detrimental to performance because these drugs lower the heart rate. Beta agonists such as

TABLE 12.5
Summary of Common Performance-Enhancing Products Banned by the International Olympic Committee (IOC)

Stimulants	Narcotics	Anabolic Agents	Diuretics	Peptide Hormones	Miscellaneous
Amphetamines	Heroin	Anabolic steroids	Furosemide	Corticotropins	Beta blockers
Caffeine	Methadone	Clenbuterol	Mannitol	Growth hormone	Beta agonists
Cocaine	Morphine	Salbutamol	Spironolactone	Insulin-like growth factor	
Ephedrines				Insulin	
				Erythropoietin	

albuterol, both oral and parenteral preparations, are prohibited. Inhaled forms of these products are restricted, and athletes who use these products to treat reactive airway disease are required to notify, in advance, their sports governing body. At the Olympic level, the diagnosis of asthma must be made or confirmed by a pulmonologist/allergist or team physician.

Alcohol is an ergogenic agent of limited use because it has adverse effects on muscle coordination, mental alertness, and muscle endurance due to dehydrating effects. It is a banned substance in sports where a reduction in heart rate and fine motor tremulousness is a competitive advantage. Sodium bicarbonate supplementation in the hope of reducing lactic acidosis during competition is not banned as it is not considered an ergogenic agent. Tribulus is an oriental plant used by young athletes in the hope of increasing endogenous testosterone production. However, it has not been shown to enhance performance in competitive scenarios and is not banned.

Because diuretics have been used by athletes to mask the use of other illicit ergogenic agents in drug-testing scenarios, to reduce the water retention side effects of other ergogenic drugs, and to reduce body weight in weight-category-dependent competition, they are banned. Choline, an amino acid, has been shown to increase endurance in swimmers. Coenzyme Q-10 (CoQ10) and the oryzanols have equivocal effects on athletic performance and growth hormone secretion/antioxidation, respectively. Use of pituitary or synthetic gonadotropin (LH) and chorionic gonadotropin (hCG) hormones is considered an offense if detected at Olympic and qualifying events. Greater than 1000 ng/mL in the urine is considered an offense at the Olympic and qualifying events for $beta_2$ agonists such as bambuterol, clenbuterol, fenotorol, formoterol, reproterol, salbutamol, salmeterol, and terbuterol. A urine concentration of greater than 5 μ g/mL for caffeine is considered an "offense" at the Olympic level.

DRUG TESTING

As long as products that enhance performance are available, be athletes will use them, regardless of the consequences. Thus, the game of cat and mouse between screening authorities and competitors continues. Unscrupulous athletes and their handlers consistently search for new ways to beat the system. The screening of athletes for drug use and misuse was adopted by the IOC in 1965 and formally introduced during the 1968 Olympics. The first comprehensive drug testing was undertaken at the 1972 Summer Olympics in Munich. In 1986, the NCAA followed suit and adopted mandatory drug testing for athletes. Many other amateur and professional sports organizations such as the U.S. Powerlifting Federation, U.S. Tennis Association, Major League Baseball, National Basketball Association, Professional Golf Association, National Football League, World Boxing Council also have formal drug-testing policies modeled after the IOC. The policy statements of these governing organizations provide a list of banned drugs (Table 12.5) as well as a procedural testing protocol that describes who should be tested, when they should be tested and how they should be tested. Sanctions and punishments for a positive testing are also described.

Athletic drug screening may be formally announced, random (unannounced), mandatory, or based on suspicion. In 1984, less than 1% of drug tests in the Olympics and 2.5% in the NCAA were positive when based on formal, announced screening.[25] Random unannounced testing, therefore, is felt to more accurately assesses the true prevalence of drug use and has been shown to act as a better deterrent to drug use. Bromantan, epitestosterone, probenicid, and all diuretics are banned at qualifying and Olympic events because they are masking agents.

SUMMARY

The use of performance-enhancing substances is quite common. Clinicians caring for competitive and recreational athletes should be familiar with the most common products that are currently in vogue. It is important to ask whether the products are safe, whether they are effective, whether they are legal, and whether their use is ethical. Armed with this information, athletes can make informed decisions about the use of performance-enhancing products.

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13 PROTECTIVE ATHLETIC EQUIPMENT

James L. Lord

INTRODUCTION

As the number of participants in sports-related activities has grown exponentially over the past 10 to 20 years, serious athletic injury numbers have risen concomitantly. Team and individual contact and collision sports account for a large proportion of the injuries,[1–2] but recreational and fitness-related activities are now adding to the injury statistics at an ever-increasing rate. National and international governing organizations for various sports have authorized rule changes mandating the use of various types of protective equipment depending upon the demands of their individual sport. In addition, standard specifications for various types of equipment (e.g., football helmets) are currently a work in progress. Professional organizations such as the American Academy of Pediatrics (AAP) and the American Academy of Orthopedic Surgeons (AAOS) have published recommendations for the use of protective equipment and have sponsored public service advertisement campaigns for athletes, parents, coaches, athletic trainers, and physicians. In this rapidly expanding information explosion, it is imperative for the sports physician to remain current with evolving standards. This chapter primarily emphasizes athletic equipment designed to protect against serious brain injury and mouth, face, and eye injuries, and it will discuss some of the safety issues in recreational and fitness activities as they relate to protective equipment. It will conclude with some information on the use of prophylactic knee braces in football.

GENERAL PROTECTIVE EQUIPMENT

Helmets

Concussive injuries to the head remain the greatest threat to the life and well-being of the athlete.[3–4] Football, baseball, ice hockey, and lacrosse helmets are now mandated at all levels of participation. Standard specifications continue to evolve for all helmets. The National Committee on Standards for Athletic Equipment (NOCSAE), the American Society for Testing and Materials (ASTM), the American National Standards Institute (ANSI), and the Snell Memorial Foundation have all developed criteria for the performance of helmets when dropped from a specified height onto a metal anvil in order to simulate the concussive force experienced by an athlete in competition. None of these groups has been able to completely duplicate real-life impact encounters that may represent not only linear but also rotational forces. One study of North Carolina high school football players favored helmets with suspension support over helmets with padding alone or a padding-suspension combination.[5–6] (Level of Evidence B, epidemiologic study). Newer football helmets offer protection with a cushion of air in

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two or more separate bladders suspended within the helmet. The air-padded helmets, in addition to decreasing the force of impact, also add the ability to easily custom fit the helmet to a variety of head sizes. The latest addition to many helmets is a rigid mandibular extension device designed to protect the angle of the mandible. Data are still sparse with regard to its effectiveness in reducing brain injury, as direct impact to the device may transmit concussive energy to the brain.

Helmets also provide some secondary protective functions. Football helmets interconnect with shoulder pads and other neck protection devices such as "cowboy" collars to prevent excessive neck motion;[7–8] therefore, proper fitting of equipment is very important. The sports physician, while not likely to be the person who distributes and fits the equipment, can still exercise a great deal of influence by recognizing that particular injuries could be lessened or avoided altogether with properly fitting equipment. Baseball helmets, in addition to protecting against impact from balls, bats, and other players, must also provide some facial protection. Lacrosse and ice hockey helmets, which are lighter in weight than football helmets, are purposely designed to provide for a greater degree of head and neck movement while still meeting maximum impact standards. All helmets should display some sort of label indicating certification by either NOCSAE or ASTM.

Data for the use of protective headgear in soccer and rugby players is still inconclusive[9-11]. One of the conclusions from the 2002 Institute of Medicine Report was that there was no available evidence to support the mandatory use of helmets in soccer players (Level of Evidence C, consensus expert opinion). And one pilot study from the University of New South Wales in Sydney, Australia concluded that current rugby headgear does not provide significant protection against concussion in rugby union football at a junior level (Level of Evidence B, cohort study).

Data for bicycle helmets strongly support the fact that they provide substantial protection from serious brain injuries for cyclists of all ages, including those involved in crashes with motor vehicles. One representative prospective case-controlled study concluded that bicycle helmets provided significant protection from all head injury categories, ranging from mild abrasions to intracranial hemorrhage and cerebral lacerations without regard to helmet type (i.e., hard shell, thin shell, or no shell) (Level of Evidence B, case-control study).[12] A meta-analysis of bicycle helmet efficacy further supports this conclusion (Level of Evidence A, meta-analysis).[13] As a result of these findings and others,[14–15] the U.S. Consumer Product Safety Commission (CPSC) mandated that all helmets manufactured or imported for sale in the United States after March 1999 must be able to withstand a drop from a 2-meter height onto a metal anvil. Helmets that comply with this standard display a CPSC approval sticker on the inside liner. Helmets manufactured or imported before 1999 may be used as long as they have been certified by ASTM and/or the Snell Memorial Foundation. If only ANSI certification is available, the helmet should be discarded because their drop test was below the 2-meter standard. If a multisport helmet is promoted (even by implication) for cycling use, it must also be certified to comply with the CPSC standards for bicycle helmets.[16] To further strengthen support for mandatory bicycle helmet use, the American Academy of Pediatrics issued its recommendations for helmet use (Table 13.1), and the AAP has further issued its recommendations for physician advocacy for universal bicycle helmet use (Table 13.2). Despite these recommendations, the barriers to bicycle helmet use persist.[17] Excuses for not using a helmet range from "they're too hot" to "they're ugly" or "they mess up my hair" (Table 13.3).

Despite the nearly 6-fold growth in inline skating and the increased popularity among U.S. adolescents of skate-boarding and scootering, a relative paucity of formal data exists regarding the frequency and severity of injuries and the proven

TABLE 13.1
American Academy of Pediatrics Recommendations for Helmet Use

Properly fitted bicycle or multisport helmets for cyclists each time they ride
CPSC certification (March 1999 standards) for all helmets intended for bicycle use[a]
Replacement of any damaged or outgrown helmet
Replacement of all helmets at least every 5 years or sooner if recommended by manufacturer
Appropriately sized helmets for young children who ride as passengers[b] either in a bicycle-mounted child seat or in a bicycle-towed trailer
Prohibit purchase of helmets from yard sales
Mastery of bicycle safety issues by parents and children

[a] Helmets manufactured before March 1999 may be used if they meet either Snell Memorial Foundation or ASTM standards but not ANSI.

[b] Passengers should be old enough to have sufficient muscle strength to control head movement during a sudden stop (at least 1 year old).

TABLE 13.2
American Academy of Pediatrics Physician Advocacy Recommendations for Helmet Use

Encourage parents and other child-care providers to require helmet use in children when they first begin to ride tricycles or other wheeled vehicles or encourage parents to wear helmets when bicycling.[a]
Serve as community and legislative advocates for mandatory bicycle helmet use.[b]
Encourage school districts to make helmets mandatory when children ride to and from school and during school-related bicycle rides.
Help develop programs to promote bicycle safety training that emphasizes helmet use.
Support campaigns to encourage retail sales companies to carry affordable helmets and include them with the purchase of every new bicycle.
Encourage and support organizations that promote helmet use with well-crafted advertisement campaigns.
Urge the popular media to feature celebrity bicyclists wearing a helmet.

[a] Parents and children should also be warned of the dangers of riding without a helmet.

[b] This includes mandating bicycle rental agencies to include helmets as part of the rental contract.

TABLE 13.3
Most Frequent Barriers to Bicycle Helmet Use

Children
It's uncomfortable.
I don't need it.
It's hot.
I don't own one.
It messes up my hair.
It doesn't fit.
It's too difficult to put on.
I forgot it.
I outgrew it.
It's funny looking.
Adolescents
It's annoying.
It's uncomfortable.
I don't own one.
It's ugly.
It's hot.
It's unfashionable.
It's funny looking.
I forgot it.
Friends tease me when I wear it.
It messes up my hair.
Adults
It's hot.
It's annoying.
It's uncomfortable.
I forgot it.
I'm not at risk for injury.
It messes up my hair.
It's unfashionable.
It's ugly.

effectiveness of helmets.[18–19] In one study of only 841 children, the rate of overall helmet use for skating activities was 13.6%; 18.2% of inline skaters and 14.3% of skateboarders wore helmets, but only 11.5% of scooter riders wore them (Level of Evidence B, observational study).[20] Interestingly, most children who wore helmets wore them incorrectly, usually tilted back onto the occiput. Helmet use by children was positively associated with helmet use by accompanying children and/or adults. One other small study reported that 5% of children who participate in inline skating sustain head injuries and at least half of them require hospital admission. While the data for the protective effect against serious brain injury remain sparse for skaters and skate- and snowboarders, it is probably safe to extrapolate from the bicycle helmet studies to provide incentive for physicians to assume a safety advocacy role (Level of Evidence C, consensus opinion).

Facemasks

As a natural progression from the basic concept of protecting athletes from the effects of head injuries during collision and contact sports, eye and face protection has benefited from the development of face masks. Following the Canadian Amateur Hockey Association's (CAHA) institution of compulsory use of face masks in amateur hockey in 1978, a precipitous and persistent decrease has occurred in the number of eye injuries in hockey and the number of blind eyes (Level of Evidence B, epidemiologic study).[21] Several styles of face masks are currently available for ice hockey. Full-face masks (constructed of polycarbonate plastic, wire mesh, or a combination of the two) cover the entire face including the chin; half-visors cover only the eyes and extend down to just below the nose. Full-face masks have offered the best protection since their

inception and are the type currently mandated for minor league hockey in Canada. The face masks that are required must be certified by the Canadian Standards Association (CSA) and must display the proper certification label. Half-visors do offer some protection from direct impact from a puck or hockey stick but only when worn correctly with the visor down and the neck strap securely fastened.

For American football, the American Academy of Ophthalmology (AAO) and the American Academy of Pediatrics recommend a full-face polycarbonate shield or steel mesh face mask attached directly to the helmet.[22] For functionally one-eyed athletes, they also recommend wearing racquet sports eye protection under the helmet and facemask. In addition, these two groups recommend ASTM-approved full-face polycarbonate face masks for baseball and softball batting helmets. Baseball catchers should wear a full-face steel mesh mask whenever they are standing behind a batter, even when they are just warming up the pitcher. Men's lacrosse helmets are required to provide full-face protection in addition to laryngeal protection with a separate neck piece attached. Steel mesh material is currently the standard.

Mouthguards

Mandatory use of mouthguards, first instituted by the National Football Alliance (NFA) in 1962, has been associated with a significant reduction in the number of serious orofacial injuries. Other sports have since mandated this simple addition to the protective equipment armamentarium with similar results. Despite this and despite the fact that the majority of studies have found the mouthguard to be the most effective way of preventing orofacial injuries[23], it has continued to be underutilized in many sports. The American Association of Oral–Maxillary Surgeons (AAOMS) currently recommends the use of mouthguards in all collision or contact sports (Table 13.4). Currently, three types of mouthguards are available (Table 13.5). The stock or off-the-shelf model (made of either rubber or plastic) is the least expensive but also offers the least protection. Mouth-formed protectors are available in most sporting goods stores. They come as a kit that includes a firm outer mold and various heat-sensitive moldable substances, which are poured into the shell. The shell is then put into the athlete's mouth to create a custom fit. These provide a better fit and better cushioning than the stock variety. The ideal mouthguard, however, is the custom-made type constructed from a plaster model of the individual's teeth provided by a dentist or oral surgeon. Wearer comfort plays a major role in the use of any mouthguard. If a mouthguard is too thick, not only is it uncomfortable to wear but it also transmits unacceptable impact energy to other critical structures, such as the brain. The most commonly used mouthguard material is ethylene vinyl acetate (EVA), which, at a thickness of 4 mm, has been shown to provide optimum protection while providing the best energy absorption and reduction of transmitted forces (Level of Evidence B, epidemiologic study).[24] The critical issue is educating physicians and the public about the benefits of mouthguard use.

TABLE 13.4
Sports for which the American Association of Oral–Maxillary Surgeons Recommends Mouthguard Use

Football
Baseball
Ice hockey
Wrestling
Boxing
Martial arts
Lacrosse
Field hockey
Street hockey
Soccer

TABLE 13.5
Types of Mouthguards

Rubber or plastic prefabricated stock protectors
Mouth-formed protectors
Custom-made plaster-molded mouthguards

SPORT-SPECIFIC PROTECTIVE EQUIPMENT

See Table 13.6.

Protective Eyewear for Racquet Sports

Racquetball, squash, and handball are responsible for up to 8% of sport-related eye injuries.[25] Badminton accounts for 3.6% and tennis for approximately 1.3%. In tennis, most eye injuries occur in doubles, especially in older players, but the approximately 2-oz compressible tennis ball can

TABLE 13.6
Recommended Protective Equipment for Select Sports

	Football	Hockey	Baseball	Cycling	Soccer
Helmet	+	+	+	+	
Headgear					
Faceguard	+	+			
Eyeguard					
Mouthguard	+	+			+[a]
Throatguard		+	+		
Shoulder pads	+	+			
Elbow pads		+			
Gloves	+	+		+	
Chest protector	+	+	+		
Hip pads	+	+			
Athletic supporter/cup	+	+	+		+
Thigh guards	+	+			
Shinguards		+	+		+
Footwear	+	+	+	+	+

[a] Recommended for goalkeepers.

achieve speeds of over 100 mph, which even a singles player may not be able to avoid. One study of eye injuries reported that 7 of 10 patients with eye injuries from tennis doubles sustained a retinal detachment requiring retinal surgery, laser surgery, or both (Level of Evidence B, retrospective study).[26] The 1-oz shuttlecock, which can achieve a speed of 160 mph, fits perfectly into the human orbit. Up to 50% of all badminton injuries seen by ophthalmologists involved some permanent loss of best-corrected vision.[26] Multiple studies have shown that open eyeguards (lensless goggles), street-wear prescription lenses, and even hardened industrial safety glasses do not prevent eye injury. The current standard set by the Protective Eyewear Certification Council (PECC) requires polycarbonate protectors that prevent eye contact from squash or racquetballs at speeds of 90 mph with impact from either the front or side. These lenses must also meet predetermined standards of clarity and field of vision.

Safety Equipment for Skaters, Scooterers, and Snowboarders

In addition to the head and serious brain injuries referenced earlier, wrist and distal forearm fractures represent a substantial proportion of injuries to inline skaters. The numbers are similar for skateboarding, but skateboarding injuries tend to be more severe. The *American Journal of Sports Medicine* reported a 1999 study of fresh-frozen cadaver forearms that found that wrist guards reduce both volar and dorsal distal radial bone strain and therefore protect the wrist during low-energy falls (Level of Evidence B, epidemiological and biomechanical study).[27] They function partly by sharing the load and partly by absorbing the energy impact. Although no universal standard yet exists that recommends acceptable specifications, preliminary data nevertheless support the recommendation to use wrist guards for all skating activities. Elbows and knees are also vulnerable to skate and scooter injuries.[28] Data on the effectiveness of appropriate protective equipment are even more scarce, but most clinicians are currently recommending the use of elbow and knee pads on a purely empiric basis (Level of Evidence C, consensus expert opinion). Thus far, the only evidence has been provided by small observational studies.

Martial Arts

The martial arts represent a diverse group of activities initially developed primarily for self-defense. Current participants engage in a variety of martial art styles for recreation, exercise, and sport. Several forms of the martial arts have introduced protective equipment unique to their requirements. Full-contact karate utilizes foam rubber protective headgear, body pads, and hand

and foot pads mainly during competitive sparring and sport karate.[29] Kendo participants wear elaborate equipment to protect against blows from bamboo swords. While most injuries occur in tournament settings,[30] the risk of serious injury in the martial arts is low.[31] Currently, no universal safety standards exist for protective equipment in the martial arts, but it is still highly recommended for beginners and novices during sparring sessions and for use in tournaments (Level of Evidence C, consensus expert opinion). Face and headgear and body, hand, forearm, and foot protection are available as various types of padded devices constructed from various combinations of foam rubber, leather, and lightweight plastic. The choice of equipment required is left up to tournament directors and studio owners.

Soccer

The Fédération Internationale de Football Association (FIFA) currently requires the use of shin guards as the only piece of protective equipment for soccer players. The only regulations for shin guards are that they be commercially manufactured specifically for soccer and that they be worn under a long sock or stocking, but shin guards that have padding over the ankle and the Achilles tendon area do help prevent injuries from direct blows. In addition, the AAOMS strongly recommends the use of mouthguards in all players.

Prophylactic Knee Braces in Football Players

The American Association of Orthopedic Surgery defines four categories of knee braces: (1) prophylactic braces intended to prevent or reduce serious knee injuries in contact and collision sports, (2) functional braces designed to provide stability for unstable knees after injury, (3) rehabilitative braces to allow protected but controlled motion after surgery or during active rehabilitation of injured knees, and (4) patellofemoral braces to improve tracking of the patella and relieve anterior knee pain. The prophylactic knee brace was first introduced into the National Football League (NFL) in an attempt to offer protection for the medial collateral ligament (MCL) during valgus stresses to the knee and to support the cruciate ligaments during rotational stress. These braces have either bilateral or unilateral bars with straps for attachment to the leg. Some are single hinged, some are double hinged. They are generally designed to absorb some of the impact of physical contact and decrease the force applied to the MCL by 10 to 30%. Players who were thought to benefit from such braces are the interior front linemen. Multiple studies have yielded insufficient evidence to recommend prophylactic knee bracing, especially in the young athlete (Level of Evidence B, epidemiologic studies).[31–32] In a position paper issue by the AAOS in 1997, the routine use of prophylactic knee braces was discouraged.[33] The main reason cited was the lack of data to support their use. Some studies have even suggested that such braces may be contributing to knee injuries (Level of Evidence B, epidemiologic studies).

SUMMARY

Protective equipment standards continue to evolve as equipment technology advances and as more and more data accumulate supporting the effectiveness of the safety equipment available. A variety of professional organizations have made recommendations for the use of protective equipment, but, unless mandatory use of at least helmets for high-risk sports activities is forthcoming, serious preventable injuries will continue to present themselves to primary care and specialty providers.

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14 PHYSICAL ACTIVITY COUNSELING AND EXERCISE PRESCRIPTION*

Mark B. Stephens

INTRODUCTION

Physical inactivity is one of the leading predictors of mortality in the United States (Level of Evidence A, quantitative systematic review).[1] Two of every three adults do not engage in regular physical activity, and one in four is completely sedentary.[2] As a result of this pandemic of physical inactivity, two of three American adults and one of six American children are currently classified as being either overweight or obese.[3] The rising trend of physical inactivity is so significant that this area has been designated as one of the top two health indicators for *Healthy People 2010*,[4] a national initiative designed to promote healthy lifestyle choices in the general public.

Primary care physicians are familiar with the sequelae of physical inactivity. Diabetes,[4] coronary artery disease,[5] hyperlipidemia,[6] hypertension,[7] and certain cancers[8] have all been associated with physical inactivity (Level of Evidence B, large cohort studies). Age, social class, and previous experience also impact an individual's attitude, motivation, and response to physical activity.[9] These are important factors to consider when designing an individual exercise prescription. A comprehensive exercise prescription also includes consideration of other health behaviors such as tobacco use and dietary habits. Physical activity counseling is a brief preventive intervention within the office. The U.S. Preventive Health Task Force currently states that the evidence is

* The opinions contained herein are those of the authors. They are not official policy of the Uniformed Services University, the Department of the Navy, the Department of the Army, or the Department of Defense.

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TABLE 14.1
Overcoming Activity Barriers

Roadblock	Strategy for Overcoming Roadblock
I am usually too tired to exercise.	Regular activity actually improves your energy level.
The weather is too bad.	Many activities can be done in the comfort of your home, no matter what the weather.
I can't afford a health club	You do not need a health club to be physically active; walk around the mall, use public facilities, find others to join you in an activity group.
Exercise is boring.	Try listening to music or exercising with a friend or family member.
I get sore when I exercise.	Slight muscle soreness after physical activity is common when just starting out; limit this by starting slowly, building up gradually, and stretching before and after each activity.
I do not enjoy exercise.	Don't "exercise." Try becoming physically active through any hobby or enjoyable activity that gets you moving.
I'm afraid of getting hurt.	Make sure you wear proper clothing and footwear; do not exercise on uneven terrain or push yourself beyond your limits.
I'm not any good at exercise.	You do not have to be good at exercise to walk. Start low and go slow; as you develop confidence and establish a pattern of activity, try new things.
I do not have time.	Thirty minutes a day is equivalent to a half-hour television show; can you give up one show per day?

Source: Adapted from Patrick, K., Sallis, J.F., Long, B., Calfas, K.J. et al., *Sportmed.*, 22, 45–55, 1994.

insufficient to recommend for or against routine behavioral counseling within primary care settings to promote physical activity.[10] An exercise prescription, on the other hand, refers to a formal, written document based on history, physical examination, clinical exercise testing, and specific patient goals. The exercise prescription is designed to provide patients with specific information regarding the frequency, intensity, type, and duration of physical activity with specific objectives in mind.

HISTORY

A thorough and accurate clinical history is one of three cornerstones upon which a formal exercise prescription is based. In addition to the patient's medical history, there are multiple social, cultural, familial, environmental, and personal factors that must be taken into consideration when developing an individualized exercise prescription. For instance, it is normal for adults to become less active as they age. Adults also often perceive themselves as being too busy to exercise (Table 14.1).[11] Women are less likely to exercise than men. Minority populations, individuals from low-income families, and adults with physical disabilities are less likely to engage in regular physical activity (Level of Evidence B, cohort studies).[12] Adults who watch more than 4 hours of television a day are more than four times as likely to be obese.[13] Adults who spend significant amounts of time using a computer are also less physically active and more often obese.[14] Each of these factors provides an important contextual framework upon which the exercise prescription is based.

Medical History

A thorough medical history is an essential element of the exercise prescription. Patients should document all medical diagnoses that might impact on their ability to exercise. Specifically, it is important to ask about and document a prior history of coronary artery disease, hypertension, diabetes, hyperlipidemia, peripheral vascular disease, asthma, chronic obstructive pulmonary disease, cancer, or musculoskeletal disease. Patients should be asked whether or not they have any known abnormalities on physical examination. If any suspicious history is documented, further evaluation is particularly indicated prior to initiating a new program of regular physical activity.

Activity History

It is important to determine the patient's experiential base of exercise and physical activity. Has it been with formal programs and sports or recreational or occupational in nature? It is also important to ask about a prior history of musculoskeletal injury, which can target recommendations for

injury prevention. Patients should also be asked to describe their current pattern of exercise. Specifically, it is important to determine whether or not patients obtain at least 30 minutes of moderate physical activity on a daily basis. Patients should describe physical activity related to their lifestyle or occupation as well. It is also important to ask patients to describe current television, computer, and video viewing habits. At the outset, patients should be asked what specific goals they have. Are they interested in losing weight, improving cardiovascular endurance, improving muscular strength, decreasing cardiovascular risk, or improving their overall health?

PHYSICAL EXAMINATION

The physical examination is the second cornerstone upon which formal exercise prescriptions are based. The physical examination should be conducted in a standard fashion. Blood pressure, height, and weight are recorded in the standard fashion. From this information, the body mass index (BMI) is easily calculated from the standard formula:

$$\text{BMI} = \text{weight (kg)}/\text{height (m)}^2$$

The body mass index is now well recognized as a standard means of expressing body composition. For adults, a body mass index of 20 to 24.9 is considered normal. An adult with a BMI between 25 and 29.9 is considered overweight. Individuals with a BMI of greater than 30 are considered to be obese.

When performing the general physical examination, paying particular attention to the cardiopulmonary, musculoskeletal, and neurological systems is important. The goal is to screen for conditions that would merit further evaluation prior to beginning an exercise program. Specifically, the cardiopulmonary exam should include documentation of pulses (rate and rhythm); auscultation for murmurs, rubs, gallops, and clicks; auscultation of lung fields; and peripheral pulses. The musculoskeletal examination should focus on the range of motion of all major joints as well as the presence of pain and/or swelling. The abdominal examination should focus on the presence of organomegaly, tenderness, bruits, or masses. An extremity examination should be performed to rule out edema. If areas of concern are identified, further evaluation is appropriate prior to initiating a new exercise program.

Who needs clinical exercise testing prior to beginning a program of physical activity? Clinical exercise testing is the third cornerstone upon which formal exercise prescription is based. An accurate decision regarding the need for clinical exercise testing requires information from the history and physical examination. Several useful clinical tools are also available to assist clinicians when confronted with the question of who needs clinical exercise testing prior to beginning a program of regular physical activity.

TABLE 14.2
Risk Stratification

Risk Category	Description
Low risk	Men under the age of 45 and women under the age of 55 who are asymptomatic and have a maximum of one clinical risk factor
Moderate risk	Men over the age of 45 and women over the age of 55 or individuals who have two or more clinical risk factors
High risk	Individuals who are either symptomatic[a] or who have known cardiopulmonary disease

[a] Symptoms include exertional chest pain, exertional dyspnea, exertional syncope, palpitations, and claudication.

Source: Adapted from American College of Sports Medicine, *ACSM's Guidelines for Exercise Testing and Prescription*, 6th ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2000.

One approach, endorsed by the American College of Sports Medicine (ACSM) is based on the concept of clinical risk stratification (Level of Evidence C, consensus opinion).[2] Using information from the patients' past medical history and physical examination, individuals are categorized into three categories: low, moderate, and high risk (Table 14.2). Patients who are low risk do not need a formal clinical exercise test before beginning a program of regular physical activity. For these patients, physical activity counseling is sufficient. Those who are moderate risk require clinical exercise testing only if they plan to only engage in activities that are vigorous in intensity. All high-risk patients should have clinical exercise testing performed before beginning a program of regular exercise (Table 14.3). The clinical exercise test provides important diagnostic and therapeutic information (e.g., maximum heart rate) directly pertinent to the exercise prescription.

PHYSICAL ACTIVITY GUIDELINES: A PRECURSOR TO EXERCISE PRESCRIPTION

The health benefits of physical activity are well recognized. Individuals who are physically active have lower rates of all-cause mortality,[15] improved

TABLE 14.3
Clinical Exercise Testing

Indications for Exercise Testing	Contraindications to Exercise Testing
Evaluation of patients with suspected coronary artery disease	Absolute contraindications
Typical angina pectoris	Recent myocardial infarction
Atypical angina pectoris	Unstable angina
Evaluation of patients with known coronary artery disease	Acute myocarditis/pericarditis
Following myocardial infarction	Acute systemic infection
Following therapeutic intervention	Symptomatic heart failure
Screening of healthy, asymptomatic patients	Symptomatic aortic stenosis
Individuals in high-risk occupations (pilots, firefighters, law enforcement officers, mass transit operators, etc.)	Relative contraindications
Men over age 40 and women over age 50 who are sedentary and plan to start a vigorous exercise program	Severe hypertension (uncontrolled)
Individuals who are identified as being-at risk based on multiple cardiac risk factors or concurrent chronic diseases	Persistent arrhythmias (poorly controlled)
Evaluation of exercise capacity in patients with valvular heart disease (excluding severe aortic stenosis)	Obstructive cardiomyopathy
Evaluation of patients with cardiac rhythm disorders	Uncontrolled diabetes or thyroid disease
Evaluation of exercise-induced arrhythmia and response to treatment	Systemic neuromuscular, musculoskeletal or rheumatologic disease that limits the patient's ability to exercise
Evaluation of rate-adaptive pacemaker setting	Heart block (second- or third-degree atrioventricular block)
	Stenotic valvular heart disease
	Undifferentiated electrolyte abnormalities (hypokalemia, hypomagnesemia, etc.)

Source: Adapted from American College of Sports Medicine, *ACSM's Guidelines for Exercise Testing and Prescription*, 6th ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2000, and Gibbons, R.J., Balady, G.J., and Beasley, J.W., *J. Am. Coll. Cardiol.*, 30, 260–311, 1997.

psychological well being,[16] lower rates of cardiovascular morbidity,[5] and a reduced risk of developing diabetes,[4] hypertension,[7] and certain cancers[8] (Level of Evidence B, cohort studies). Additionally, physical activity promotes bone, joint, and muscular health; enhances work performance capability; and is associated with improved self-esteem.

It is recommended for all Americans to engage in regular physical activity of moderate intensity for at least 30 minutes on most, preferably all, days of the week according to their individual abilities.[17] This guideline is important in that it suggests that the benefits of physical activity can be accumulated in relatively short bouts throughout the course of the day, as opposed to single daily "training" sessions. Repetitive bouts of activity, as short as 8 to 10 minutes totaling 20 to 60 minutes a day, have a cumulative effect and provide significant health benefit (Level of Evidence C, consensus statement).[18]

During the past decade, attention has moved away from exercise training toward health-related "lifestyle activities" such as stair climbing, brisk walking, housework, yard work, or other recreational activities. Health-related gains can be realized with mild-to-moderate levels of activity, particularly in previously sedentary individuals. Moderate-to-vigorous levels of physical activity, however, are still recommended for individuals who wish to achieve higher levels of cardiovascular conditioning. In short, any level of physical activity is beneficial for health, but more does appear to be better. These guidelines also recommend that activities promoting muscular strength and flexibility also be included to improve balance, strength, and coordination.[19]

Recognizing the importance of physical activity in combating chronic disease, the U.S. Department of Health and Human Services has made physical activity one of the leading health indicators by which the nation's health will be measured over the next decade. The specific goals of *Healthy People 2010* are to reduce the burden of chronic disease and to improve health, fitness, and quality of life by increasing daily physical

Name: Date:

Age: Sex:

Medications:

Type of physical activity:

Frequency:

Duration:

Intensity: Training heart range:

Maximum heart rate:

Special instructions:

Precautions:

Risk factors to work on:

Reevaluation date:

_____

(Doctor's signature)

_____

(Patient's signature)

_____

(Guardian's signature, if applicable)

Figure 14.1 Suggested exercise prescription form.

activity.[4] The following points can be summarized from current guidelines (Level of Evidence C, expert opinion):[3,17]

- All individuals should engage in at least 20 to 60 minutes of physical activity of moderate intensity on most, preferably all, days of the week.
- Additional health benefits can be achieved by increasing the time spent in moderate-intensity activity or by increasing the intensity of the activity.
- Activities that promote muscular strength (resistance training) should be performed at least twice a week.
- Previously inactive men over age 45, women over age 55, and people at high risk for coronary artery disease should consult a physician prior to beginning a new exercise program, especially if they plan to incorporate *vigorous* physical activity into their routine.

PUTTING THE GUIDELINES TO WORK: THE EXERCISE PRESCRIPTION

General physical activity guidelines provide a solid foundation for activity counseling within the context of an office visit. An exercise prescription is analogous to any other medical prescription.[19]

TABLE 14.4
Components of an Exercise Prescription: The "FITT" Principle

Frequency
Intensity
Type of activity
Resistance training
Cardiovascular training
Flexibility
Time (duration of activity)

It gives patients clear instructions regarding how to dose their exercise routine. Specifically, the exercise prescription must address the frequency, intensity, type, and time (duration) of exercise each patient is to perform. These components of the exercise prescription are easily remembered as the *FITT principle* (frequency, intensity, type, and time; see Table 14.4 and Figure 14.1).

Frequency

All patients should strive to be physically active every day. This is particularly true for patients who are most interested in improving their overall general health. Although improvements in cardiorespiratory conditioning are possible in previously sedentary individuals who exercise only once or

twice a week, programs with such limited activity are ineffective at increasing stamina and are often abandoned. Individuals interested in improving cardiovascular endurance should exercise three or four times per week. More than four exercise sessions per week do not provide any added benefit in terms of cardiovascular fitness, but this many sessions may increase the risk of injury.

Intensity

Exercise intensity represents the central component of the exercise prescription. Intensity represents the strength of the training stimulus. In general, the benefits of physical activity follow a typical dose–response curve. Previously sedentary patients and others who are interested in health-related physical fitness should always begin at low intensities, typically 40 to 50% of their maximum aerobic capacity (VO_{2max}); patients interested in improving cardiovascular endurance should exercise at between 50 and 85% of VO_{2max}. VO_{2max} represents the maximum amounts of oxygen that an individual can breathe in, transport to metabolically active tissues, and use for productive work during maximal exertion. VO_{2max} can be measured directly during clinical exercise testing or estimated using several simple tools such as target heart rates, ratings of perceived exertion, or the talk test (discussed below). The intensity of activity must also be specifically tailored to the patient's goals.

Target Heart Rate

At moderate levels of exercise a linear relationship exists between heart rate and VO_{2max}. The patient must be comfortable taking his or her own pulse. If individuals have a difficult time finding their pulse, commercial heart rate monitors are also available. The most common areas for a pulse check during exercise are the distal radial artery and the carotid artery. Patients should be instructed to count the number of pulsations in 15 seconds and multiply this number by four to determine their heart rate in beats per minute (bpm). Maximum heart rate can either be measured directly during clinical exercise testing or calculated based on the patients' age.

Maximum heart rate (MHR) = 220 – patient age

For the exercise prescription, patients should be instructed to keep their heart rate within the target zone for the duration of their training session. The most common target heart rate range is between 40 and 80% of maximal heart rate. Patients who are new to their exercise prescription should exercise toward the lower portion of the target zone. The target zone is easily modifiable if patients are on cardioactive medications and can be readily adjusted as patients become more conditioned with continued exercise.

Heart Rate Reserve

A modification of the target heart rate range is the heart rate reserve. This formula compensates for individual differences in resting heart rate. The heart rate reserve is calculated by subtracting the patient's resting heart rate from his or her age-predicted maximum heart rate (220 – age):

Heart rate reserve (HRR) =
Maximum heart rate – resting heart rate

Heart rate reserve (HRR) =
[(220 – age) – resting heart rate]

The target heart rate range (THRR) is calculated by multiplying the desired training intensity (typically 40 to 80%) by the heart rate reserve and then adding the resting heart rate:

THRR = [(MHR – RHR) × % training heart rate]
+ resting heart rate

THRR = [(HRR) × training intensity]
+ resting heart rate

For example, a 45-year-old male has a resting heart rate of 85 beats per minute (bpm). His calculated maximal heart rate is 220 bpm – 45 bpm = 175 bpm. His heart rate reserve is 175 bpm – 85 bpm = 90 bpm. He plans exercise at 40 to 60% of his maximal capacity. Using the heart rate reserve formula, his target heart rate range is 121 bpm to 139 bpm. Note that this rate is slightly higher than if it were based on the age-predictable maximum heart rate. The target heart rate range is a useful concept that patients can use to monitor exercise intensity.

Rating of Perceived Exertion

For patients who are not interested in using the target heart rate or are uncomfortable taking their pulse, self-ratings of perceived exertion (RPE) are another useful tool for prescribing activity intensity (Table 14.5). As with the association between heart rate and VO_{2max}, scales of perceived exertion are also based upon a relatively linear association between heart rate and the level of perceived physical exertion.[20] The scale of perceived exertion

TABLE 14.5
Borg Scale of Perceived Exertion[88]

Traditional Borg RPE Scale		Modified Borg RPE Scale	
6		0	Nothing
7	Very, very light	0.5	Extremely weak
8		1	Very weak
9	Very light	2	Weak
10		3	Moderate
11	Fairly light	4	
12		5	Strong
13	Somewhat hard	6	
14		7	Very strong
15	Hard	8	
16		9	
17	Very hard	10	Very, very strong (maximal)
18		11	
19	Very, very hard	12	Absolute maximum

Source: Borg, G.A., *Borg's Perceived Exertion and Pain Scale,* Champaign, IL, Human Kinetics, 1998. With permission.

are very useful for assessing exercise intensity, particularly for individuals on medications that slow the heart rate (e.g., beta blockers, certain calcium channel blockers) or patients with abnormal cardiac rhythms (e.g., atrial fibrillation, atrial flutter). For example, using the original scale of 6 to 20, it is easy to teach patients the concepts of rest (scale = 6) and maximal exertion (scale = 20). Based upon these anchors, patients can grade their exercise intensity in a relatively linear fashion from light to moderate to difficult. The relative perception of intensity of each patient is a reproducible level that can be used reliably for exercise prescription. The concept of perceived exertion is easiest to reinforce during the clinical exercise test but can be taught to anyone.

Talk Test

Using this tool, patients should be instructed to exercise to a level where they are able to carry out a conversation without undue breathlessness. Using the principle of the talk test, it is relatively easy to ensure that patients do not exceed their ventilatory threshold (the intensity of exercise whereby respiratory rate markedly increases in an attempt to buffer lactate accumulation from exercising muscle). For example, patients who are exercising at a moderate intensity should be able to engage in conversation with an exercise partner without stopping or slowing down to catch their breath. A useful variation of the talk test is the talk–sing test. Using this variant, patients are instructed to exercise at an intensity where they can comfortably talk but would be unable to sing. Here again, the principle is to ensure that patients exercise at an adequate intensity without exceeding the ventilatory threshold.

Type (Choice of Activity)

When writing an exercise prescription, the type of physical activity should be based on each individual's interests as well as his or her current level of fitness. It is also important to recommend a variety of activities to maintain patient interest and discourage boredom. Activities should employ large-muscle groups that are used in continuous and rhythmic motion. By far, the most common physical activity is walking. Walking is an excellent way to begin an exercise prescription. Walking requires no specialized equipment or exercise facilities. Walking can also be used as both a fitness activity and a lifestyle activity. Examples of fitness activities include traditional sports and recreational activities such as swimming, running, basketball, tennis, or bicycling. Lifestyle activities include walking, yardwork, housework, and stair climbing. Both types of activity can be used to provide an adequate exercise stimulus. The choice of activity depends on each patient's goals and preferences. Depending

on availability, accessibility, and affordability, patients can also use specialized facilities such as gymnasiums, pools, and fitness centers to increase the variety of their exercise program (Tables 14.6 and 14.7).

Time (Length of Activity Sessions)

The goal for every individual is 30 to 60 minutes of physical activity each and every day. Short, intermittent bouts of low-intensity exercise can be accumulated throughout the day[3] and are useful for improving overall health. Individuals who are interested in developing cardiorespiratory fitness or increasing their VO_{2max}, however, must exercise at progressively greater intensities for increasingly longer periods of time. Individuals who are just beginning an exercise program can be safely instructed to limit their initial activity sessions to 10 to 15 minutes. This helps reduce the risk of injury and assists patients with acclimation to an increasingly active lifestyle. Once patients are comfortable with this routine, they should be instructed to gradually increase their activity sessions to the recommended 30 to 60 minutes. A common strategy is to have patients perform an initial warm-up period of 5 to 10 minutes. During the warm-up, patients should engage in light calisthenics and stretching activities in preparation for more strenuous activity. The actual training or activity session follows the warm-up and should be 20 to 40 minutes in length depending on the patient's level of interest and ability. Following the activity session, patients should spend an additional 5 to 10 minutes performing cool-down activities. While this traditional paradigm has never been shown to augment patient compliance or reduce the rate of musculoskeletal injury, it is familiar to many patients and can add variety to the exercise routine. To avoid injury, patients should be instructed to gradually increase the frequency, duration, and intensity of their exercise sessions over a period of several months.

MUSCULAR STRENGTH AND CONDITIONING

The most recent guidelines from the American College of Sports Medicine[2] regarding exercise prescriptions specifically recommend that muscular conditioning be included for all patients (Level of Evidence C, consensus statement). Adequate muscular conditioning is essential for patients to perform their routine activities of daily living without undue fatigue or strain. The exercise prescription, therefore, should include activities that focus on muscular strength and muscular conditioning. Muscular endurance is the ability of a muscle group to engage in repetitive movements without undue fatigue or strain. Muscular strength is the ability of an individual muscle group to move a maximum resistive force. Adequate muscular strength and endurance are both necessary for optimal performance of the routine activities of daily living.

FITT Principle for Muscular Conditioning

The same FITT principle used for the cardiovascular exercise prescription can be used to prescribe muscular conditioning activities.

Frequency

Muscular conditioning activities, for either muscular strength or conditioning, should be performed two or three times per week.[2] A common strategy is for individuals to work on upper extremity resistance training one day of the week and lower extremity training another day of the week.

Intensity

The intensity of muscular conditioning is expressed in terms of resistance and repetitions. Muscular endurance is developed using light resistance with many repetitions. Muscular strength is developed using greater resistance and fewer repetitions. Intensity is expressed as a function of an individual's repetition maximum (RM). One RM is the maximum amount of weight for a particular exercise that the individual can lift once using proper technique; 12 RM is the amount of weight the individual can lift 12 times to the point of muscular fatigue using proper technique. To develop muscular strength requires the use of greater resistance and fewer repetitions. For example, individuals training the upper extremity can perform exercises for each major muscle group using two to three sets of a 3-RM to 5-RM resistance. To develop muscular endurance, a lower resistance and greater numbers of repetitions are recommended; commonly, this would include two to three sets using an 8-RM to a 20-RM resistance.

All muscular conditioning exercises should be performed at a rhythmical pace through a full range of motion without undue strain or exertion. Breath holding should be avoided during lifting activities. For individuals who are uncomfortable in the gym, or cannot afford membership to a fitness club, many simple activities to develop

TABLE 14.6
Approximate Energy Expenditure for Common Physical Activities

Energy	Recreation	Occupation
1.5–2 MET	Standing	Shaving/dressing/showering
4–7 mL O_2/min/kg	Card playing	Desk work
2–2.5 kcal/min	Sewing/knitting	Electric typing/writing
	Flying, motorcycling	Auto driving
	Walking (1–2 mph/1.6–3.2 kph)	Dusting/light housework
	Model ship building	Calculator machine operation
	Bed exercise	Watch repair
		Light assembly work
		Hammering
2–3 MET	Golf (powercart)	Car washing
7–11 mL O_2/min/kg	Bowling, billiards	Auto repair
2.5–4 kcal/min	Darts, piano playing	Kneading dough
	Fishing (standing, sitting), croquet	Manual typing
	Power boating, shuffleboard	Ironing/tailoring
	Skeet shooting, light woodworking	Bartending
	Walking (2–3 mph/3.2–4.8 kph)	Heavy level work, dredge
	Cycling (5 mph/8 kph)	Riding power lawn mower
	Shopping	Television, radio repair
	Kayaking, rowing, canoeing (2.5 mph/4 kph)	Crane operator
	Horseback riding (walk)	Upholstering
	Horseshoe pitching	Using hand tools
		Cleaning/scrubbing/waxing
		Washing/polishing/sanding
Aerobic Threshold		
3–4 MET	Calisthenics (light)	Janitorial work
11–14 mL O_2/min/kg	Fly fishing	Brick laying/plastering
4–5 kcal/min	Archery	Twisting cables/pulling on wires
	Badminton (doubles)/tennis (triples)	Raking leaves
	Sailing (small boat)	Machine assembly
	Ice boating	Hitching trailers
	Horseback riding (trot)	Cranking up dollies
	Badminton (social doubles)	Mopping/hanging wash
	Softball (noncompetitive)	Window cleaning
	Music (energetic)	Light plumbing
	Golf (pulling bag cart)	Welding (moderate)
	Volleyball (six-man noncompetitive)	Plowing, tractor
	Walking (3–3.5 mph/5–5.8 kph)	Trailer-truck driving
	Cycling (6 mph/10 kph)	Light lawn mower pushing

TABLE 14.6 (CONTINUED)
Approximate Energy Expenditure for Common Physical Activities

Energy	Recreation	Occupation
		Wheelbarrow (100 lb or 45 kg); assembly line work (light or medium, parts appearing at an approximate rate of 500/day or more); lifting a 45-lb/20-kg part every 5 min or so)
		Stocking shelves, packing or unpacking light or medium objects
4–5 MET	Table tennis	Heavy machine repair (farm, airplane, plumbing)
14–18 mL O_2/min/kg	Golf (carrying bag)	Using powersaw on hardwood
5–6 kcal/min	Dancing (social)	Pushing a power mower/cart/dolly
	Tennis (doubles)	Gardening/hoeing
	Badminton (singles)	Baling hay
	Calisthenics (moderate)	Carpentry (light)
	Swimming (light)	Painting
	Tetherball	Masonry
	Baseball (noncompetitive)	Paperhanging
	Walking (3.5–4 mph/5.8–6.4 kph)	Interior repair/remodeling
	Cycling (6.5–8 mph/10.4–12.8 kph)	Stair climbing (slow)
	Kayaking, rowing, canoeing (3 mph/4.8 kph)	Putting in a sidewalk
		Carrying trays/dishes
		Gas station mechanic
		Military marching
		Lifting and carrying 20–44 lb/9–20 kg
		General heavy industrial labor
		Walking from room to room
		Pumping tire
		Plowing with a horse
5–6 MET	Weight training (light to moderate)	Garden digging
18–21 mL O_2/min/kg	Hunting (small game)	Shoveling light earth
6–7 kcal/min	Fishing (wading)	Stair climbing (moderate)
	Softball (competitive)	Exterior remodeling or construction
	Horseback riding (posting at a trot)	
	Soccer (noncompetitive)	
	Walking (4 mph/6.4 kph)	
	Ice/roller skating (9 mph/15 kph)	
	Cycling (8–8.5 mph/12.8–13.6 kph)	
	Kayaking, rowing, canoeing (4 mph/6.4 kph)	
6–7 MET	Backpacking (5 lb/2.2 kg)	Shoveling 10 lb or 4.5 kg 10×/min
21–25 mL O_2/min/kg	Tennis (singles)	Snow shoveling
7–8 kcal/min	Scuba diving (warm water)	Mowing lawn (push mower)

TABLE 14.6 (CONTINUED)
Approximate Energy Expenditure for Common Physical Activities

Energy	Recreation	Occupation
	Downhill skiing (light)	Splitting wood
	Water volleyball	Using pneumatic tools
	Ski touring (2.5 mph/4 kph)	Carrying/lifting 45–64 lb/20–29 kg
	Calisthenics (heavy)	Spading
	Water skiing	
	Cycling (9 mph/14.4 kph)	
	Badminton (competitive)	
	Walking (5 mph/8 kph)	
	Cross-country hiking	
	Dancing (rumba, square)	
	Swimming (moderate)	
7–8 MET	Horseback riding (gallop)	Stair climbing (fast)
25–28 mL O_2/min/kg	Snowshoeing (3 mph/4.8 kph)	Ditch digging
8–10 kcal/min	Sledding/tobogganing	Hand saw/ax on hardwood
	Ice hockey	Carrying or lifting 65–84 lb/30–38 kg
	Touch/flag football	Laying railroad track
	Paddle ball	Carrying 20 lb/9 kg up stairs
	Swimming (fast)	
	Mountain climbing	
	Basketball (nongame)	
	Walking (6 mph/9.6 kph)	
	Cycling (12 mph/19 kph)	
	Kayaking, rowing, canoeing (5 mph/8 kph)	
	Jogging (5 mph/8 kph)	
	Downhill skiing (vigorous)	
	Aerobic dance	
8–9 MET	Light sparring (boxing, martial arts)	Carrying/lifting 85–100 lb/39–45 kg
28–32 mL O_2/min/kg	Fencing	Shoveling 14 lb or 6 kg/10×/min
10–11 kcal/min	Scuba diving (cold water)	Tending furnace
	Handball (social)	Using a pick
	Endurance motorcycling	Using bar, sledgehammer
	Basketball (vigorous)	Climbing a ladder or stairs
	Soccer (competitive)	Moving or pushing desks, file cabinets
	Squash (social)	Stocking furniture; also pushing against heavy spring tension
	Running (5.5 mph/9 kph)	
	Cycling (13 mph/21 kph)	
	Ski touring (4 mph/6.5 kph) in loose snow	
	Backpacking (30 lbs/14 kg)	
	Rope jumping (60–80/min)	

TABLE 14.6 (CONTINUED)
Approximate Energy Expenditure for Common Physical Activities

Energy	Recreation	Occupation
	Weight training (heavy)	
10+ MET	Basketball (competitive)	Shoveling 16 lb or 7 kg 10×/min
32+ ml O2/min/kg	Hunting (big game)	Heavy labor
11+ kcal/min	Heavy sparring (boxing, martial arts)	Ax chopping (heavy)
	Ski touring (5+ mph/8+ kph)	
	Running, 6 mph = 10 MET	
	Running, 7 mph = 11.5 MET	
	Running, 8 mph = 13.5 MET	
	Running, 9 mph = 15 MET	
	Running, 10 mph = 17 MET	
	Cycling, 14 mph = 10 MET	
	Cycling, 15 mph = 11.5 MET	
	Cycling, 16 mph = 12.5 MET	
	Swimming 850 yd/18–20 min = 10 MET	
	Swimming (crawl) 950 yd/20–22 min = 11 MET	
	Swimming 1000 yd/20–22 min = 12 MET	
	Paddleball or racquetball (competitive)	
	Rope jumping (120–140/min)	
	Snowshoeing	
	Judo	
	Squash (competitive)	
	Handball (competitive)	
	Gymnastics	
	Spaceball	
	Trampolining	
	Wrestling	
	Backpacking (heavy)	

muscular strength and endurance can be easily performed at home. For example, the use of rubber tubing can develop dynamic muscular strength. Many common objects found around the home can also be used as a substitute for free weights. Use of the patient's body weight as the resistive force (for example, push-ups, sit-ups, or chin-ups) is also very effective.

Type

This refers to the specific muscular conditioning activity. Muscular strength and endurance are most commonly developed using either free weights or resistance machines. Small hand-held weights, wrist/ankle weights, and therapeutic tubing can also be used for resistance training.

Time

Whereas time refers to the amount of time spent engaged in an activity for purposes of cardiovascular fitness, for muscular conditioning it refers to the number of repetitions performed. Typically, several groups of repetitions referred to as a set are performed during muscular conditioning sessions. Typically, two sets are performed for each muscle group. For example, if an individual engages in two weekly muscular strength or endurance sessions per week, a common strategy

TABLE 14.7
Energy Expenditure of Common Activities

Activity	MET[a]	Kilocalories per hour[b]
Aerobic dancing	6–9	440–660
Canoeing (leisure)	3–6	220–440
Cycling <10 mph	3–6	220–440
Cycling >10 mph	6–8	440–600
Dancing	3–7	220–510
Desk work	1.5–2.5	110–180
Fishing (sitting)	1.5–3	110–220
Fishing (standing/casting)	3–6	220–440
Football, touch	6–10	440–740
Golf (walking)	2–3	150–220
Handball	8–12	600–880
Hiking, cross-country	3–7	220–510
Lawn mowing	3–8	220–600
Running at 5 mph	8.7	640
Running at 6 mph	10.2	750
Running at 10 mph	16.3	1200
Shoveling	4–7	300–510
Skiing, cross-country	6–12	440–880
Skiing, downhill	5–8	370–600
Soccer	5–12	370–880
Softball	3–6	220–440
Stair climbing	4–8	300–600
Swimming (moderate)	4–6	300–440
Swimming (fast treading)	6–8	440–600
Tennis	4–9	300–660
Volleyball	3–6	220–440
Walking at 2 mph	2	150
Walking at 4 mph	4.5	330
Walking at 4 mph uphill	6	440

[a] MET refers to a metabolic equivalent; 1 MET is the resting metabolic rate, equivalent to the consumption of 3.5 mL of O_2/kg/min.

[b] Kilocalorie expenditure is based on a body weight of 70 kg (154 lb).

is to focus on the upper extremity during one session and focus on the lower extremity during the other.

SUMMARY

When writing an exercise prescription, physicians must always consider each patient's goals and objectives. Physical activity has an unparalleled impact on health and disease. Physical activity should be a routine part of everyone's lifestyle. Prescribing regular physical activity has a profound impact on the ability of patients to live better and healthier lives. No other medical prescription can make such a claim.

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PART III
PROFILES OF ATHLETES

15 THE COMPETITIVE ATHLETE*

Joseph E. Allen

INTRODUCTION

The competitive athlete can be defined as an individual who aims to excel in a given sport, devoting a significant amount of personal time and physical effort to achieve superior athletic performance. In contrast, the recreational athlete tends to focus on personal fitness in a noncompetitive setting.

There are at least 50,000 professional and amateur athletes in the United States. C.W. Burt et al. determined that sports-related injuries for all ages account for an estimated 3.7 million emergency room visits per year, with 2.6 million (68%) of those patients being between the ages of 5 and 24 years.[2]

E.F. Luckstead et al. noted supporting population study data from 1988 to 1993 which revealed a 10% increase in athletic participation for boys and a 40% increase for girls.[11] With the contemporary emphasis on athletic achievement and equal opportunities to compete, the playing field has expanded to include greater participation in the preadolescent and female populations. Consequently, the risk for psychological stress, physical injury, and burnout has been highlighted by excessive sports training regimens used with younger athletes. Performance enhancement and overtraining injuries have thus come into focus for physicians and trainers involved with athletics on multiple levels.

Physician association with athletics occurs at multiple levels: community (Amateur Athletic Union), state (Athletic Commission), and national or international (Olympics), as well as in private (professional sports team) and public sectors. Preparation of an athlete for competition requires intense training regimens in order to maintain or improve the individual's strength, endurance, and skill level. The athlete is subject to numerous external and internal stressors throughout the training year and competitive season. These pressures may be financial, educational, psychological, and physical.[1]

TRAINING AND CONDITIONING

The modern competitive athlete pursues a year-round conditioning program. The ultimate aim of the individual or team is to achieve progressive strength, endurance, coordination, and flexibility, while maintaining adequate rest and nutrition throughout the sporting season. Training sessions often require 4 to 6 hours per day, 5 to 7 days per week. The start of an intense practice or competition has traditionally involved an adequate warm-up period, followed by a cool-down period in order to prevent muscle soreness and thus shorten recovery time. Stretching before and after exercise may not confer significant protection from muscle soreness nor decrease the risk of injury (Level of Evidence B, systematic review).[6,14]

INJURIES

Even with proper training, the competitive athlete is prone to suffer injuries resulting from long hours of repetitive microtrauma to skeletal muscle and tendons. Overuse injury begins as microscopic soft tissue tearing or bony stress, which usually presents within 48 hours as muscle pain and soreness. Further physical stress and exertion may progressively result in significant injury, such

* In the second edition, this chapter was authored by Herbert G. Parris.

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as muscle or tendonous strain, ligament sprain, or bone stress fracture. A period of relative rest and modified exercise prescription incorporating a cross-training regimen may suffice to heal a minor overuse injury (i.e., active rest). Low-static, low-impact aerobic sports (e.g., jogging, lifecycle, swimming) may provide an acceptable alternative during the rehabilitation period. Inability to train or compete due to injury may be psychologically perceived as personal failure or weakness by the athlete and his or her teammates. With acute, sports-specific injuries, aggressive treatment is the key to a successful outcome. The standard RICE (rest, ice, compression, elevation) regimen is the mainstay for most acute athletic trauma. Despite alterations in the training regimen, the question of when to return to full competition arises. The sports medicine physician should negotiate a therapeutic plan at the time of injury, including the criteria that must be met before allowing return to play. To ensure a full recovery, it may be prudent to downplay expectations, despite external pressures (e.g., parents and coaches). Risks of further injury with continued participation must be clearly explained to the athlete, parents, and coaches, and these discussions documented. Reasonable criteria for return to play include full painless range of motion, minimal strength deficits, and adequate flexibility and proprioreceptive response to provocative testing. Regular assessment and revision of the rehabilitation program should occur at designated intervals.

PSYCHOLOGICAL ISSUES

Competitive athletes suffer less from generalized anxiety disorder and depression than the general population.[1] They also have better pre-morbid mental health as compared to non-athletes; however, athletes are at risk for psychological problems and psychiatric illnesses, as well as substance abuse.[3] For example, research has not indicated an optimal precompetition anxiety response among athletes but performance may be enhanced with high levels of anxiety.[13] Nevertheless, as many as 10% of the athletic population may suffer clinically from anxiety and depression.[1] The percentage increases when an athlete has suffered physical injury. This may result from lack of mature coping mechanisms, loss of self-esteem, and failure to seek professional attention. With increasing frequency, the illicit use of drugs is a common method used to enhance faltering performance and may contribute significantly to behavioral problems (e.g., violence and antisocial behavior).[5,8] The signs and symptoms of psychiatric conditions should be recognized and pursued by the sports medicine physician. Evaluation methods include a thorough history and physical, interviews with family and friends, routine lab testing, toxicology screens, and psychological testing.[5] A number of psychological objective tests may be useful; for example, the Profile of Mood States (POMS) scores four areas of mood (anger, anxiety, depression, and obsessive–compulsive behavior) and can be used periodically to identify significant psychological stressors and thus prompt professional intervention.

SPECIAL ISSUES

Maturity matching is important in preadolescent competitors. Size and/or strength mismatch in young athletes is not uncommon and creates significant risk for physical injury as well as psychological stress.[1,2,11] Growth plate injuries are commonly misdiagnosed in the pediatric/adolescent population, and any acute joint injury should be carefully evaluated. The primary care sports medicine physician should be familiar with the diagnosis and management of the female athlete triad (anorexia/bulimia, amenorrhea, and osteoporosis), as well recognize the potential signs and symptoms of performance enhancing substance abuse.[3,7,8]

Burnout or overtraining syndrome (OTS) is one of the most common sequelae of long-term, high-performance athletic training.[4,12] Primarily a neuroendocrine disorder, OTS occurs when an athlete's tolerance to heavy training is exceeded, resulting in poor athletic performance, decreased maximal training thresholds, chronic fatigue, increased susceptibility to illness, poor sleep, and depressed mood. Current research is focused on the role of increased cytokine production due to microtrauma from overtraining, which may alter immune function and result in the increased incidence of upper respiratory illness associated with OTS.[9] By modifying the exercise prescription (e.g., cross training) or by employing short periods of rest, overtraining syndrome can be avoided or alleviated and performance maximized. Total rest and change of environment may be necessary with profound physical and mental exhaustion. Training sessions should never be increased during a staleness period.

The careful implementation of a long-term training program can allow an athlete to peak at the proper time during competition.[4] Long-term planning should include a number of short-range goals for the athlete to accomplish. Peaking may be achieved by altering the training schedule as

a competition nears (e.g., shorter and more intense sport-specific practices, carbohydrate loading, proper rest, and nutrition). Randomized controlled trials support the use of high-intensity interval training (HIT) to maximize performance in the final stages leading up to competition.[10] In general, one peak per season, lasting 1 to 2 weeks, is advisable, with a 2- to 3-day training break before major competition. If the training plan for an entire year or longer is synchronized to the specific sports season and competition schedule, the athlete will optimize performance and minimize injury and performance errors.

SUMMARY

The sports physician's primary aim is to maximize the competitive athlete's performance while minimizing time lost due to injury. The past quarter century has seen a significant increase in participation in preadolescent and women's sports. With the pressure to win at all costs, the issues of overuse injury syndromes and performance-enhancing substance abuse have gained medical and public notoriety. Injury prevention and safety should be the teaching priority for young athletes. Adult training programs are inappropriate for young children and adolescents. When an injury occurs, a therapeutic plan should be agreed upon, including specific criteria for return to competition. The physician also should anticipate stress-related anxiety and depression in the injured competitive athlete and provide professional intervention as needed.

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16 THE WEEKEND ATHLETE

E. James Swenson, Jr.

BACKGROUND

Many Americans enjoy watching and participating in sports. Unfortunately, only a minority of Americans engage in regular aerobic exercise, and our society as a whole is tending toward obesity and a sedentary lifestyle. Approximately 70 to 80% of adults participate in some form of physical activity, but only 20% are in a regular exercise program and 20 to 30% are completely sedentary. Obesity increases the incidence of hypertension, diabetes, cardiovascular disease, and certain forms of cancer. If Americans were motivated to participate in regular aerobic exercise as per the guidelines of the American College of Sports Medicine (ACSM) or the American Heart Association, we would observe a significant decrease in obesity and the degenerative diseases associated with that condition, as well as less risk of injury to the musculoskeletal system. Patients should also be encouraged to eat a healthy diet following the guidelines of the American Heart Association or the American Cancer Society.

PHYSIOLOGICAL ADAPTATIONS

Part-time athletes participate on weekends at irregular intervals. While activities vary, many are vigorous sports and exercise. Both the cardiovascular system and the musculoskeletal system adapt to the stresses placed on them. Part-time athletes are at risk for both traumatic and overuse injuries because of the deconditioning maladaptations that have taken place in the microstructure of the bone, muscle, tendons, and ligaments.

EXERCISE PRESCRIPTION

Part-time athletes should be encouraged to engage in regular aerobic exercise to develop and maintain fitness that improves the quality of life, sense of well-being, and independence and decreases the risk of injury. For those individuals who have not participated in regular exercise and ultimately wish to engage in vigorous activity such as basketball, racquetball, or soccer, taking 6 to 10 weeks to progress through a walking program is recommended to build an aerobic base and prepare the musculoskeletal for more vigorous activity. All athletes should be encouraged to exercise in preparation for any sports activity. Exercise-related topics to be discussed with such athletes include adequate hydration, protective equipment, acclimatization to hot weather, and appropriate warm-up and flexibility exercises prior to vigorous activity.

COMMON INJURIES AND TREATMENT

Because part-time athletes are susceptible to upper and lower extremity tendinitis, bursitis, strains, and stress fractures, teaching them correct treatment methods, such as the following, will aid in early treatment and sometimes prevention:

- Proper use of ice
- Adjusting activity as needed to control symptoms
- Use of nonsteroidal anti-inflammatory drugs (NSAIDs)
- Identification and correction of training errors
- Cross-training (swimming, biking, etc.)

The concepts of functional testing and functional progression should also be explained. Part-time athletes are susceptible to muscle strains and ligament sprains; for acute injuries, they can be taught the principles of RICE:

- Rest
- Ice
- Compression
- Elevation

When in doubt about whether to use ice or heat, patients will rarely go wrong using ice, ice, and

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more ice. Heat is best reserved for relaxing chronic low back pain and for adductor and hamstring muscle strains. If symptoms persist despite applying the RICE technique, part-time athletes should seek additional evaluation from their primary care physician or a sports medicine professional.

SUMMARY

All physicians will have the opportunity to care for part-time athletes. Everyone can benefit from eating right and exercising regularly. By learning correct diet and exercise principles, motivated part-time athletes are able to modify their exercise, appreciate the risk of injury, and initiate treatment following overuse and traumatic injury. The physician's goal should be to encourage patients not only to enjoy watching sports but also to develop healthy, lifelong habits of nutrition and exercise. Husbands and wives should be encouraged to participate together. Parents can teach their children by example and develop close family ties in the process. A family that plays together stays together.

17 THE FEMALE ATHLETE*

Rochelle M. Nolte

INTRODUCTION

The first generation of girls that grew up with Title IX has just reached adulthood, so the culture of girls' and women's athletics is relatively new. As the number of girls and women participating in sports grows, the amount of information available about female athletes continues to increase, but as with any other relatively new area of medicine the initial information being gathered points out how much more there is to learn. While many of the benefits (physical, psychological, and social) and risks (injury) of sports and exercise are the same for women and men, some issues are specific to women. This chapter addresses issues for adolescent, reproductive-aged, and postmenopausal women.

EXERCISE IN ADOLESCENTS

Primary Amenorrhea

Primary amenorrhea is defined as delayed menarche, or the absence of menses by the age of 16.[1] Athletes who begin intensive training before puberty are at risk, especially gymnasts and ballet dancers.[2] The athlete presenting with primary amenorrhea should have a thorough history, including pubertal milestones.[1] A lack of any pubertal development can indicate hypothalamic, pituitary, or gonadal failure. An interruption of normal pubertal development can indicate ovarian failure or pituitary failure, as happens with a pituitary neoplasm. Normal breast and pubic development in the absence of menstrual periods can indicate an abnormality of the reproductive organs.[2]

A training history should include a dietary history, including a history of weight gain or loss, and a detailed history of the type, frequency, duration, and intensity of exercise. A history of the athlete's perception of stress associated with her sport, school, work, home, and peers should be elicited, as well as the support system available to her and an assessment of her coping methods. Athletes who associate more stress with their sport and competition are more likely to be amenorrheic.[2]

A full review of systems should be done, including a history of sexual activity. A thorough history of past medical problems and treatments, especially chemotherapy or radiation therapy, is also important. The athlete should be questioned about medications, illicit drug use, and anabolic steroids, which can result in amenorrhea. A family history of age of menarche or any family history

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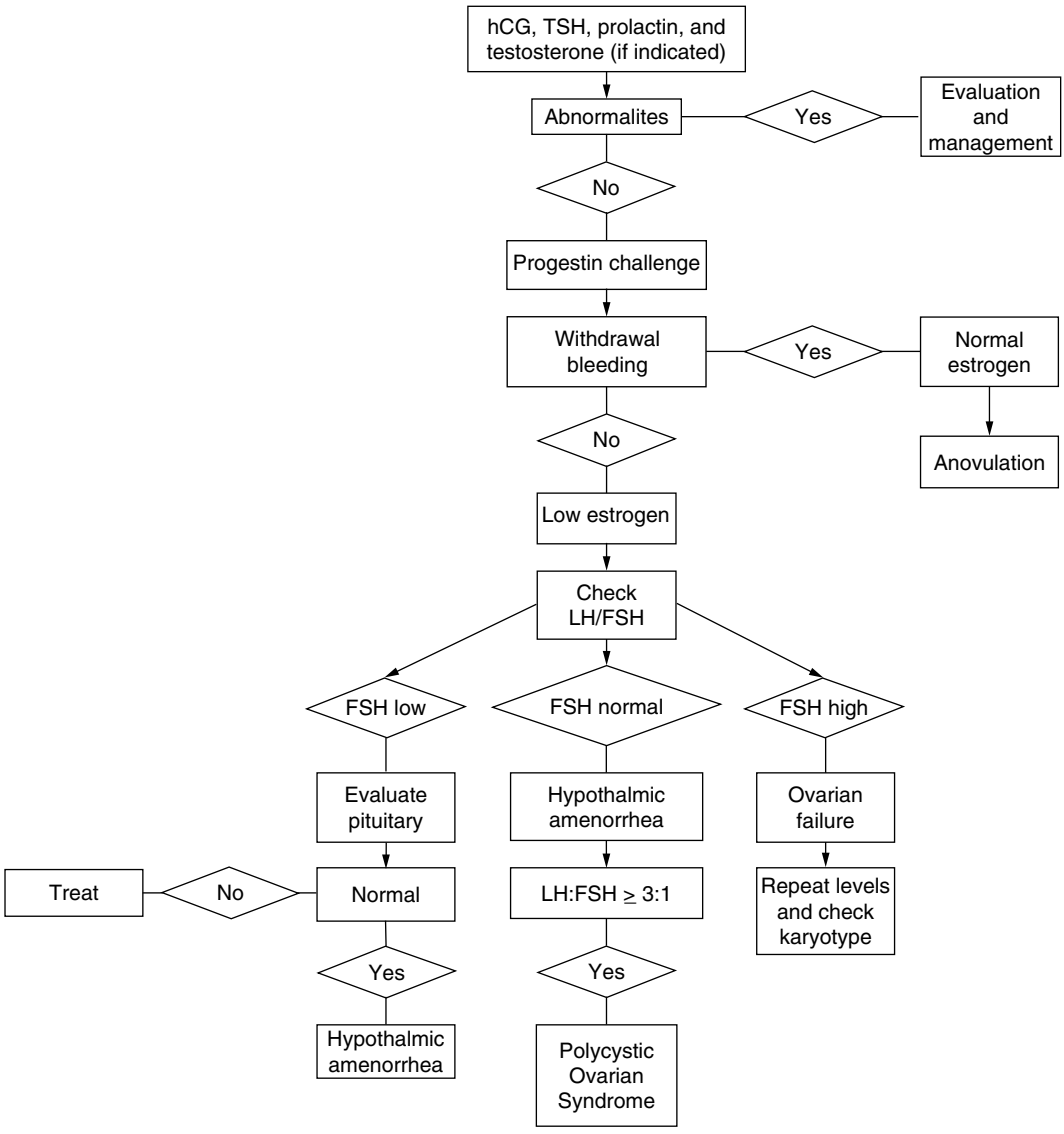

Figure 17.1 Special considerations for the female runner. (Adapted from Fieseler, C.M., *J. Back Musculoskel. Rehab.*, 6, 37–47, 1996.)

of endocrinopathies, hirsutism, amenorrhea, or congenital anomalies may be important.[2]

The physical exam should include vital signs, height, weight, body fat, arm span, Tanner stage, any characteristics of chromosomal anomalies, any traits of androgen excess such as hirsutism or acne, fundoscopic exam and visual field confrontation, evaluation for galactorrhea, palpation of the thyroid, and a pelvic exam to assess for normal anatomy.[2]

Laboratory testing for amenorrhea is shown in Figure 17.1. The progestin challenge is done by giving medroxyprogesterone as a 10-mg daily dose for 5 to 10 days.[3] A karyotype should be done in any patient who is found to have no uterus on pelvic exam or pelvic ultrasound, as well as any patient under the age of 30 found to have ovarian failure.

Hypothalamic amenorrhea can be a cause of secondary or primary amenorrhea. In hypothalamic amenorrhea, the pulsatile gonadotropin-releasing hormone (GnRH) is abnormal. Rarely, this can be caused by a tumor or trauma to the hypothalamus. A developmental defect in the hypothalamus can result in an isolated gonadotropin deficiency characterized by a lack of sexual

development, primary amenorrhea, low luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels, normal female karyotype, and sometimes an undeveloped sense of smell.[2] Psychological stress (such as moving away to college, divorce, death in the family, stressful social situations, or stress associated with scholastic or athletic performance) and physical stress (such as weight loss or gain or increase in training) are thought to affect neurohormones that regulate GnRH, leading to hypothalamic amenorrhea.[2] It is also thought that all women must reach a critical body weight and body fat percentage before reproductive function spontaneously starts. Exercise-associated or athletic amenorrhea is considered to be a subset of hypothalamic amenorrhea.[2]

If exercise-associated amenorrhea is suspected, the patient should be counseled on the risk of having decreased bone mass. Because women reach their peak bone mass in the third decade of life and then the bone mass starts to decline, the concern is that not only will these athletes be at an increased risk of stress fractures now but they will also be at risk of osteoporosis in the future secondary to never achieving their peak bone mass.

Amenorrhea is a chronically estrogen-deficient state. Because estrogen facilitates calcium uptake into bone,[4] athletes with amenorrhea are at risk of osteoporosis and stress fractures, much like postmenopausal women. The weight-bearing exercise done by athletes can help protect them but may not be enough to overcome long periods of estrogen deficiency, especially as the third decade of life is the point when women should be reaching their peak bone mass. Prevention of this loss of bone mass is one of the main goals of treating amenorrhea. Oral contraceptives are a good choice for athletes who are not trying to conceive and have no contraindications (Level of Evidence B, systematic review). This should be combined with an evaluation of the athlete's diet and exercise regimen and any underlying stress she may be experiencing so all aspects that may be contributing can be addressed. If osteoporosis is suspected, dual-energy x-ray absorptiometry (DEXA) may be indicated to see if the athlete's bone density is below the standard (Level of Evidence C, expert opinion).[5]

Secondary Amenorrhea

Secondary amenorrhea is the absence of menstrual bleeding in a woman who previously has had menstrual cycles.[2] Some sources define amenorrhea as being the absence of menstrual cycles for a length of time equal to the total time of the three previous menstrual cycles.[5] Some sources define amenorrhea as the absence of menstrual bleeding for 6 months.[5] The International Olympic Committee (IOC) has defined amenorrhea as one period or less per year.[2] Exercise-related amenorrhea is a diagnosis of exclusion. Pregnancy is the most common cause of amenorrhea in sexually active women and must be excluded. Other diagnoses to rule out include polycystic ovarian syndrome (PCOS), Asherman's syndrome, and thyroid or pituitary abnormalities. If a woman over the age of 30 is presenting with secondary amenorrhea, other etiologies to be considered are premature ovarian failure, endometrial hyperplasia, and carcinoma. Exercise-related amenorrhea can be considered a subset of hypothalamic amenorrhea, which also includes amenorrhea related to anorexia nervosa and weight loss, as well as psychological stress, such as moving away to college, divorce, or death in the family. The evaluation and management of secondary amenorrhea is similar to that of primary amenorrhea discussed above.

The Female Athlete Triad

As the name implies, the female athlete triad has three components: (1) disordered eating, (2) amenorrhea or oligomenorrhea, and (3) osteoporosis or osteopenia. The prevalence of the female athlete triad is unknown, given the secretive nature of eating disorders and underreporting by female athletes. Risk factors include chronic dieting, low self-esteem, family dysfunction, physical abuse, biologic factors, perfectionism, and a lack of nutrition knowledge.[6] Trigger factors include an emphasis on body weight for performance or appearance; pressure to lose weight from parents, coaches, judges, and peers; a drive to win at any cost; self-identity as an athlete only (no identity outside of sports); a sudden increase in training; exercising through injury; overtraining (especially when undernourished); a traumatic event, such as an injury or loss of a coach; and vulnerable times such as an adolescent growth spurt, entering college, retiring from athletics, and postpartum depression.[6]

The *Diagnostic and Statistical Manual*, fourth edition (DSM-IV) from the American Psychiatric Association lists the diagnostic criteria for anorexia nervosa and bulimia nervosa. The category of eating disorder not otherwise specified (NOS) is also included and is used for patients who have a problem but do not meet the criteria of anorexia

nervosa or bulimia nervosa. While we do not know the prevalence, we do know that adolescent girls and young adult women are at risk for developing eating disorders. Whether or not the prevalence of anorexia or bulimia is higher in the athletic population is unknown, but disordered eating does seem to be a problem for female athletes, especially those who participate in sports where leanness is encouraged (runners) or where appearance is judged (dancers, gymnasts).

Female athletes with disordered eating and decreased energy intake can develop amenorrhea. As noted previously, the athlete with amenorrhea is at an increased risk of low bone mass secondary to either inadequate bone formation or premature bone loss. This leads to an increased risk of fractures, including stress fractures.[2,6]

When taking the history from a female athlete, the physician should be aware of the characteristics of an eating disorder, such as severe self-imposed weight loss, altered body image, an intense fear of becoming fat, lightheadedness, syncopal episodes, weakness, palpitations, overuse injuries, and decreased school and sport performance. They may also complain of fatigue, chest pain, abdominal pain, bloating, diarrhea, constipation, recurrent pharyngitis, and decreased school and sport performance. Other history to obtain is a family history of disordered eating, obesity, depression, anxiety, or substance abuse. Psychological history should include smoking, alcohol, and other substance abuse; life stresses; self-esteem and control issues; and symptoms of depression.[6] Important historical questions related to amenorrhea were discussed in the previous sections.

When examining the female athlete, the physician should be aware of physical characteristics of eating disorders, including fat and muscle loss, dry hair and skin, lanugo (particularly on the trunk), cold and discolored hands and feet, decreased body temperature, hypotension, bradycardia, swollen parotid glands, conjunctival petechiae, periodontal disease and caries, pharyngeal erythema, face and extremity edema, knuckle scars, and rectal fissures.[6]

Laboratory tests to order include a complete blood count (CBC), electrolytes, calcium, magnesium, phosphorus, cholesterol, albumin, total protein, blood urea nitrogen (BUN) and creatinine, and urinalysis.[5] Other tests to consider are a pregnancy test, FSH, estradiol, prolactin, and thyroid function tests.[5] An electrocardiogram may also be indicated.

Imaging studies that may be useful in the evaluation and management of a patient with the female athlete triad include a DEXA to evaluate bone density and a triple-phase bone scan to evaluate for stress fractures.[5] If the bone scan picks up stress fractures in different stages of healing, this would help determine that the athlete has an ongoing problem and not an isolated overuse injury.

Management of the female athlete triad is multidisciplinary and requires involvement by the primary care physician, a mental health professional, and a nutritionist. These patients need guidance and education about their condition, their diet, their training, their stress, and other issues in their life. They may also be candidates for hormone replacement therapy (HRT) in the form of oral contraceptive pills, which can help with the amenorrhea (after a thorough evaluation, as discussed previously), as well as additional calcium and vitamin D. While *bis*-phosphonates are indicated in the treatment of post-menopausal osteoporosis, their use in young amenorrheic athletes is not recommended.

EXERCISE IN REPRODUCTIVE-AGED WOMEN

Exercise in Pregnant Women

Pregnancy is a normal condition. In the absence of medical or obstetric complications, pregnant women should follow the recommendations of the Centers for Disease Control and Prevention and the American College of Sports Medicine on exercise. The guidelines are accumulation of 30 minutes or more of moderate exercise daily on most, if not all, days of the week (Level of Evidence C, consensus opinion).[1] The specific exercise prescription will have to be tailored for each woman based on her baseline level of fitness, her preferred activities, and her gestational age.

Exercise during pregnancy has many benefits. Exercise helps maintain or improve cardiovascular fitness. Exercise also helps control maternal weight gain. The American Diabetes Association endorses exercise in the management of gestational diabetes when euglycemia is not achieved by diet alone. Evidence also indicates that exercise is beneficial in the primary prevention of gestational diabetes, especially in morbidly obese women (body mass index [BMI] >33) (Level of Evidence B, systematic review).[7] Exercise can help reduce musculoskeletal and other discomforts during pregnancy.[8] Exercise is also associated with improved body image and sense of well-being, much as it is in non-pregnant patients.

No evidence indicates that exercise has a detrimental effect on pregnancy or labor or fetal well-being.[9] The babies of regularly exercising women appear to tolerate labor well. They also have been shown to have a similar head circumference and length, but lower body fat, than babies born to non-exercising mothers.[10]

An exercise program should be safe and comfortable and allow the mother to maintain her fitness while minimizing risk to the fetus. The highest risk to mother and fetus with exercise is probably trauma, and this can be minimized by avoiding sports with a high risk of contact or falling, especially as the woman's balance changes in the later stages of pregnancy

Environmental conditions should be taken into account when exercising. Pregnant women should dress appropriately and drink appropriate amounts of fluids during exercise and avoid exercising in extreme heat or on uneven terrain. Exertion at altitudes of up to 6000 feet appears to be safe (Level of Evidence C),[9] but engaging in physical activities at higher altitudes carries various risks, and women who travel to higher altitudes should be aware of the signs and symptoms of altitude sickness, for which they should cease activity and descend to a lower altitude. Scuba diving should be avoided throughout pregnancy as the fetus is at increased risk for decompression sickness secondary to the inability of the fetal pulmonary circulation to filter bubble formation (Level of Evidence C).[7]

There are a few absolute contraindications to exercise during pregnancy:

- 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

The American College of Obstetrics and Gynecology has also identified some relative contraindications to exercise:

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

Any woman with these conditions who wishes to begin an exercise program during her pregnancy should be under the close supervision of a physician.

Physicians should ensure that their pregnant patients are aware of the signs and symptoms for which they should stop exercise, including dizziness, chest pain, vaginal bleeding or amniotic fluid leakage, decreased fetal movement, or preterm labor.[7]

No definitive guidelines have been agreed upon for elite or endurance athletes during pregnancy. Elite athletes have to deal with the changing physiology of pregnancy, which may necessitate changing their exercise regimen. After the first trimester, the uterus can cause obstruction of venous return in the supine position, so athletes will have to adjust weight-training and floor exercises appropriately. Motionless standing also causes a significant decrease in cardiac output and should be avoided as much as possible. Athletes should stop exercising when they feel fatigued; they should not try to "train through" fatigue during pregnancy. The increased oxygen consumption of pregnancy will lead to most women experiencing a decline in their exercise tolerance. The goal of exercise during pregnancy is to maintain fitness while avoiding fetal distress. Common sense about the environment, and the type, frequency, duration, and intensity of exercise should be recommended.

Contraception

Combination oral contraceptives that contain both an estrogen and progesterone have benefits for female athletes that extend beyond pregnancy prevention. They can help with cycle control, premenstrual syndrome, dysmenorrhea, menorrhagia, and iron-deficiency secondary to excessive blood loss.[4] The use of oral contraceptives can

also help prevent the bone loss associated with amenorrhea and oligomenorrhea (Level of Evidence B, systematic review). For athletes who prefer to avoid menses during competition (adventure race participants, triathletes, swimmers, distance runners, and potentially dancers and gymnasts) monophasic pills can be manipulated to take the 7 days of hormone-free pills once every 3 months rather than monthly. Not enough information on the transdermal form of estrogen is available to compare its efficacy in the protection of bone mass in young women to that of oral contraceptives.

Progesterone-only pills do not have the same protective effect on bone as the estrogen-containing combinations. Medroxyprogesterone given intramuscularly (Depo-Provera®) has actually been shown to have an adverse effect on bone mineral density,[11] and this should be taken into account when counseling athletes on their choices of contraception. Levo-norgestrel (Norplant®) contraceptive implants also provide no estrogen to protect bone mass and have been associated with irregular bleeding and occasionally with weight gain, both side effects that may be undesirable in athletes. Intrauterine devices (IUDs) may be a good option for athletes who are looking for long-term contraception who want to avoid hormonal manipulation, but the athlete should be counseled on the possibility of heavier menstrual bleeding and increased uterine cramping with use of an IUD.

Fertility

Athletes who wish to conceive should be advised to do the healthy things we advise all women to do, such as get all of their healthcare maintenance up to date and ensure that they are eating a healthy diet and including adequate amounts of folic acid and avoiding tobacco and alcohol. They should also have their physician evaluate any medications they are taking to ensure they are safe to be taken during pregnancy. If a woman is exercising heavily and not having regular menstrual cycles, she should be advised to decrease her exercise intensity in order to allow resumption of normal ovulatory cycles. It is thought that endorphins, cortisol, or other neurohormones such as melatonin or dopamine suppress the pulsatile GnRH[1,2] when they are released with extensive exercise, having an adverse effect on fertility, but more research is needed on this hypothesis. If decreasing the exercise intensity does not work, other causes of infertility should be ruled out. If the woman is over the age of 35 and worried about age-related infertility, ovulation induction may be offered. Any effects of athletic amenorrhea on fertility and reproductive health appear to be reversible with treatment of the amenorrhea, after the athlete has decreased her activity level and increased her caloric intake and body fat percentage.

EXERCISE IN POSTMENOPAUSAL WOMEN

The health benefits of exercise for postmenopausal women include decreased hypertension and diabetes, improved muscle strength and balance, weight control, and psychological benefits. Exercise is an important part of a preventive health program for the postmenopausal woman, as is osteoporosis prevention, and is relevant to the question of whether or not to use hormone replacement therapy. Postmenopausal women (as well as some younger women) may also face a barrier to exercise if they are experiencing urinary incontinence. This must be addressed, just as for all the other barriers to exercise, if a woman is really going to have an exercise program she is comfortable with and will continue.

Hormone Replacement Therapy

Whether or not to use hormone replacement therapy is a decision each woman will have to make with the counseling and guidance of her physician. Benefits of HRT include control of perimenopausal vasomotor symptoms (hot flashes, flushes, night sweats), prevention of urogenital atrophy, and prevention of osteoporosis. HRT has not been shown to be beneficial in preventing cardiovascular disease (Level of Evidence A, randomized controlled trial).[12] Deciding who should take HRT depends on what the patient's risk factors are for mortality and morbidity as she ages. The only absolute contraindications for HRT are known or suspected estrogen-receptor positive breast cancer, thromboembolic disease, undiagnosed uterine bleeding (which could indicate uterine cancer), and liver dysfunction. The use of HRT is an individual decision each woman must make based on her risks for osteoporosis, cardiovascular disease, breast and gynecological cancers, and vasomotor symptoms.

Osteoporosis

Over 20 million Americans have osteoporosis.[13] Osteoporosis is associated with about 600,000 vertebral fractures and over 200,000 hip fractures annually. Over 400,000 radial and other extremity fractures occur annually that are associated with osteoporosis. Women have an 8-fold increase in

their risk of vertebral fracture and a 2-fold increase in their risk of hip fracture compared to men. Women experience a 4-fold increase in the risk of hip fracture with each decade after the age of 50. Over 25% of the patients who present with a hip fracture will be discharged to a nursing home. Over 35% will never walk normally again.[13]

Osteoporosis is defined as bone mass >2.5 standard deviations below the normal bone mass of a normal sex-matched young adult (*T*-score). Postmenopausal osteoporosis in women is the most common form with which people are familiar, but other groups are at risk of developing osteoporosis as well. Elderly men, people who have been taking steroids or high doses of levothyroxine for a long period of time, and young female athletes with eating disorders and amenorrhea are at risk, as well. Osteopenia is defined as a *T*-score of –1 to –2.5. The method of diagnosing osteoporosis is by DEXA, which is used to measure the bone density at the hip, spine, and usually the radius. This is used to generate the above mentioned *T*-score and an age-matched *Z*-score.

The U.S. Preventive Services Task Force recommends bone mineral density (BMD) screening by DEXA for women over age 65. They also recommend beginning routine screening at age 60 for women identified as being high risk for developing osteoporosis (Level of Evidence B, Systematic review).[14]

Determining who is at risk for osteoporotic fractures is based largely on history. Non-modifiable risk factors such as age, sex, race, history of a low-trauma fracture, and family history of osteoporosis should all be noted. Modifiable risk factors include low body weight, low calcium intake, tobacco use, excessive alcohol use, deconditioning, low muscle mass, poor balance, poor vision, estrogen deficiency, and fall hazards in the environment, all of which can be addressed in the treatment and prevention of osteoporosis and fractures.

Non-pharmacologic measures used in the treatment and prevention of osteoporosis include decreasing tobacco and alcohol use and encouraging weight-bearing exercise, which helps increase bone density and muscle mass and improves balance. Nutritionally, women should be taking in 1500 mg of calcium daily, divided into at least three doses, as more than 500 mg of calcium cannot be effectively absorbed at one time. Women should also be taking 800 IU of vitamin D daily (Level of Evidence C, expert opinion). The vitamin D should be taken with the calcium to aid in the absorption of the calcium. More research needs to be done on other nutrients to determine their effect on osteoporosis before specific recommendations can be made. Pharmacologic treatment and prevention of osteoporosis includes estrogen, bisphosphonates, selective estrogen receptor modulators (SERMs), and calcitonin.

Appropriate prevention and treatment of osteoporosis is something that can have a huge impact on the quality of life of patients by helping them remain active and independent. Osteoporosis is clinically silent until the very late stages. Thinking about screening for osteoporosis so treatment can be started early is one of the most important things we can do to help prevent fractures from osteoporosis.

Urogenital Symptoms

Urinary incontinence is defined by the International Continence Society as the "involuntary loss of urine which is objectively demonstrable and a social and hygienic problem."[15] Urinary incontinence affects up to 30% of community-dwelling postmenopausal women and can be a huge barrier to exercise.[16] It can also be an embarrassing topic a patient will not bring up voluntarily. The most common types of incontinence include stress urinary incontinence, urge incontinence, or a mixed type that has symptoms of both stress and urge incontinence. Overflow incontinence resulting from urinary retention is less common in active community-dwelling individuals and is more specific to certain medical and neurological conditions.

Stress urinary incontinence (SUI) is the loss of urine related to increased intraabdominal pressure. This type of incontinence is common in premenopausal women as well as postmenopausal, especially after vaginal deliveries, but has also been reported in 28% of nulliparous elite athletes during exercise.[15] The primary complaint is a loss of urine with activities that increase intraabdominal pressure, such as sneezing or coughing, or exercises that include running or jumping. Athletes involved in high-impact activities such as gymnastics (dismount/tumbling) or basketball (jumping) are also at risk.[15,16] Management is directed at correcting the underlying pelvic relaxation using exercises that strengthen the muscles of the pelvic floor. These exercises can be taught by providing the patient with a Kegel exercises patient handout or having the patient see a clinic that has nursing or physical therapy staff instructed in biofeedback or electrical stimulation to follow the patient through their rehab program and measure their progress every 2 to 6 weeks with a manometer. Doing these exercises requires a very motivated patient who is willing to learn

how to do the exercises and continue them indefinitely, but there is no guarantee that they will work for everyone. Some women have extreme pelvic floor relaxation and pelvic prolapse that cannot be overcome with exercises and will require surgery as a final treatment or a pessary as a temporizing measure. The estrogen deficiency associated with menopause can cause vaginal and urethral atrophy, contributing to incontinence. Systemic or topical estrogen can be used to correct the deficiency.

Urge incontinence is the loss of urine associated with an uncontrollable sensation of having to void immediately. Patients complain of urgency, frequency, and dribbling on the way to the bathroom or spontaneous complete bladder emptying. They also can complain of feeling the urge to void with provocative stimuli such as running water. Urge incontinence can be secondary to detrusor instability or hyperreflexia and can be managed with bladder retraining through the use of a prompted voiding program or with medications that decrease detrusor activity by blocking muscarinic receptors on detrusor smooth muscle. Oxybutynin (Ditropan®) was the most commonly used antimuscarinic, but the side effects of dry mouth and constipation and occasionally sedation led many women to discontinue this medication. Newer antimuscarinics have been developed with improved side-effect profiles and dosing regimens, such as a sustained-release version of oxybutynin (Ditropan XL®) and tolterodine (Detrol®). Other drugs used in the treatment of incontinence include imipramine (Tofranil®), a tricyclic antidepressant with anticholinergic properties, and hyoscyamine (Levsin®).

Postmenopausal women also have a higher incidence of mixed incontinence, with traits of both stress and urge incontinence. Treatment should address both aspects of the problem.

SUMMARY

Many of the risks and benefits of exercise are the same for men and women, but certain issues are gender specific. Female athletes of all ages have issues such as primary and secondary amenorrhea, contraception, fertility, hormone replacement therapy, pregnancy, incontinence, osteoporosis, eating disorders, and the female athlete triad that must be addressed. This is a relatively new area of medicine, and we should be able to make more specific recommendations in the future as we learn more about the physiology of the female athlete.

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18 THE CHILD ATHLETE

Sally S. Harris

INTRODUCTION

Pediatric and adolescent sports medicine is a relatively new and rapidly growing field. It has developed in response to the explosion of organized sports participation for children that has occurred over the last several decades. The participation of girls in sports has increased dramatically since the passage of Title IX in 1972, raising specific concerns with regard to the health consequences of sports participation for young girls. We have also witnessed the emergence of the elite child athlete. In certain sports, such as gymnastics, figure skating, swimming, and tennis, young girls in particular manifest a unique ability to excel on an international level during the pubertal years. Children are specializing at younger ages in one specific sport and training exclusively in that sport year-round. In addition, training programs are becoming increasingly rigorous, and it is no longer unusual to find children training multiple hours each day, with regimens of intensity equivalent to those formerly demanded only of adult athletes.

The trend toward greater participation by children in organized sports and their involvement in intensive training has raised a number of concerns with regard to the appropriateness and health consequences of these activities. For instance, concurrent with the rise of organized sports has been a dramatic increase in musculoskeletal injuries, particularly overuse injuries, which were previously encountered almost exclusively in adults. Other concerns include the risk of injury to the growth centers of the immature skeleton, as well as the effects on overall growth, maturation, and psychological well-being. Optimal care of young athletes requires an understanding of the fact that child athletes are not merely small adults, but have distinct physiologic, psychologic, and developmental responses and needs with regard to sports training and participation.

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GENERAL ISSUES

Youth Fitness

Concern is growing that children are becoming more overweight and less physically fit. Studies suggest that increasing levels of obesity among children are due in part to declining levels of physical activity over the past several decades.[1] Measures of physical fitness and endurance among children have also declined, and measures of body fatness, such as skinfold thickness and body mass index (BMI) have increased (Level of Evidence B, epidemiologic study).[1–3] Children spend less time in physical education classes in school and more time in sedentary activities such as watching television and playing computer games. Low levels of physical activity are associated with several negative health behaviors such as cigarette smoking, marijuana use, lower consumption of fruit and vegetables, greater television watching, failure to wear seat belts, and low perception of academic performance (Level of Evidence B, epidemiologic study).[4] Studies show that participants in sports and exercise programs are less likely to be overweight[5] (Level of Evidence B, epidemiologic study) and that children who watch more television are more overweight.[6] The prevalence of obesity is lowest among children watching 1 or fewer hours of television daily and highest among those watching 4 or more hours of television per day (Level of Evidence B, epidemiologic study).[7] One randomized controlled trial provides evidence that television viewing is a cause of increased body fatness and that reducing television watching reduces childhood obesity (Level of Evidence A, randomized controlled trial).[8]

Physical activity is an important factor for preventing obesity in children (Level of Evidence B, systematic review).[9] Overweight children are more likely to become overweight adults. As the prevalence of obesity increases, the need to reduce sedentary behaviors and to promote a more active lifestyle becomes essential. Studies suggest that physical activity in childhood is an important determinant of physical activity in adulthood (Level of Evidence B, epidemiologic study).[10] It is estimated that at least 50% of today's youth do not engage in appropriate levels of physical activity (Level of Evidence B, epidemiologic study).[3,12] Unfortunately, participation in regular physical activity declines consistently from ages 12 through 21, particularly declining from ages 15 to 18, and continues into young adulthood (Level of Evidence B, epidemiologic study).[11] Adolescent females are less active than their male counterparts. Early and ongoing intervention is necessary to offset these declines in physical activity throughout adolescence and young adulthood. Three factors have been identified as important in promoting physical activity in young people: use of afternoon time for sports and physical activity, enjoyment of physical education, and family support for physical activity (Level of Evidence B, epidemiologic study).[12]

The long-term health benefits of physical activity during childhood are unclear; however, the benefits of physical activity in adulthood are well documented and include prevention of coronary artery disease, stroke, hypertension, obesity, non-insulin-dependent diabetes mellitus, osteoporosis, mental illness, and some cancers (Level of Evidence B, systematic review).[13] Studies show a modest relationship between physical fitness in adolescence and favorable lipid profiles and body fatness in young adulthood (Level of Evidence B, epidemiologic study).[14] Physical activity during childhood and adolescence may also be important in maximizing bone density (Level of Evidence B, epidemiologic study).[15] Sixty percent of children exhibit at least one modifiable adult risk factor for coronary heart disease by age 12 (Level of Evidence B, epidemiologic study).[2] Promotion of physical activity in adolescence may reduce exposure to other risk factors lasting into early adulthood. Establishment of regular physical activity during childhood is an important foundation for continued physical activity in adulthood and the associated health benefits (Level of Evidence C, consensus expert opinion).[16]

It may seem ironic that, despite the growth of organized sports participation, children in general are more overweight and less fit. In part, the selective competitive structure of organized sports limits opportunities for large numbers of children who are not talented or competitive athletes or do not have the means to participate. Informal, recreational physical activities available to such children in the past are increasingly limited by urbanization and safety concerns.

It is important to make the distinction between sports performance and health-related measures of physical fitness. The emphasis in organized sports is typically on acquisition of sports-specific skills and other attributes related to performance, such as power, agility, and speed. Health-related physical fitness, on the other hand, refers to those components of fitness required for optimal health and disease prevention, such as cardiorespiratory endurance, muscle strength and flexibility, and body composition. Although optimal physical

fitness can improve performance in sports and certain sports activities do enhance physical fitness, many children involved in organized sports may not be achieving adequate fitness levels. Children should be encouraged to participate in activities that will develop both sports skills and physical fitness.

Sports Readiness and Selection of Developmentally Appropriate Sports Activities

A child's readiness to participate in organized sports or structured training sessions depends on a combination of factors: (1) neurodevelopmental level (motor skills acquisition), (2) social development (interaction with coaches and teammates), and (3) cognitive level (ability to understand instructions).[17,18] No evidence indicates that a child's motor development can be accelerated or their subsequent sports ability maximized by physical training at very young ages;[19] for example, we have no proof that special training can groom a preschooler to become a future champion. The acquisition of motor skills appears to be an innate process that follows the same sequence in all children; however, the rate at which children master motor skills is highly variable and cannot be predicted on the basis of age, size, weight, or strength of the child on an individual basis.[20] Specific skills can be refined through repetitive practice only after the relevant level of motor development has been reached.

At what age is a child ready to begin participation in a specific activity? Although this is a commonly asked question, it has no scientific answer from a neurodevelopmental standpoint. The best answer is that sports activities should match, or be modified to match, the developmental capabilities of the individual child. Sports activities requiring skills beyond the developmental level of the participants are unlikely to be successful. When given the opportunity, children naturally select and modify activities so that they can participate successfully and have fun. Therefore, modification of equipment and rules should be made to suit the developmental level of the participants, such as smaller balls, smaller fields, shorter duration of games and practices, reduced number of participants playing at the same time, frequent changing of positions, and less emphasis on score keeping. An example of such an adaptation is the game of T-ball, in which children hit a stationary baseball mounted on a stand rather than a pitched ball that requires more advanced visual tracking skills.

Prior to age 6, most children do not have the basic skills required to participate in organized sports. Balance and attention span are limited, and vision and ability to track moving objects are not fully mature. Emphasis in this age group should be on development of fundamental motor skills, such as running, tumbling, swimming, throwing, and catching, in an environment emphasizing fun and experimentation, limited instruction, and avoidance of competition.

By age 6, most children have acquired the fundamental motor skills to begin participation in simple organized sports activities. They still lack the hand–eye coordination necessary to perform complex motor skills and the cognitive ability to understand and remember concepts of teamwork and strategies. Organized sports that can be played without complex motor skills and strategies, such as entry-level baseball and soccer, are more appropriate than sports such as football that do not lend themselves as easily to adaptation to a more basic level. Emphasis should be on skill acquisition rather than winning. By age 10 to 12, most children have acquired the motor skills and cognitive ability to begin participation in sports at a more sophisticated level, requiring complex motor skills, teamwork, and strategies.

Is it all right for young children, such as 9 year olds, to participate in a contact sport, such as football? When parents ask this question, their main concern is risk of injury. They can be reassured that young children actually have a lower risk of injury in contact sports such as football than do older children, because they do not have the size and strength to generate forces great enough to cause more serious injuries. In addition, significant physical mismatches that could put a smaller child at increased risk of injury do not occur until puberty. A more relevant concern is whether the physical contact and associated aggressiveness and competition are developmentally appropriate or enhancing the value of the experience at this age. The child's enjoyment and eagerness to participate are some of the best indicators of the appropriateness of the activity.

Motivational factors for children's participation in sports include fun, success, skill development, variety, freedom, family participation, participation with friends, and enthusiastic leadership, whereas failure, embarrassment, competition, boredom, and regimentation discourage participation.[21] In general, children are much more interested in personal involvement and lots of action than in winning and scores. Attrition in children's sports occurs to a large extent because of lack of

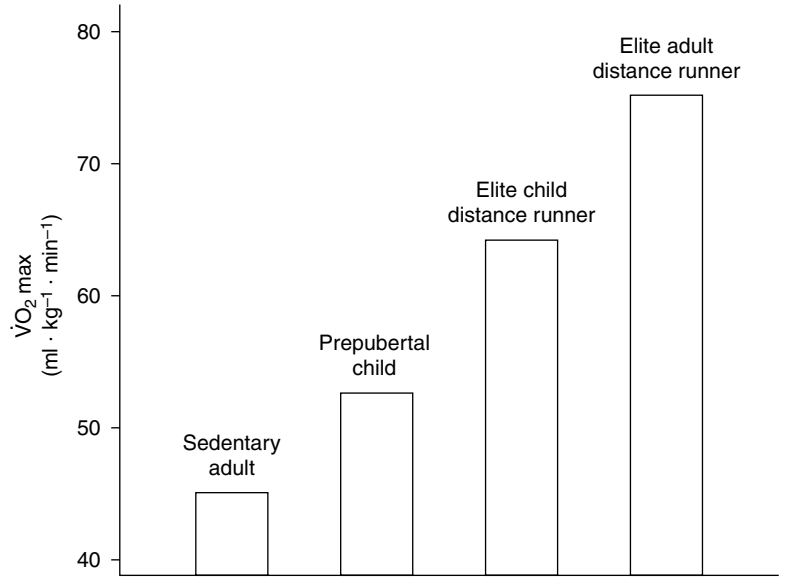

Figure 18.1 Typical values of maximal oxygen uptake in trained and athletic children and adults (males). (From Rowland, T.W., *Exercise and Children's Health*, Human Kinetics, Champaign, IL, 1990, p. 57. With permission.)

playing time, feelings of failure, and overemphasis on competition (Level of Evidence B, epidemiologic study).[22] Sports programs for children should be designed with these factors in mind in order to promote long-term involvement.

EXERCISE PHYSIOLOGY

Aerobic Training

An increase in maximal oxygen uptake (VO_{2max}), a measure of aerobic fitness, occurs in part as a function of growth alone. Absolute VO_{2max} increases with age during childhood and adolescence as a function of increasing body size.[23] In this regard, growth mimics the effects of training, as even untrained children will show an increase in VO_{2max} over time due to growth; however, maximal oxygen uptake relative to body mass (VO_{2max}/kg) remains essentially stable during childhood and adolescence, then slowly declines with age in adulthood.[23] Throughout childhood, boys have a slightly higher VO_{2max}/kg than girls. Gender differences become most apparent at puberty, when VO_{2max} rises dramatically in boys, reflecting increased muscle mass, and declines somewhat in girls, owing to an increase in body fat.

Some evidence suggests that children do experience a training response from aerobic activities, although less dramatic than that seen in adults. Cardiorespiratory profiles of child athletes are superior to those of unathletic children, although the difference is smaller than that seen between sedentary and athletic adults (Figure 18.1). When training programs meet adult standards (in terms of the intensity, frequency, and duration of the training stimulus), most studies in children show improvements in VO_{2max}.[24] The magnitude of improvement in VO_{2max} is equal to that observed in adults (7 to 26%) (Level of Evidence B, systematic review).[24] Children show other adaptations to training similar to those seen in adults, such as lower resting heart rate, decreased submaximal heart rate, increased left ventricular mass, and higher stroke volume with exercise. However, children's response to aerobic training is limited due to several factors. Children are metabolically less efficient than adults and therefore require more oxygen per kilogram of body weight to perform the same level of exercise. Children work at a higher heart rate and lower stroke volume to achieve the same cardiac output as adults. Ventilation rates are also higher, reflecting a less efficient ventilatory system.

The yield of additional aerobic training in child athletes who are already relatively fit appears to be very low. Studies have consistently failed to show a training response in child athletes as opposed to unathletic children. The difficulty in demonstrating a training response in athletic children is probably due to several factors: (1) it is

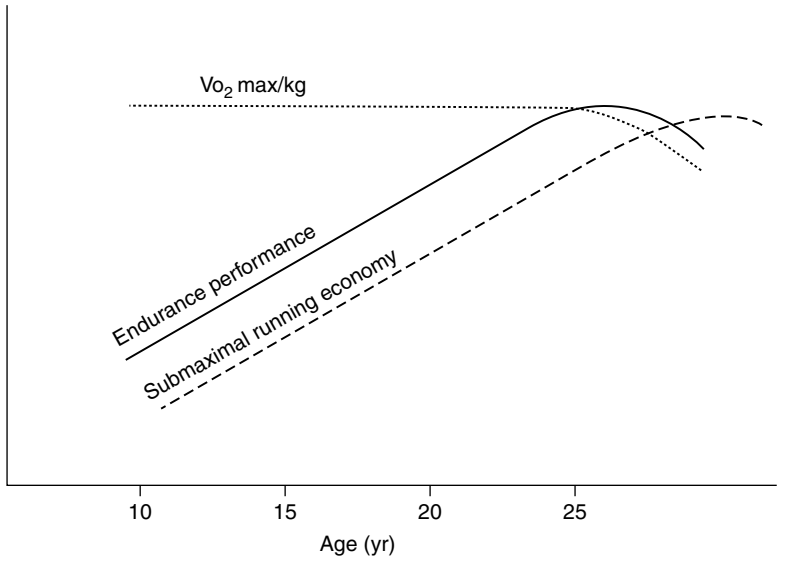

Figure 18.2 Changes in maximal oxygen uptake, running economy, and endurance performance with age. (From Rowland, T.W., *Exercise and Children's Health*, Human Kinetics, Champaign, IL, 1990, p. 56. With permission.)

difficult to separate the effects of training from those of growth alone, (2) the magnitude of the training response in children is limited by certain physiologic factors, and (3) significant gains are more difficult to detect in individuals who are already highly fit.

It is interesting to note that in children significant improvements in endurance performance can occur even though VO_{2max} does not change. For instance, although VO_{2max}/kg remains essentially unchanged during childhood, progressive improvement in endurance performance is observed with age (Figure 18.2). This apparent discrepancy can be explained by the fact that in children improvements in economy of motion (metabolic cost of the activity) contribute substantially to gains in endurance performance, irrespective of improvements in VO_{2max}.

Strength Training

Development of muscle strength during childhood is in part a function of the cross-sectional area of muscles; therefore, strength gains parallel growth. An individual is born with a fixed number of muscle fibers, and growth occurs as a result of hypertrophy and increase in fiber diameter. Prior to puberty, females and males have similar muscle mass and strength. At puberty, boys show marked acceleration in development of strength secondary to increased muscle mass, while in girls strength and muscle mass do not change appreciably.

Traditional dogma held that prepubertal children were incapable of improving muscle strength, a belief based on the fallacy that improvement in strength is dependent on the presence of androgens and associated increase in muscle mass; however, significant strength gains can occur independently of increases in muscle size. This is particularly true for children and women and is even seen in adult males during the early phases of a strength training program, before any change in muscle size occurs. Neurologic factors, such as increased neural drive, synchronization of motor unit fibers, and improved motor skill coordination, appear to be important mechanisms for strength gains in these instances. Studies of strength training in children show that both boys and girls demonstrate significant gains in strength (evidence level B, systematic review).[25] Prepubescent children make similar relative strength gains (percent improvement) compared to older children and adults but demonstrate smaller absolute strength increases.

From a practical standpoint, the value of strength training in children is of low yield. Prior to puberty, the gains in strength due to strength training are relatively small and unlikely to confer any significant performance advantages. No compelling evidence suggests that strength gains translate to improved athletic performance.

Although strength training has been shown to improve performance on selected motor fitness tests, the improvement is less than that gained by practicing the skill itself. For instance, knee extension weight training improves performance of the vertical jump, but not as much as practicing the vertical jump itself (Level of Evidence B, clinical cohort study).[26] Children are more likely to improve sports performance by practicing and perfecting the skills of the sport itself, rather than from strength training.

Although the efficacy of strength training in children has been confirmed, concern has been raised about the safety of this activity with regard to risk of injury to the immature skeleton. Although numerous case reports of epiphyseal fracture due to weight training in children have been reported, the majority appear preventable and occur as a result of improper technique, excessive loading, and ballistic movements (Level of Evidence B, systematic review).[27] The majority of such injuries occur in the home setting, where supervision may be inadequate. Supervised, prospective studies of resistance training in prepubescent children have not reported epiphyseal injury (Level of Evidence B, systematic review, epidemiologic study).[25,28–30] The majority of injuries are neither epiphyseal nor acute but rather are soft-tissue injuries, such as sprains and strains, especially of the low back (Level of Evidence B, epidemiologic study).[31,32] No evidence suggests that weight training is more risky in this regard than participation in other sports and recreational activities. Concerns that weight training might lead to muscle-boundness and decreased flexibility, which could predispose to injury, appear unwarranted; numerous prospective studies show no change or show slight improvements in flexibility (Level of Evidence B, systematic review, epidemiologic study).[25,28,30] In addition, some evidence suggests that strength training in adolescents may result in a decreased rate of injuries and rehabilitation time during other sports activities (Level of Evidence B, clinical cohort study).[33,34]

The term "weight training" should be distinguished from "weight lifting" and "power lifting." Weight training refers to a variety of resistance training modalities (free weights, weight machines) designed to increase muscle strength and endurance by performing multiple repetitions and sets of each exercise. Weight lifting and power lifting, on the other hand, are competitive sports emphasizing maximal lifts, such as the clean and jerk and the snatch (weight lifting), and the squat, dead lift, and bench press (power lifting). These activities are not recommended for children and adolescents (Level of Evidence C, consensus expert opinion).[35] Weight training is thought to be safe when closely supervised and appropriately designed. This would include emphasis on sets of low resistance and high repetitions, no maximal lifts, and no Olympic-style lifts. A sample weight training program for children and adolescents is listed in Table 18.1.

TABLE 18.1
Sample Weight Training Program

1 to 3 sets of 6 to 15 repetitions per set
Frequency of 2 to 3 sessions per week with rest day in between
Duration of 20 to 60 minutes
Progressive resistance:
Start at no resistance/weights until proper form is achieved.
Initiate resistance at the 6-repetition level; advance to 15 repetitions.
Add weight in increments of 1 to 3 lb until child can do just 6 repetitions.
Advance again to 15 repetitions before increasing weights.

Thermoregulation

Children are at increased risk for heat stress due to a multitude of predisposing factors.[36] Children produce more metabolic heat during exercise than adults. Although sweat-gland density is higher in children, they produce less sweat to dissipate the heat. Children's larger ratio of surface area to mass allows more heat uptake from the environment. Lower cardiac output leads to less peripheral perfusion and thereby lower capacity for heat transfer from body core to skin. In addition, children take longer than adults to acclimatize to new environments. Often children are less aware of early signs of heat stress and may fail to decrease their activity level. Voluntary hypohydration occurs frequently in children, and for a given level of dehydration children experience a faster rise in core temperature than adults. Obese children and very young children may be at particular risk, due to the insulating effect of increased layers of adipose tissue. The most common cause of heat-related illness in healthy children is insufficient acclimatization. In this regard, heat-related illness is entirely preventable by taking appropriate precautions and providing unrestricted access to fluids. Although water is adequate for

fluid replacement, children voluntarily drink more of flavored beverages. The additional carbohydrate in sports drinks is helpful only for sustained activities of over 90 minutes' duration.

Less is known with regard to cold tolerance of children. Like adults, most children generate adequate metabolic heat to maintain and usually increase body core temperature during moderate and intense exercise in cold weather. Children do not appear to be at greater risk of hypothermia than adults except with regard to exercise in water. In this situation, the child's larger ratio of surface area to mass allows for greater conductive heat loss: the smaller and leaner the child, the faster the cooling rate and the greater the risk of hypothermia.

Intensive Training, Growth, and Maturation

Does intensive training have adverse effects on growth and pubertal maturation? Children performing heavy physical labor have been noted to have decreased stature (Level of Evidence B, epidemiologic study).[37] Nutritional deprivation is often a confounding factor. Cause for concern is also raised by studies in animals showing shortened long bones due to prolonged training. It is reassuring that studies of child athletes show no apparent adverse effect of intensive training on growth or skeletal maturation (Level of Evidence B, systematic review, epidemiologic study);[38,39] however, proper nutrition may be particularly important in growing children involved in intensive training.

It is well recognized that athletic girls experience menarche at least 1 to 2 years later than other girls. This has raised the concern that training may adversely affect sexual development and reproductive function. Frisch[40] reported a statistical association of a 0.4-year delay in menarche per year of prepubertal training (Level of Evidence B, epidemiologic study). It remains controversial whether delayed menarche is a direct consequence of athletic activity. More likely, the relationship is a result of selectivity. Girls with delayed menarche are perhaps more likely to engage and succeed in sports, as a prepubertal body habitus (i.e., slender physique, narrow hips, long legs, and low body fat) may be advantageous in sports such as track, gymnastics, ballet, and swimming (Level of Evidence B, systematic review).[41] However, some athletes have experienced extreme delays in menarche, beyond age 16 or until after cessation of their high school or college athletic careers. In these cases, it is believed that the associated long-standing caloric deprivation and extreme weight restrictions, rather than the intensive training of the sport itself, are significant contributing factors. Evidence also suggests that intense training combined with insufficient caloric intake may restrict development of height during adolescence; however, in most cases, catch-up growth occurs when training is reduced or ceases, and final adult height is not affected.[38,39] In extreme cases, though, catch-up growth may be incomplete. Children participating in intensive training programs over many years should have their growth parameters monitored closely so that reductions in training intensity and increases in caloric intake can be implemented if growth problems are detected (Level of Evidence C, consensus expert opinion).[42]

In boys, on the other hand, evidence of delay of secondary sexual characteristics associated with sports participation is lacking. Sports such as football, baseball, and basketball seem to favor early maturers, who are temporarily stronger, heavier, and taller than their later maturing peers.

Delayed menarche has no effect on ultimate fertility; however, concern exists with regard to the effects on bone density (Chapter 17). It is certainly well recognized that athletes with secondary amenorrhea experience progressive and irreversible loss of bone density, probably due to hypoestrogenism (Level of Evidence B, epidemiologic study).[43] Amenorrhea and osteoporosis often occur in conjunction with disordered eating and comprise a condition referred to as the female athlete triad (Level of Evidence C, consensus expert opinion).[44] Premature osteoporosis is associated with increased risk of stress fractures (Level of Evidence B, epidemiologic study).[45] It is unclear whether delayed menarche has similar effects on bone density; however, studies in young ballet dancers have found that delayed menarche is associated with a delay in bone (Level of Evidence B, epidemiologic study). It remains to be seen whether the delay in bone density development is merely temporary, with catch-up bone development after menarche occurs, or whether the reduction of bone density is permanent.

Gender differences in both aerobic capacity and muscle strength become apparent at puberty, due to the increase in muscle mass in boys and the rise in body fat in girls. Prior to puberty, no appreciable differences between boys and girls in endurance, strength, height, or body mass are observed; therefore, coeducational participation prior to puberty is not thought to place girls at a

competitive disadvantage or at increased risk of injury. After puberty, however, most girls are unlikely to compete on an equal basis with boys their age and may be at increased risk of injury due to the discrepancy in strength and size.

There may be dramatic physical differences between individuals of the same gender, particularly boys, as children experience puberty across a wide age spectrum. For this reason, many authorities feel that children's participation in contact sports would be more appropriately matched on the basis of size and maturational stage than on the basis of chronologic age. Skeletally immature athletes should be counseled with regard to the potential risks of injury in competing in contact sports against athletes who are physically more mature.

INJURIES IN THE IMMATURE SKELETON

Musculoskeletal injuries in sports typically fall into two categories: (1) acute traumatic injuries (macrotrauma), which generally occur as a result of single events such as a blow or twist, and (2) overuse injuries (microtrauma), which occur insidiously as a result of repetitive musculoskeletal stress, such as occurs with rigorous training and/or biomechanically incorrect activity. Although overuse injuries used to be considered rare in children, they now account for the majority of sports injuries seen in children. The rise in overuse injuries is thought to be a direct consequence of the rise in organized sports and the repetitive training programs often associated with these activities. Risk factors are outlined in Table 18.2.

TABLE 18.2
Risk Factors for Overuse Injury in Children

Training error
Incorrect biomechanics
Anatomic malalignment
Improper environment
Muscle–tendon imbalance
Vulnerability of growth cartilage
Growth process
Overtraining
Associated disease states
Nutritional factors
Cultural deconditioning

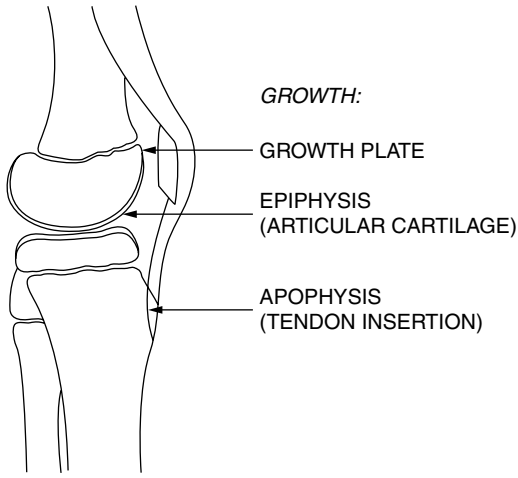

Figure 18.3 Growth cartilage is present at three sites, the growth plate, the articular surface, and the apophyses and is susceptible to overuse injury at each of these sites. (From O'Neill, D.B. et al., in *Injuries in the Young Athlete*, Micheli, L.J., Ed., *Clin. Sports Med.*, 7(3), 591–610, 1988, p. 596. With permission.)

Children differ from adults with regard to susceptibility and patterns of sports injuries. Both the presence of growth cartilage and the growth process itself are thought to place children at increased risk for injury. Growth cartilage occurs at three sites in the immature skeleton: (1) the epiphysis (growth plates), (2) the joint surface (articular cartilage), and (3) the apophysis (secondary growth centers around joints that are the attachment sites of ligaments and tendons) (Figure 18.3). Both acute injury (macrotrauma) and overuse injury (microtrauma) can occur at all three sites (Table 18.3). The musculoskeletal system appears to be most vulnerable to injury during the period of peak height velocity during adolescence,[48] perhaps due to biochemical changes that occur during rapid growth. In addition, rapid bone growth that occurs during the adolescent growth spurt results in relative tightness of muscle–tendon units spanning the joints. The resulting diminished flexibility places the adolescent at increased risk of overuse injury. For these reasons, it may be prudent to decrease the intensity of training during the periods of rapid growth and to place increased emphasis on stretching exercises to improve flexibility.

Surveys of high school sports injuries show that 70 to 80% are considered minor, causing the athlete to miss less than a week of participation. Most are sprains, strains, and contusions. Highest

TABLE 18.3
Common Injuries of Growth Centers of the Immature Skeleton

Site	Macrotrauma	Microtrauma
Epiphysis	Acute epiphyseal fracture:	Epiphysitis:
	Distal femoral epiphysis	Distal femoral epiphysis (swimming)
	Distal fibular epiphysis	Proximal humeral epiphysis (Little League shoulder)
	Distal radial epiphysis	Distal radial epiphysis (gymnast's wrist)
	Slipped capital femoral epiphysis	Proximal radial epiphysis (throwing)
Articular cartilage	Osteochondral fracture:	Osteochondritis dissecans:
	Knee	Femoral condyles
	Talus	Talus
	Elbow	Radial head
		Capitellum
Apophysis	Acute apophyseal avulsion fracture:	Apophysitis:
	Iliopsoas insertion at lesser trochanter	Apophyses about the hip and pelvis (see left column)
	Sartorius origin at anterior superior iliac spine	Tibial tubercle (Osgood–Schlatter's)
	Rectus femoris origin at anterior inferior iliac spine	Posterior calcaneus (Sever's)
	Hamstring origin at ischium	Patella (Sinding–Larsen–Johansson)
	Abdominal muscles at iliac crest	Medial humeral epicondyle
	Hip adductors at pubic rami	Olecranon
	Flexor/pronator origin at medial humeral epicondyle	

rates of injury occur in the sports of wrestling, football, gymnastics, cross-country, and soccer. Girls sustain a higher proportion of injuries to the lower extremities than boys. Younger athletes (prepubertal) are more likely to sustain injuries to the upper extremity, such as fractures of the wrist, forearm, and clavicle, due to falls on the outstretched arm. These injuries occur more commonly from accidents during recreational activities such as skating, cycling, and climbing than during organized sports. Lower extremity injuries predominate in older children and adolescents, particularly in girls.

Injury Patterns in Girls

In 1970, 1 out of 27 girls played sports as opposed to 1 out of 2.5 today. Between 1972 and 2001, the number of girls participating in high school sports exploded from 294,000 to 2.8 million. The increased participation of girls has raised concerns whether girls are at higher risk of injury than boys. Evidence suggests that injury rates for girls are different from those for boys, especially with regard to anterior cruciate ligament (ACL) tears of the knee (Level of Evidence B, epidemiologic study).[49] Adolescent and collegiate female athletes are two to eight times at greater risk for ACL tears than males, especially in the sports of basketball, soccer, and volleyball. Possible risk factors include deficiencies in training or skill level, lower extremity malalignment, smaller femoral notch size, hormonal factors, muscle firing and strength imbalances, and suboptimal biomechanics during jumping, landing, cutting, and pivoting. Recent evidence suggests that training girls to land, cut, jump, and pivot more like boys may reduce ACL injuries (Level of Evidence B, clinical cohort study).[50,51] A cohort study done in adolescent female soccer players found an 88% reduction in ACL tears in the group participating in a preventive training program including stretching, strengthening, plyometrics, agility drills, and avoidance techniques (Level of Evidence B, clinical cohort study).[52]

After puberty, girls seem more prone to overuse injuries, particularly of the lower extremities. This is thought to be due to lower extremity malalignment (femoral anteversion, genu valgum,

external tibial torsion, hindfoot pronation) that is more prevalent in girls than boys and becomes more pronounced with the body changes of puberty in girls. An additional contributing factor is the increase in body fat that girls experience at puberty, in contrast to the increased muscle mass that occurs in boys. Increased body fat may also contribute to decreased performance and a less fit appearance, triggering extreme weight control behaviors and eating disorders.

Epiphyseal Injury

Children are at unique risk of injury to the epiphysis. It is estimated that 10% of all skeletal trauma in childhood involve epiphyseal injury.[53] Epiphyseal injury can lead to major skeletal growth disturbances such as limb length discrepancies and joint angle deformities; however, epiphyseal injuries associated with sports are usually not severe, and less than 5% of epiphyseal injuries result in subsequent growth disturbance.[54]

The epiphyseal plate is two- to fivefold weaker than the surrounding ligamentous structures. The epiphysis is particularly vulnerable during the period of rapid growth at puberty. For this reason, the incidence of epiphyseal injuries peaks during puberty in conjunction with the adolescent growth spurt. In the skeletally immature, acute physeal fractures rather than ligament injuries usually occur. As the skeleton matures, ligament injury is more likely. Therefore, injuries that would result in ligamentous damage in adults may cause epiphyseal fracture in children and adolescents with open epiphyseal plates. A common example is a valgus injury to the knee, which would result in a sprain of the medial collateral ligament in an adult but may cause epiphyseal fracture of the distal femoral epiphysis in adolescents. Similarly, inversion injury at the ankle, which results in the typical sprain of lateral ankle ligaments in adults, may cause a fracture of the distal fibular epiphysis in children. Similarly, injury to the wrist in children often results in distal radial epiphyseal fracture rather than the wrist sprains seen in adults. A slipped capital femoral epiphysis is a variant of epiphyseal fracture that presents as hip pain at the onset of the adolescent growth spurt, particularly in children who are obese or of large build.

Epiphyseal fractures are usually categorized according to the Salter and Harris classification scheme (Figure 18.4).[55] The majority of epiphyseal injuries in athletes are type I and type II injuries. If good reduction and adequate immobilization are achieved, type I and type II injuries rarely result in growth disturbances. Orthopedic consultation is advisable. Type III, IV, and V injuries have a higher likelihood of subsequent growth disturbance due to bone bridge formation across the physeal plate and often require surgical intervention to obtain appropriate alignment. These types of injuries occur infrequently in sports and are usually associated with trauma from falls or motor vehicle accidents. The possibility of epiphyseal injury should be suspected in any joint injury in a child with open epiphyses, particularly during the time of the adolescent growth spurt. The diagnosis of "sprain," implying ligamentous injury only, should be made with caution in the immature skeleton.

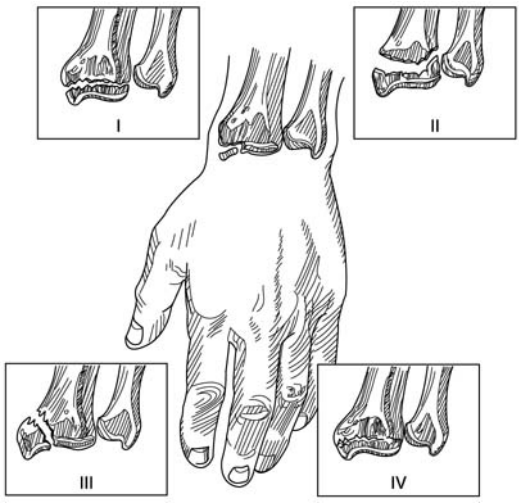

Figure 18.4 The Salter–Harris classification of epiphyseal fractures. A Salter V typically is not visualized radiographically. The epiphysis usually fuses in whole or in part with the metaphysis within 1 year.

The epiphysis is not only vulnerable to fractures as a result of macrotrauma but is also susceptible to overuse injury as a result of repetitive stress to the region. This is most likely to occur in children involved in intensive training programs with emphasis on a single sport and presents as persistent pain at the location of the physis. Common sites include the distal femoral epiphysis associated with breast-stroke swimming or water polo, and the proximal humeral epiphysis in baseball ("Little League shoulder") and tennis. These conditions usually respond well to temporary activity modification and lead to no long-term sequellae. Epiphyseal growth arrest and associated degenerative changes secondary to

sustained repetitive microtrauma are observed at two sites: the distal radius in gymnasts (Level of Evidence C, consensus expert opinion),[56,57] and the distal humerus in baseball pitchers.[58] Concern has been raised with regard to the potential of similar epiphyseal damage in the lower extremities due to repetitive impact of long-distance running in children. To date, such injury has not been documented. Similarly, studies of weight lifting in children have found no evidence of even subclinical epiphyseal microtrauma on the basis of bone scan[28] (Level of Evidence B, systematic review) or evidence of cartilage or connective tissue injury on the basis of various serum and urinary markers[29] (Level of Evidence B, systematic review). However, caution should be exercised with regard to the skeletal consequences of intensive training during childhood[59] (Level of Evidence C, consensus expert opinion) and participation in endurance events such as marathons[60] (Level of Evidence C, consensus expert opinion) and triathlons[61] (Level of Evidence C, consensus expert opinion), because long-term studies are lacking.

Apophyseal Injury

The apophysis is a center of ossification similar to an epiphysis, but it does not contribute to the long growth of bones. It is the site of attachment of muscle–tendinous units on bone and often is associated with a prominence of the bone at the site, such as the tibial tubercle at the knee or the epicondyles of the elbow. In children, the junction between the apophysis and the underlying bone has not completely ossified and is weaker than the muscle or tendon attached to it; therefore, tensile forces at this site result in injury to the apophysis rather than to the muscle–tendon unit (as would present as muscle strains in adults). Injury from both macrotrauma and microtrauma can occur at these apophyseal sites.

Sudden contractile forces during sports can result in avulsion fracture of the apophysis, in which a portion of the apophysis is pulled off the underlying bone with the muscle–tendinous unit still attached. Typically such injuries are associated with a sudden, painful popping sensation. Most commonly they occur around the hip and pelvis at the following sites: the iliopsoas insertion into the lesser trochanter, the sartorius origin at the anterior superior iliac spine, the rectus femoris origin at the anterior inferior iliac spine, the hamstring origin at the ischium,[62] the abdominal muscles attachment along the iliac crest, and the hip adductors attachment at the pubic ramus. Although associated with marked pain and disability, such injuries generally respond to rest and rehabilitation and rarely require surgical intervention, unless the avulsed fragment is large or significantly displaced.

In addition to acute avulsion fractures, overuse injuries of the apophysis also occur. They are often referred to as apophysitis and represent chronic repetitive traction at the site. These injuries can occur at all the aforementioned apophyses about the hip and pelvis, in addition to several other sites. Perhaps the four most common sites are at the calcaneus (Sever's disease), the tibia (Osgood–Schlatter's disease), medial epicindyle of the elbow, and the iliac crest. Iliac crest apophysitis occurs as a result of traction of the abdominal muscles at their site of insertion along the iliac crest. Fusion of the iliac crest apophysis occurs at about age 16 in boys and age 14 in girls. The condition presents as tenderness over the iliac crest and is exacerbated by activity requiring extension of the hip or extension and rotation of the back. It is frequently seen in association with hill running, throwing, rowing, and hurdling.

Articular Cartilage Injury: Osteochondritis Dissecans

Osteochondritis dissecans (OCD) refers to the development of an osteochondral fragment of articular cartilage at a joint surface. The fragment may be composed entirely of cartilage or have an osseous component of various sizes. The fragment may be *in situ*, partially detached, or completely detached. Although the etiology of OCD is most likely multifactorial,[63,64] it is thought that trauma may play a role.[63] OCD can occur as a result of acute macrotrauma to the joint surface, leading to fragment formation and chronic nonunion secondary to avascular osteonecrosis. In addition, growing evidence suggests that repetitive microtrauma to the joint surface may also contribute to the development of OCD. Osteochondritis dissecans usually presents during the second decade of life. The vast majority of cases occur at the distal femur, classically on the lateral aspect of the medial femoral condyle. The patella, the talus, and the capitellum of the humerus are other common sites. Initial symptoms are often vague and consist of pain, often related to activity level. If the fragment becomes detached, mechanical symptoms such as catching, locking, and joint effusion may develop. Because history and physical findings are usually nonspecific, the diagnosis is usually made on the basis of x-rays. Often special x-ray views, such as a notch view of the

TABLE 18.4
Osteochondroses

Femoral head (Legg–Calve–Perthes)
Capitellum (Panner's)
Vertebral endplates (Scheuermann's)
Tarsal navicular (Kohler's)
Metatarsal head (Freiberg's)
Lunate (Kienbock's)

knee, or advanced imaging techniques such as magnetic resonance imaging (MRI) or computed tomography (CT) are required to visualize the lesion. Prognosis and treatment depend on the age of the patient, degree of symptoms, and stability and location of the lesion. The goal is to achieve union of the fragment and restore the integrity of the joint surface. Nonoperative treatment is often successful in patients with open epiphyses, and spontaneous healing can occur with activity modification, protection, and perhaps immobilization. Surgical treatment is usually indicated for those who fail conservative treatment, have detached fragments, or are skeletally mature. Surgery involves either debridement to the fragment or internal fixation with drilling or bone grafting of the base.

The Osteochondroses

Osteochondritis dissecans should be distinguished from the osteochondroses. The osteochondroses represent a group of developmental abnormalities of ossification that can occur at any epiphysis and can involve the articular surface, secondary ossification center of the epiphysis, or the physeal plate. The most common of these are listed in Table 18.4. It is often difficult to distinguish osteochondrosis from normal variations in ossification on radiographs. Some osteochondroses, such as Legg–Calve–Perthes disease and Kienbock's disease, are thought to be due to avascular osteonecrosis. For other osteochondroses, such as Panner's disease of the elbow, the etiology is unclear but is thought to be due in part to stress applied to bone in which ossification is delayed, resulting in disordered endochondral ossification of the epiphysis.[65] In most cases, the condition is self-limited, and reconstitution of the ossification center will occur with evidence of radiographic healing and relief of symptoms.

GUIDELINES FOR EXERCISE PROGRAMS FOR CHILDREN

1. Children should participate in activities that promote physical fitness as well as acquisition of sports skills.
2. To encourage long-term participation, sports programs for children should emphasize personal involvement, variety, success, and fun rather than competition, regimentation, and winning.
3. Modification of equipment and rules should be made to suit the developmental level of the participants.
4. Safety should be a priority with regard to appropriate setting, equipment, protective gear, program design, and rules of play.
5. Prior to puberty, no appreciable differences exist between boys and girls in endurance, strength, height, or body mass, and they can compete in a coeducational setting on an equal basis.
6. Children are at increased risk for heat stress; appropriate precautions should be taken to ensure adequate hydration and acclimatization.
7. For optimal physical matching of children in contact sports, consideration should be given to body size and maturational stage in addition to chronological age (see Chapter 3).
8. The immature skeleton is uniquely vulnerable to injury to growth centers at the epiphysis, articular cartilage, and apophysis. Risk of injury is increased during periods of rapid growth, such as the adolescent growth spurt. To reduce risk of injury, it is prudent to decrease intensity of training and increase emphasis on stretching to improve flexibility during this period.
9. Pubertal changes in girls raise specific concerns. Beginning at puberty, female athletes are at higher risk of lower extremity injuries, particularly overuse injuries and anterior cruciate ligament tears of the knee. They are also at increased risk of engaging in extreme weight control behaviors and developing eating disorders. When intensive training is combined with dietary restriction, female athletes are at risk of delays in height, pubertal maturation, and bone density development.

Growth and maturation of girls engaging in intensive training should be monitored closely so that training intensity and dietary restriction can be modified if problems are recognized.

10. Early specialization in a single sport, intensive training, and year-round training should be undertaken with caution with regard to increased risk of overuse injury, psychological stress, and burn-out.
11. Prior to puberty, aerobic conditioning to improve endurance is of limited value. Similarly, strength gains due to strength training programs are small and unlikely to improve athletic performance. Children are more likely to improve sports performance by practicing the skills of the sports itself, rather than from additional aerobic or strength training programs.
12. Strength training for children should emphasize sets of low-resistance and high-repetition exercises utilizing appropriate-size equipment and supervision by qualified adults. No resistance should be applied until proper form is achieved, then 6 to 15 repetitions per set, 1 to 3 sets per session, 2 to 3 times per week, for periods of 20 to 30 minutes. Resistance or weight is increased in 1- to 3-lb increments only after 15 repetitions in good form can be demonstrated. Weight lifting, power lifting, body building, maximal lifts, and competition should be prohibited until growth and maturation are complete.

SUMMARY

Recreational and sporting activities for children are generally safe. Most injuries are minor and self-limiting; catastrophic injuries are rare. While youth are capable of high levels of training intensity and competition, enjoyment with acquisition of sport-specific skills is the appropriate goal for children's physical activities. Sound coaching, careful preparticipation assessment, regular preventive strategies, and effective injury management will maintain children's already high levels of health and reinforce a lifelong habit of fitness through exercise.

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19 THE OLDER ATHLETE

Ted D. Epperly and Steven Newman

INTRODUCTION

America has two very interesting processes occurring today. One is that the U.S. population is growing older (the euphemistically termed "graying of America"). The other is that Americans are becoming progressively more health conscious and exercising more. As these two processes overlap, an unprecedented number of elderly athletes (65 years of age or older) are lacing up their jogging or walking shoes, putting on their golf gloves, toning up, trimming down, and basically deciding to "use it or lose it."

That our society is aging is of little doubt. Demographers have relished pointing out that, in 1995, 34 million people were over age 65 (12.5% of the population) and that, in 2030, when all of the baby boomers have reached 65, the country will have 69 million elderly (20% of the total population). The average life expectancy is increasing, in part through better exercise and nutrition, so that persons reaching age 65 will have a life expectancy of 81.4 years for men and 85 years for women. The older population is itself becoming older, with those in the 65 to 74 age group being 3 times more numerous than the population of that age in 1900, those in the 75 to 84 year age group being 13 times greater, and the 85-plus age group (the fastest growing segment of our population) being 24 times more numerous.[1]

That America and elderly Americans are being encouraged to exercise more is also undisputed.

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The American Heart Association, President's Council on Fitness, American Association of Retired Persons, and U.S. Preventive Services Task Force are encouraging the elderly to become more physically active and to develop a program of physical activity tailored to their health status and life-style.[2,3]

Not only are the elderly responding to this call to athletic arms by becoming more active, but they are also becoming more competitive. The Senior Olympics and the Senior Professional Golf Tour are just two recent examples of this trend, and many more competitions for the elderly will emerge soon. Dramatic improvements have also occurred in most seniors- or masters-level world records in swimming and track and field.[4] The intensity and commitment to training and exercising that characterize many young adults is now being carried into the elderly years. Examples of these older athletic "All Stars" include Bobby Riggs, Chi Chi Rodriguez, Jack LaLanne, Satchel Paige, and Arnold Palmer.

PHYSIOLOGIC CONSIDERATIONS/BENEFITS

It is important to keep in mind that sometimes it is very difficult to determine which bodily changes are due strictly to aging and which are due to disuse. These physiologic changes probably result from a combination of both factors.

Muscle

Sarcopenia is a decline in muscle mass and strength that occurs with the normal aging process. All humans lose muscle mass and function as they age, even master athletes who are very active into advanced age.[5] The loss of muscle tissue is both quantitative (decline in myocytes) as well as qualitative (decline in strength). Sarcopenia is no doubt multifactorial, with neurological, hormonal, nutritional, metabolic, and physical-activity-related changes and other disease co-morbidities all playing key roles. As one ages from 30 to 80, muscle mass decreases in relation to body weight by about 30 to 40%.[1]

Most studies indicate that maximal strength measures peak in the third decade of life and plateau until about age 50, with a steady decline after that.[6] Muscle size and mass appear to decrease with aging; by age 65 to 75, men show a 20 to 25% decrease in quadriceps size by ultrasound scanning compared to men in their 20s.[4,6] Isometric strength of the quadriceps is also reduced by 39% in this elderly group.[4] Computed tomography (CT) scanning of arm and leg muscle size in young (25 to 38) and elderly (65 to 90) men demonstrated that the elderly men's muscles were smaller by 28 and 36%, respectively.[4] The lower extremity musculature declines at a faster rate than upper extremity musculature.[5] Other studies, however, have demonstrated little or no change in strength up to age 60.[4] An age-related decrease in muscle fibers and number of motor units has been observed, as well as a possible reduction in the number of Na–K pumps, which effect excitation–contraction coupling.[4]

Nonetheless, isometric and isokinetic strength and muscle mass in an older athlete can improve with a training program, and the training response is similar to that seen in younger men.[4,6,7] Aerobic capacity has also been shown to decline with age in active runners and swimmers, reflecting a decline in muscle and aerobic capacity.[5]

In sum, muscle mass and strength are lost slowly with aging. How much of this loss is secondary to disuse and how much is strictly age related is unclear, however. No doubt this loss can be minimized or even negated by continual use and exercise of the body's musculature, and improvement in strength and size of "old muscle" can be achieved.

Bone

Bone is a very dynamic tissue that is constantly undergoing deposition and reabsorption. The aging process slows the process of resorption and redeposition of the salts and protein in bone matrix, resulting in a weaker bone. Additionally, an age-related decrease in total body calcium occurs.[4] Bone increases in mass by radial deposition until about the age of 30. Bone mass then plateaus until about age 40 in both sexes, after which cortical bone loss occurs at approximately 0.3 to 0.5% per year in both men and women.[4,8] At menopause, bone loss accelerates in women, with 2 to 3% being lost per year for approximately 5 years after menopause.[6] Trabecular bone loss is more variable and independent of menopausal changes, with a loss of roughly 1.2% per year in men and women.[4] Trabecular bone loss precedes cortical bone loss by at least a decade in both sexes. At this rate, women may lose 30 to 35% of their cortical bone mineral mass and 50% of their trabecular mass by age 70.[4,6] The corresponding loss in men is about 20% of cortical and 33% of trabecular bone.[4]

Bone loss can be diminished by regular activity and exercise of the skeleton, in combination with good nutrition. Cross-sectional studies of athletes have demonstrated a larger bone mass and density

compared to age-matched sedentary controls.[4] Additionally, CT scans of the first lumbar vertebrae of runners with osteoarthritis and osteoporosis, when compared to age- and sex-matched controls who did not run, showed them to have maintained greater bone density than the nonrunners.[9]

Cartilage

Aging produces change in articular cartilage structure and function. Those changes primarily include smaller proteoglycan subunits and a loss of cartilage water content. These alterations decrease the elasticity of cartilage and may lead to an increased incidence of osteoarthritis. Joints postulated to be of greatest risk for this degenerative cartilaginous change include the knees, hips, ankles, and spinal facet joints.[8] Additionally, the small distal joints of the feet and hands are often involved. Multiple studies and papers have addressed the issue of degenerative arthritis or osteoarthritis and its association with running.[9–13] None of these studies has demonstrated evidence of premature osteoarthritis changes in patients studied, and it appears that running actually slows the rate of premature degenerative arthritis.[8] Clinically, if an older runner already has degenerative arthritic changes, the impact of running may accelerate the condition.[8] It is therefore prudent to suggest for these patients an alternative exercise regimen that does not have the same impact on the ankle, knee, and hip. Athletic endeavors that involve quick cutting, jarring, and impact motions, such as tennis, racquetball, and basketball, carry the most injurious forces to cartilage.

Ligaments and Tendons

Ligaments and tendons become less elastic secondary to a decrease in water content, predisposing the elderly to an increased risk of sprains and strains.[8] Decreased flexibility is common with both disuse and aging. A well-designed and regular stretching program before and after exercising is very important in the elderly and helps to maintain muscle flexibility, decreases the formation of excess collagen fiber cross-linkages, and helps to preserve full joint range of motion.[6] Lack of flexibility and decreased range of motion in the elderly increase the stress and force directly borne by the joint and predispose the musculature to tears.[6]

Nervous System

Aging produces a 37% decrease in the number of spinal cord axons and a 10% decline in nerve conduction velocity.[8] The sensory nerves are also more predisposed to neuromas and hypersensitivity secondary to weather changes.[8] Vision and hearing also decrease, as do reaction time and quickness. In fact, quickness is lost long before flexibility and strength.

Cardiovascular System

As one ages, maximum oxygen uptake (VO_{2max}) declines steadily, and anaerobic endurance decreases by approximately 35%, or 6.9% per decade.[8,14] Cardiac output similarly decreases by about 6 to 8% per decade throughout adulthood, and maximum heart rate declines by 3.2% per decade, or roughly according to the following equations:[1,8,14]

220 − age (years) = maximum heart rate (beats per minute) for men

190 − (0.8 × age [years]) = maximum heart rate (beats per minute) for women

Average 10-km run times slow by approximately 6% per decade.[4] The cardiovascular–pulmonary system, however, is exceedingly responsive to conditioning and with regular exercise, such as running, maximum heart rate decline can be slowed and VO_{2max} and endurance can be maintained.[8,15,16] Not surprisingly, it has also been demonstrated that a running or similar aerobic program is a better way to improve cardiovascular endurance, VO_{2max}, and physical capacity than is a weight-lifting program for trained, healthy elderly.[17] Cross-training in the elderly utilizing a combination of aerobic conditioning (such as running, swimming, cycling, or rowing) and weight lifting for potential additive results makes intuitive sense.

Regularly performed endurance exercise favorably modifies the lipid and lipoprotein profile in elderly male runners (66 years ± 5 years) by producing an increase in high-density lipoprotein cholesterol (HDLC) and thus may reduce the risk of coronary artery disease in the elderly.[18] Regular exercise, however, is not a panacea or vaccine against coronary artery disease, and the athlete's perception of risk reduction should be discussed.[19]

Kidneys

Glomeruli are lost as one ages, with a corresponding loss in kidney filtration, and a decrease in cellular and total body water predisposes the elderly to dehydration.[2] It is therefore recommended that the elderly move indoors in

TABLE 19.1
Sports Injuries in the Elderly

Diagnosis	Number	Percent (%)
Tendinitis	181	23.0
Patellofemoral pain syndrome	79	10.0
Osteoarthritis	73	9.3
Muscle strain	69	8.8
Ligament sprain	64	8.1
Plantar fasciitis	47	6.0
Metatarsalgia	45	5.7
Meniscal injury	39	5.0
Degenerative disk disease	34	4.3
Stress fracture/periostitis	29	3.7
Unknown	26	3.3
Morton's neuroma	22	2.8
Inflammatory arthritis	20	2.5
Multiple diagnoses	16	2.0
Vascular compartment	10	1.3
Bursitis	10	1.3
Adhesive capsulitis	8	1.0
Rotator cuff tear	5	0.6
Subcromial impingement	4	0.5
Achilles tendon rupture	3	0.4
Spondylarthritis of C-spine	2	0.25

Source: Data from Mathewson et al.,[22] Kannus et al.,[23] and DeHaven and Lintner.[25]

extremely hot weather and/or break up workouts into alternating sequences of jogging or cycling for 10 minutes and walking for 5 minutes.[2] Because the elderly are often not aware of dehydration because of blunted thirst, it is advisable to have them drink fluids before, during, and after exercising.

Psychologic Benefits

The psychologic benefits of regular exercise cannot be overemphasized. Older athletes who run and walk regularly have been found to be less tense, depressed, fatigued, angry, and confused and to have greater vigor, a more positive attitude, and higher self-esteem with regard to themselves and their physical fitness.[20,21] When asked why they exercised, 93% of the population over 55 years of age reported exercising to feel better.[22]

COMMON INJURIES

Injuries seen in the exercising elderly are almost exclusively overuse injuries (Tables 19.1 and 19.2).[22–25] Overuse syndromes account for 70 to 85% of the total injuries in these tables.[22,23] The knee is by far the most common injury site in the elderly, similar to injuries in younger athletes.[22–26] Most of these injuries are related to running or walking. The elderly tend to gravitate away from high-impact, contact, and team sports and settle on more individual sports that they can master and control. The reason for the increase in knee and foot problems in the older athlete is probably secondary to reduced strength and flexibility of the lower limb and thus decreased shock-absorbing capabilities of the knee and foot.[22] The clinician should examine the patient carefully to determine if the elderly athlete's symptoms are due to osteoarthritis or to an extra-articular soft tissue syndrome occurring in conjunction with radiographic evidence of osteoarthritis.[22] The elderly often take a wait-and-see attitude toward their injuries, as many of the overuse injuries are slow to develop;[23] therefore, they tend to present late, may be more recalcitrant, and have been treating

TABLE 19.2
Sites of Sports Injuries in the Elderly [22,23,24]

Location	Number	Percent (%)
Knee	237	31.0
Foot	139	18.0
Lower leg	78	10.1
Shoulder	68	8.8
Ankle	63	8.1
Lumbosacral spine	43	5.6
Multiple sites	43	5.6
Elbow	34	4.4
Hip/pelvis	31	4.0
Upper leg	20	2.6
Neck	11	1.4
Wrist/hand	7	0.9

Source: Data from Mathewson et al.,[22] Kannus et al.,[23] and Hogan and Cape.[24]

their injuries at home. Most of the older athlete's shoulder, tendon, and ligament problems are secondary to degenerative changes in combination with overuse.

DIAGNOSIS AND TREATMENT

Diagnosis can easily be made in 70 to 84% of injured older athletes with nothing more than a good history and physical exam.[22,23] The following questions are helpful and necessary to ask these elderly athletes: Are your symptoms aggravated by activity? Exacerbated by a pre-existing problem? Precipitated by a sudden change in intensity level, a single severe session, or trauma?[21] Further diagnostic aids such as a plain radiograph, CT scan, magnetic resonance imaging (MRI), or radionuclide scan are obtained when felt necessary. Consultation may be helpful in about 15% of cases.[22]

Because most of the elderly athlete's problems are overuse injuries, a conservative treatment policy is warranted. These problems, as in younger athletes, should respond well to PRICEMM (see Chapter 27). In treating the elderly with drugs, three caveats should be remembered. First, *start low and go slow*. Start any nonsteroidal anti-inflammatory drug (NSAID) or COX II inhibitor at a low dose and proceed slowly before raising the dose. Second, remember that the elderly are frequently on other medications, so check for potential drug–drug interactions. Third, remember that therapy of injuries in the elderly takes longer than it does for younger persons, so begin rehabilitation as soon as possible. A useful guideline is that treatment duration should be at least twice as long for an athlete 60 years or older than for a 20-year-old athlete, and three times as long for athletes older than 75.[2] A successful return to activity can be obtained if adequate treatment, rest, and rehabilitation are provided. One useful approach is to decrease activities by 15 to 25% of usual activities until symptoms disappear. Similarly, an increase in activities can gradually be returned in increments of 15 to 25% over 3 to 6 weeks, depending on the condition.

Physical therapy has much to offer the older athlete including ultrasound, diathermy, iontophoresis, and range of motion and stretching exercises. Simple, inexpensive items such as hand-held free weights and elastic rubber tubing can be used at home following simply written, printed instructions to increase patient compliance and reduce patient cost for rehabilitation. Muscle strengthening, particularly quadriceps strengthening, is crucial for knee-related problems and can easily be taught to the elderly. Orthotics and braces may also play a role for such problems as Achilles tendinitis, ankle instability, posterior tibial tendinitis, plantar fasciitis, and Morton's neuroma. If the above measures do not work, then local steroid injections and/or surgery may be necessary. Local steroid injection was used in 10% of the elderly seen for their sports-related injuries in one study.[22] Most sports-related injuries in the elderly are easily managed with a common-sense and conservative approach, with only 2 to 4% requiring surgery.[22,23]

Contributing factors and age-related changes that may contribute to athletic problems in the elderly include decreases in muscle mass, flexibility, reaction time, bone mass, and cartilage thickness and resilience, as well as impaired vision, hearing, proprioception, temperature regulation, and balance. A regular program of exercise and range of motion activities will help maintain muscle mass and flexibility. In return, muscle mass and flexibility will decrease musculoskeletal injuries. Problems with arthritis can subsequently be helped with reduced-weight-bearing activities such as swimming, stationary cycling, or elliptical training devices. Osteoporosis can be helped by a regular exercise program (full weight bearing). For elderly patients who suffer from poor vision, hearing, and balance problems, a stationary bicycle or elliptical training machine may provide good exercise while maintaining balance in a safe environment.

Exercise after myocardial infarction is in no way contraindicated; however, a program must be tailored to the individual's condition and should start with low-impact aerobic exercise (such as walking or stationary cycling) and should build up slowly in duration, frequency, and intensity as the patient demonstrates tolerance. Exercise after arthroplasty or joint replacement also is not contraindicated. However, limited-weight-bearing activity is preferred, such as water conditioning, stationary cycling, or elliptical training for endurance while decreasing joint stress.

PREPARTICIPATION SCREENING AND THE EXERCISE PRESCRIPTION

Because chronological age does not equate to physiologic function, it is extremely important that the physician individualize the exercise prescription to the needs and abilities of the particular older athlete being evaluated. The principles of exercise prescription are similar for people of all ages. The goal is to improve cardiovascular and muscular fitness.[27] The physician's role is to understand these principles and suggest an exercise plan that takes into account an individual's goals and physical abilities and to help keep the patient motivated and compliant. Many older patients, however, have some unique challenges that must be overcome in order for them to be compliant with an exercise prescription.

Preparticipation Screening

Before beginning an exercise program, older adults should have a complete history and physical.[27,28] In this physical, the physician should try to identify and modify any risk factors or physical conditions that could adversely affect the patient while exercising. There are very few absolute and relative contraindications to aerobic and resistance training for the older athlete. The absolute contraindications include recent electrocardiogram changes or myocardial infarction, unstable angina, third-degree heart block, acute heart failure, uncontrolled hypertension, and uncontrolled metabolic disease. Relative contraindications include cardiomyopathy, valvular heart disease, and complex ventricular ectopy. However, even patients with these contraindications can exercise at low levels once appropriate modifications have been made. Patients who are low risk and are asymptomatic are able to exercise without any further evaluation beyond a comprehensive history and physical exam. However, moderate risk individuals — men, ≥45 years of age and women ≥55 years of age or persons who have two or more risk factors, diabetes, or known or major symptoms of pulmonary or metabolic disease — should undergo cardiac stress testing before beginning vigorous exercise (>6 MET or exercising at >60% VO_{2max}). These individuals can engage in moderately intense exercise (3 to 6 MET or >40% and <60% VO_{2max}) without a stress test (Level of Evidence C, consensus opinion).[27] Frequently, information gathered in a stress test, even in low-risk individuals, can be used in establishing a safe and effective exercise prescription. The stress test can aid in assessing an individual's fitness level and defining an appropriate range of exercise intensity; however, most elderly patients can begin a low to moderate resistance and aerobic training program without a stress test if they begin slowly, and gradually increase their exercise load. Patients should be reevaluated if they develop any symptoms of concern during exercise. In general, a preparticipation screening electrocardiogram has limited use and is not required. For individuals with known cardiac disease, the 1995 American Hospital Association guidelines provide recommendations for participant and or patient monitoring and supervision and for activity restriction. Furthermore, the 26th Bethesda Conference provided recommendations for patients with hypertension, valvular, and other cardiovascular diseases (Level of Evidence C, consensus opinion).[27,29]

Many older athletes take medications that can cause adverse effects during exercise. While taking the medications should not stop an athlete from exercising, a provider or pharmacist should provide counseling about possible side effects. For example, diuretics can cause cramping and dehydration, beta blockers and calcium channel blockers can mask hypoglycemia, beta blockers can impair muscle uptake of glucose, insulin and sulfonylureas can cause hypoglycemia, psychotropic medications can cause hyperthermia and dehydration, and antidepressants and tranquillizers can cause orthostatic changes.[30]

The physical exam should focus on identifying any potential physical aliments that can be corrected and improved to make exercise more successful. Muscular strength should always be assessed. Most injuries are to the lower extremity, so assessment of quadriceps, knee, and ankle strength is important. Flexibility should also be examined with particular focus on the lower extremity. The heel should have at least 10° of dorsiflexion and the hip at least a 60° of arc motion. Furthermore, the modified Thomas test can be used to test for hip flexion contractures, illotibial band tightness, and rectus femoris tightness. Sensory testing should be performed on the feet for deficits in sharp/dull sensation. If a deficit exists, shoes with excellent shock absorption should be used along with meticulous care of the feet. Also, a check of hearing and vision should be performed. Finally, the feet, lower legs, knees, thighs, and trunk should be inspected for obvious problems. Any obvious or troublesome deformities may have to be corrected with orthotics before exercise begins.

Furthermore, part of the screening should include helping patients identify reasonable and realistic individual goals for exercise. For example, does the patient want to lose weight, increase conditioning, or modify risk factors for heart disease? It is important to determine what the patient wants out of exercise in order to write a successful prescription aimed at achieving those goals and to help keep them motivated. However, it is also important to identify realistic and attainable goals. It is not realistic for a 70-year-old to begin training for a marathon if he or she has never run before, but it is realistic to improve endurance, strength, and flexibility.

Exercise Prescription

The prescription includes three components: aerobic conditioning, strength training, and flexibility (Level of Evidence C, consensus/expert opinion).[27] The components of conditioning and strengthening are warm-up, intensity, duration, frequency, and exercise type/mode.[31] Flexibility can be included in the warm-up phase of strengthening and conditioning.

Aerobic Conditioning

Aerobic conditioning involves improving cardiovascular strength and conditioning. The five key components to aerobic conditioning are:

- *Warm-up.* Warming up should be performed with gentle stretches before exercise. This helps reduce exercise-induced musculoskeletal injures. In addition, light movements simulating the actual exercise should be performed.
- *Intensity.* Low- to moderate-intensity workouts are recommended for the older athlete because of cardiovascular advantages, lower injury rates, and improved compliance.[31] Moderate intensity is defined as exercise at 3 to 6 MET, 40 to 60% of VO_{2max}, 55 to 70% of maximal heart rate, 40 to 60% of heart rate reserve, or perceived exertion of 12 to 13 (somewhat hard) on the Borg relative perceived exertion scale.
- *Duration.* Moderate aerobic activity should be performed for a total of at least 30 minutes a day, most days of the week. Exercise up to 1 hour is even more beneficial. The total cumulative exercise time spent each day is more important than a single bout of exercise. Three 10-minute exercises work as well as one 30-minute exercise session. For older adults, it is recommended to exercise at low to moderate intensity for a longer duration of time, and duration should be increased as one progresses before intensity is increased.
- *Frequency.* Adults should perform moderate exercise on most and preferably all days of the week.
- *Type/mode.* The type/mode of exercise should be based on the patient's fitness level along with the patient's interest and available resources.[31] Furthermore, the ability to incorporate the exercise plan into daily activities has a higher success rate. Walking, gliding activities, swimming, cycling, and jogging are examples of typical successful activities for elderly athletes. Furthermore, cross-training is

becoming increasingly popular. Some advantages include fewer musculoskeletal injuries and less boredom.[27,31]

Strength Training

Strength training involves overcoming sarcopenia and restoring strength. The five key components in strength training are:[31]

- *Warm-up.* Warm-up should be similar to aerobic conditioning and should include flexibility training.
- *Intensity.* Elderly should start at low intensity and gradually increase over time. Weights should be lifted comfortably through a full range of motion slowly and smoothly with a focus on good posture and mechanics. Furthermore, individuals should avoid Valsalva maneuvers while lifting. The amount of weight to be lifted corresponds to approximately 60 to 80% of the maximum lift. Elderly athletes should focus on lower intensity of weights and higher repetitions.
- *Duration.* Usually, two to three sets of 8 to 15 repetitions are used in a workout routine.
- *Frequency.* Strength training is usually performed two to three times a week.
- *Type/mode.* Strength training should include all the large muscle groups (back, legs, hips, chest shoulders, and arms).

Flexibility

Flexibility involves stretching major muscle groups at least once per day when muscles are compliant.[27] This is usually incorporated into the warm-up phase of aerobic conditioning and strength training. Furthermore, stretching can also be repeated while cooling down from a workout routine while muscles are warm and more compliant. Patients should avoid ballistic stretches and focus on smooth prolonged stretching to avoid injuries to muscles. Improved flexibility is thought to decrease the risk of falls in the elderly.[31]

Promoting Exercise

One of the most difficult challenges of the exercise prescription is to keep patients motivated and compliant with the exercise plan;[30] however, the strength of the physician's advice is significantly correlated with the likelihood of adopting increased physical activity in patients.[28] Furthermore, if a physician is able to identify goals the patient desires and offers frequent praise and encouragement, compliance with an exercise prescription is more likely. Other key factors help with exercise compliance: Encourage elderly patients to exercise with a friend or family member or even a pet. Recruit family members to offer encouragement and assistance in training routines. Socializing is important for many elderly. Activities that involve exercising with other cohorts help with compliance. Variety in an exercise program is important for preventing boredom. Cross-training cannot only increase variety but also helps to reduce over-use injuries. Physical factors are also important. Patients should feel safe while exercising. The environment should be free of obstacles, well lit, and void of extreme environmental conditions. Encourage activities that are affordable such as walking in a park or a mall. Incorporate physical activity into daily routines and build on previous activities; for example, move a treadmill in front of the television. Finally, suggest activities that are enjoyable. All of these suggestions can help overcome the high rate of non-compliance in the elderly athlete.[28]

NUTRITION

Nutrition for the older athlete is also very important and should not be overlooked by the physician. In fact, nutritional advice for many older athletes may have a greater impact on improving general health than on maximizing athletic performance.[3] Nutrition for the exercising elderly can be broken down into five areas: (1) energy (calories), (2) macronutrients (protein, carbohydrates, fat), (3) vitamins, (4) minerals, and (5) water.[3]

Calories

The average young adult's diet consists of approximately 1700 kcal/day for women and 2700 kcal/day for men. This caloric need decreases in those over 75 to approximately 1400 kcal/day for women and 1800 kcal/day for men. Because balancing caloric intake with caloric expenditure is the key to weight control, a diet of 1000 to 1800 kcal/day with increased exercise will result in roughly 1 lb/week of weight loss in the elderly.[3] Diets below 800 kcal/day are not associated with maintenance of the weight loss and are not recommended for the obese elderly.[3]

Macronutrients

Caloric balance in the elderly is the same as that recommended for the general population. Carbohydrates should account for 50 to 60% of calories, proteins for 10 to 20%, and fats for no more than 30%. If the elderly athlete is involved

with prolonged endurance activities (e.g., marathon running), then the carbohydrate intake should increase to 60 to 70%.[3] Older athletes will achieve the recommended dietary allowance (RDA) of 0.8 g/kg of body weight of protein per day through usual dietary habits. Some evidence demonstrates that this intake may be increased to 1.0 to 1.5 g/kg of protein per day for vigorously exercising older athletes, but the 0.8 g/kg/day will suffice.[3] Diets consisting mainly of vegetables will not meet the exercising elderly person's protein requirements and must be supplemented.

Vitamins

The exercising elderly are unlikely to need vitamin supplements if eating a well-balanced diet. The myth that vitamins will enhance performance, prevent injury, or improve recovery time should be broken. In fact, toxicity of excess vitamins, particularly hepatic toxicity of vitamin A and in some cases of even the water-soluble vitamins, can occur in the elderly.[3]

Minerals

A balanced diet will meet most of the exercising elderly's mineral needs. Iron and calcium may sometimes have to be supplemented. The RDA for iron in elderly men and postmenopausal women is 10 mg/day. If the elderly are involved in distance running, that requirement may increase to 18 mg/day and supplementation may be necessary.[3] The RDA for calcium is 800 mg/day in the elderly. If the older athlete is not eating or drinking calcium-rich foods (yogurt, cheese, milk, broccoli), then supplementation with 1000 to 1500 mg/day to help support bone mineral content will be helpful.

Water

Dehydration is the most common fluid and electrolyte problem seen in the elderly.[1] Drinking adequate amounts of water before, during, and after exercise is important to prevent dehydration and to help with temperature regulation in the elderly. Two cups of water before the activity and 300 cc (10 oz) every 20 minutes thereafter will help control core body temperature and decrease dehydration, which is the primary cause of muscle cramps in the elderly.[3]

ENVIRONMENT

The environment is often overlooked as a factor that can cause problems with exercise for the elderly. Heat, cold, high relative humidity, strong winds, and high altitudes can adversely affect exercise. In addition, the elderly often have risk factors that can make them less tolerant of environmental hazards. The use of diuretics or psychotropic medications, low-sodium diets, impaired thirst drive, poor conditioning, and sarcopenia can cause the elderly to be more susceptible to the environment.[32] For example, exercise in heat and high relative humidity along with the use of diuretics can easily lead to dehydration. It is important to educate patients about these risks and offer suggestions about alternative exercise plans. For example, walk inside a mall during extreme heat or cold, or garden in the early morning hours rather than in the middle of the day. This will make the activity not only safer for the patient but also more enjoyable. A more detailed discussion of these factors can be found in Chapter 10.

SUMMARY

That America is aging and that the level of exercise in the elderly is increasing is undisputed. For many years, society supported the myth that the elderly should not be active and athletic. Studies demonstrate that the elderly person's musculature and cardiovascular status can adapt to and improve performance with exercise. The physician's responsibility is to evaluate a potential older athlete thoroughly with a pre-exercise screen; appropriately prescribe and direct a slowly progressive, individualized program of training that includes conservative treatment and adequate rehabilitation for overuse injuries; and educate the patient with regard to preventive strategies (nutrition, safety, and special precautions).

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20 THE ATHLETE WITH CHRONIC ILLNESS*

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INTRODUCTION

Medical problems are responsible for approximately 70% of the visits that athletes make to physicians. The majority of these are respiratory and other episodic infectious illnesses; however, a significant number of athletes have one or more chronic medical conditions. For individuals over age 35, the risk of known and occult disease increases substantially. Important to the care of these patients is an understanding of how sports activity affects their illness. The corollary to this concern is that the illness may also affect strength, endurance, coordination, or other parameters that can impact sports performance. The physician's ultimate goal becomes maximizing the athlete's ability to pursue sport safely and successfully.

While athletes potentially experience any of the diseases seen in the non-athletic population, certain conditions occur frequently enough that physicians should have specific knowledge of the effect exercise has on disease management and participation risks. Individuals with obesity, hypertension, coronary artery disease, and diabetes mellitus benefit from physical activity, but sport poses risks for each condition. These disorders occur in a significant portion of the population, including athletes. Exercise-induced asthma affects numerous athletes, particularly those in winter and endurance sports. Seizure disorders alter consciousness so that safe participation in water sports, vehicular activity, equestrian sport, and other special situations cannot be assured even with treatment. This chapter reviews these common medical conditions and how best to care for athletes affected.

HYPERTENSION

The *JNC VI*[2] defines high blood pressure based on population demographics. Individuals above 90% for adjusted norms are considered to have high normal pressures. Those above 95% are classified as having hypertension, and severe hypertension exists in those with levels above 99%. This type of classification takes into account that pediatric patients and younger adults merit special assessment and may be diagnosed as hypertensive with much lower pressures than those seen in adult patients. For an adult, a resting blood pressure of 140/90 or above on a consistent basis (usually three or more measures) with an appropriate-sized cuff confirms the diagnosis of hypertension (Level of Evidence C).[2]

Physical activity increases the heart rate and cardiac output. Blood pressure varies in relation to cardiac output and total peripheral resistance. Exercise generally causes some increase in pressure levels because peripheral resistance rarely decreases enough to counter the effect of tachycardia and greater blood flow. Resistance (isometric) and dynamic (isotonic) exercise trigger different pressor responses. Typically, heavy resistance activities cause dramatic rises in blood pressures. Dynamic exercise leads to gradually higher blood pressures during activity, but the

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overall effect of continuous training is to lower both systolic and diastolic pressures. A meta-analysis of 54 clinical trials of aerobic exercise showed an average drop of about 10 mmHG in each parameter with ongoing training (Level A).

One special concern for the hypertensive athlete is the effect of training on overall blood pressure. While it is agreed that most resistance training has a long-term beneficial effect on blood pressure, dramatic blood pressure elevations in given athletes have led to a cautious approach. Currently, the American College of Sports Medicine (ACSM) recommends that resistance training in hypertensive patients should be conducted as circuit-training sessions so that a mix of resistance and aerobic activity occurs (Level of Evidence C).[4] Aerobic activity generally benefits blood pressure, with recommendations for low-intensity warm-up followed by moderate-intensity training at 55 to 70% of maximum heart rate. Because competitive athletes will need to spend some of their training in high-intensity workouts, periodic monitoring of blood pressure during training and competition helps determine whether control is adequate. Established hypertensive patients may experience a drop in exercise capacity of 16 to 30% (Lim) without adequate treatment, and good control improves performance.

A second concern for the hypertensive athlete is finding an effective medication with a side-effect profile that will not hinder sport performance. Angiotensin-converting enzyme inhibitors (ACEs) have become the drug of choice for most athletes, as they effectively control pressures during exercise with minimal side effects (Level of Evidence C).[1] Calcium channel blockers and angiotensin-II receptor blockers control exercise pressures equally well and also have limited adverse effects. Beta blockers and diuretics are first-line therapy for hypertension but pose specific risks for athletes that limit their use in this population. Beta blockers limit maximum performance and generally are avoided in asthmatic athletes. Diuretics may reduce maximal exercise capacity and increase risks of dehydration, cramps, and heat illness. Alpha blockers, central alpha agonists, and direct vasodilators are not primary choices for antihypertensive medications in athletes. Concomitant nonsteroidal anti-inflammatory drug (NSAID) use may limit effectiveness of antihypertensive agents and also increase the risk of side effects.

A serious concern for hypertensive athletes or athletes who have any major cardiac risk factor is occult coronary artery disease. Coronary artery disease often occurs in conjunction with hypertension, diabetes mellitus, obesity, and/or hyperlipidemia. Silent myocardial ischemia and infarction occur in a significant portion of men and women with high blood pressure. Because athletes place extreme physical demands on their cardiovascular systems, an exercise tolerance test becomes an important test to determine the likelihood of coronary artery disease and can also measure the blood pressure response to exertion.

CORONARY ARTERY DISEASE

Athletes with known coronary artery disease merit close monitoring for sports participation. While individuals have completed extreme exercise, including marathon runs, after a myocardial infarction, balloon angioplasty, or coronary artery bypass grafting (CABG), rarely would a physician recommend intense exercise. Because patients with known coronary syndromes can reduce their risks by maintaining high fitness levels, moderate exercise improves their prognosis. Patients with coronary artery disease who can achieve more than 10.7 MET of work load on standard exercise testing have a normal age-adjusted mortality (Level of Evidence A).[5] Careful adjustment of cardiac medications, including deciding when pre-exercise nitroglycerin should be utilized, improves the ability of these individuals to exercise safely. The current view of coronary artery disease is that this is a dynamic process. Research shows that plaque instability, platelet adhesiveness, epithelial responsiveness, and intravascular nitrous oxide levels all influence the likelihood that a specific lesion will lead to a myocardial infarction. With the myriad of possible triggers for acute coronary syndromes, specific research focuses on determining the effect of exercise on each of these parameters. Currently, our best approach to secondary prevention of cardiac events centers around appropriate drug treatments, including aspirin, lipid-lowering agents, beta blockers, ace inhibitors, and other individualized medications. Because we expect patients with coronary artery disease to exercise, yearly monitoring through the exercise tolerance test (ETT) seems reasonable. Frequency of testing increases if the athlete shows any signs of instability with his or her disease.

OBESITY

Based on National Heart, Lung, and Blood Institute (NHLBI) data published in 1998, an estimated 97 million U.S. adults are overweight or obese (Level of Evidence B).[7] Obesity contributes to

excess morbidity from hypertension, type 2 diabetes, coronary artery disease, stroke, gallbladder disease, osteoarthritis, respiratory difficulty, and sleep apnea. In addition, multiple cancers have higher incidences in the obese, including endometrial, breast, prostate, and colon. Not surprisingly, higher body weights are associated with greater all-cause mortality. Centripetal obesity with a high waist-to-hip ratio indicates a subtype of obesity with an independently elevated level of risk, particularly for cardiovascular disease (CVD) (Level of Evidence A).[8]

Many obese athletes have achieved the highest pinnacles of athletic success. Some sports have such a focus on size that obesity has traditionally been considered advantageous, including football, weight throwing in track and field, heavyweight wrestling, and power lifting. Rather than the athletic activity leading to a healthier lifestyle, sports with too much emphasis on size and strength have often encouraged dietary excess and other harmful practices such as using anabolic agents. Deaths from heat illness are more common in obese athletes. Fortunately, most athletic programs have redirected their focus to discourage obesity and to look more critically at lean body mass.

A more common problem confronting athletes is post-competition weight gain. An average professional football lineman weighing 300 pounds can rapidly develop to morbid obesity if he ceases activity and continues the same diet. Highly competitive athletes consume 1500 to 2000 excess calories a day to replace the expenditure from multiple training sessions. Considering that the body requires approximately 10 calories per pound to maintain body weight, a 300-pound athlete could be consuming an average intake of 4500 to 5000 calories daily. Even if this individual persists in exercising at a level considered vigorous by most Americans, without a marked reduction in calorie intake he could stand to gain 30 to 50 pounds in his first year out of serious training.

With this in mind, sound nutritional practices must be implemented during the competitive life of most athletes. Focus should be toward ideal weight based on body fat, not on total pounds. Simple waist-to-hip ratios can identify individuals who have higher risk. Athletes need to be informed about the calorie demand of various training activities so they do not overestimate the impact on calorie need of a low-intensity workout. Strategies should teach healthy eating and avoidance of fast foods to establish patterns that lead to better post-competition diet practices. Data from the Harvard Growth Study, with a follow-up period of 55 years, indicate that overweight adolescents have a higher risk of all-cause mortality and other adverse health effects independent of their adult weight (Level of Evidence A).[6] This emphasizes the importance of sound weight management starting at the middle school and high school sports participation level.

DIABETES IN THE ATHLETE

Regular exercise has been shown to improve glycemic control, increase self-esteem, and decrease the incidence of cardiac disease in those with diabetes mellitus. Close monitoring of blood glucose and appropriate adjustments in medications and diet aid in optimizing performance and preventing complications associated with exercise. Diabetes mellitus affects 16 million Americans, with approximately 10% having type 1 diabetes. Type 1 is characterized by autoimmune destruction of insulin- producing beta cells in the pancreas and these diabetics require a specialized diet along with exercise. In addition, exogenous insulin administration is needed to utilize glucose. Type 2 diabetes mellitus affects 3 to 7% of people in western countries and accounts for 90% of diabetes. In addition to impaired production of insulin, type 2 diabetes is characterized by reduced sensitivity to insulin and excessive production of glucose by the liver. Eighty percent of type 2 diabetics are obese. Although they may be able to control their blood glucose by diet alone, type 2 diabetics often require oral medications or exogenous insulin.

As in the general population, diabetics who exercise enjoy several benefits, including improved fitness levels, which correspond to longevity and decreased risk of cardiovascular death. Studies also show that diabetics who exercise have lowered blood pressure and improvement in hyperlipidemia. Exercise is considered a cornerstone in the control of diabetes. Although studies have failed to show a consistent reduction of hemoglobin A1C or fasting blood glucose with exercise, improvements have been found in insulin sensitivity and glycogen utilization. Additional benefits of exercise include increases in psychological well-being and self-esteem.

Risks of exercise for the diabetic include hypoglycemia, hyperglycemia, and neuropathy. Hypoglycemia (defined as blood glucose <65 mg/dL) is most common in those requiring exogenous insulin and can lead to fatigue, confusion, and coma. Exercising with an elevated blood

glucose may cause a rapid rise in glucose and ketoacidosis, and fluid loss can ensue. Peripheral neuropathy can cause ulceration and infections in the feet of diabetics who lack adequate pain sensation.

Every athlete with diabetes needs to have a thorough medical evaluation that includes history, physical exam, and appropriate laboratory tests. The history should elicit information about the degree of glycemic control, any diabetic complications, additional medical problems, and plans to change exercise regimens. This is also an excellent opportunity to gauge the patient's understanding of the disease and provide education.

The physical exam should focus on the cardiovascular, ophthalmologic, and neurologic systems. An ETT is important for detecting silent coronary disease. Diabetics should undergo exercise testing if they meet any of the following criteria: (1) age greater then 35 years, (2) type 1 diabetes for longer than 15 years or type 2 diabetes for longer than 10 years, or (3) presence of additional cardiac risk factors, autonomic neuropathy, or peripheral vascular or microvascular disease (Level of Evidence C).[17] In addition to detecting coronary disease, an exercise test can aid in the diagnosis of hypertension or orthostatic hypotension and can establish a baseline fitness level.

The ophthalmologic examination must include a funduscopic examination to assess for diabetic retinopathy. If proliferative retinopathy is present, isometric exercises should be avoided (Level of Evidence C).[11] Other eye diseases to be assessed include cataracts and glaucoma.

A thorough neurologic and foot exam is important to assess for peripheral neuropathy and signs of infection. Blisters and abrasions can cause ulcerations and serious infections in diabetics with neuropathy. In addition, Charcot deformity may develop in patients with severe peripheral neuropathy. Appropriate footwear, evaluation of the need for othoses, and frequent examination may help prevent complications.

Controlling glucose levels during exercise is optimized by adjusting the intensity of exercise, carbohydrate intake, and insulin or medication dosage. For type 1 diabetics, additional carbohydrate snacks may be all that is needed to supplement metabolic needs. A 15- to 30-g, readily absorbable carbohydrate snack should be consumed for every 30 minutes of exercise. For prolonged exercise, a reduction in insulin may be required. Decreasing short-acting insulin 30 to 50% within 2 to 3 hours of exercise may help to prevent hypoglycemia. Short-acting insulin can be reduced by 30% for exercise less than 1 hour, 40% for 1 to 2 hours of activity, and 50% for more than 3 hours of activity. If the athlete uses an insulin pump, a reduction in the basal rate of 50% may be necessary, while a 50% decrease in the pre-meal bolus may be necessary for diabetics exercising after meals (Level of Evidence C).[11]

Type 2 diabetics who are diet controlled rarely need any adjustments for exercise. Because of increased glucose utilization, the dose of oral hypoglycemic medication may have to be reduced by 50% or more on days of extended exercise. Type 2 diabetics, likewise, should have quick access to carbohydrate snacks to prevent hypoglycemia.

Prevention of hypoglycemia requires close monitoring of blood glucose before, during, and after exercise. Those at greatest risk for exercise-induced hypoglycemia are type 1 diabetics and type 2 diabetics who inject exogenous insulin. Carbohydrates should be given to a diabetic with a glucose level below 100 mg/dL at the start of activity.

Athletes who exercise with hyperglycemia (blood glucose >240 mg/dL) are at risk of rapidly rising glucose levels, dehydration, and ketoacidosis. Exercise should be avoided if the blood glucose level is greater than 300 mg/dL or if greater than 250 mg/dL and ketones are present (Level of Evidence C).[17]

The athlete with diabetes is able to compete at the highest levels. Close monitoring of glucose levels to establish glycemic patterns, adjustment in medication and food intake, and being familiar with potential complications will allow the diabetic athlete to compete effectively and safely.

EXERCISE-INDUCED ASTHMA

Respiratory disease can significantly impair athletic performance, particularly during sports with high aerobic demand. Exercise-induced asthma (EIA) affects up to 20% of all athletes, approximately half of whom are not known to have asthma. For individuals with chronic asthma, 90% or more will have flares with sporting activity, and as many as 40% of individuals with allergic rhinitis have EIA symptoms. The diagnosis of EIA may be subtle, and delayed treatment can lead to suboptimal performance.

The ACSM defines EIA as acute, reversible lung airway narrowing that occurs during and/or after physical activity (Level of Evidence C).[31] Several theories as to the etiology of EIA have been proposed in the medical literature but the specific

causes have not been identified. As with chronic asthma, a variety of stimuli may trigger airway hyperactivity and bronchospasm. Among these are environmental and/or local airway conditions that are potentiated by inflammatory mediators including leukotrienes, histamine, and neutrophil chemotactic factor.

Current research indicates that the epithelial osmotic gradient produced by airway dehydration is an important contributor to EIA. This exceeds the importance of airway cooling or airway hyperemia, both of which were identified in earlier research as primary factors. In addition, some preliminary studies implicate nitric oxide and adenosine as more integral components of the inflammatory formula of EIA than traditional mediators such as histamine and the leukotrienes.

The incidence and prevalence of EIA have been shown to increase in athletes participating in certain sports. Sports requiring high minute ventilation levels and/or cold, dry air exposure (such as long-distance running, cross-country skiing, ice skating, soccer, rugby, and wrestling) are particularly asthmogenic. One study of cross-country skiers found that 79% of them had asthma symptoms (Level of Evidence C).[18] Urban athletes who have more exposure to air pollution also have more EIA attacks due to higher levels of airborne pollutants, especially ozone. Physicians treating winter sport or urban athletes should maintain a high suspicion for EIA (Level of Evidence B).[23]

Clinical suspicion of EIA arises from specific symptoms. Cough is the most common symptom, followed by chest tightness, dyspnea, wheezing, chest pain, and fatigue. More subtle signs include chest congestion, side stitches, lack of energy, inconsistent performance, frequent colds, a feeling of being out of shape, and better performance during short rather than long exercise sessions. An athlete's only symptom may be poor performance on days with cold, dry air.

Typically, symptoms of EIA appear 6 to 12 minutes after the start of exercise and peak 5 to 15 minutes after they begin. Inducing EIA symptoms requires the athlete to achieve greater than 85% of maximum predicted heart rate and maintain this level of exertion for at least 4 minutes. Environmental, sport, and athlete characteristics may influence these parameters. If untreated, the majority of EIA attacks resolve spontaneously 30 to 90 minutes after exercise ends.

Sometimes a second exercise challenge does not provoke symptoms in an EIA patient and a "refractory period" occurs. One theory purports that a relative depletion of inflammatory mediators and correction of local airway lessens the degree of bronchospasm in the refractory period. During this phase, less than half of the original degree of obstruction will occur. Knowledgeable athletes may take advantage of this relative protection but only about half of patients experience this change and it is not predictable. Because the refractory period is unique to each individual, it should be defined for each athlete before incorporating this strategy into training or competition. A typical approach is for the athlete to complete a prolonged warm-up to induce EIA symptoms. If, after recovery, the athlete feels normal, he or she can compete without medication. The refractory period ordinarily disappears in nearly all athletes after 2 or 3 hours. Even in athletes with a refractory period, late-phase and recurrent attacks can occur without another provocative bout of exercise.

Definitive diagnosis of EIA requires a standard exercise challenge to demonstrate at least a 15% reduction in pulmonary function tests compared to pre-exercise values. After the challenge, repeat pulmonary function evaluation may be done with spirometry or peak expiratory flow rate (PEFR) measurements. PEFR testing is more convenient and cost-effective for sideline or training room evaluation but has less diagnostic accuracy. Normal resting pulmonary function tests do not rule out EIA. Because athletes can have a variable response depending on environmental conditions, repeat evaluations may be required.

An exercise challenge can be performed either in a controlled, laboratory-like setting or in the environment where the athlete competes. Debate exists as to whether testing in either site yields higher diagnostic accuracy. As long as the athlete's cardiopulmonary system is stressed to achieve and maintain at least 85% of predicted maximum heart rate, the test is considered adequate. Some pulmonologists consider the methacholine inhalation challenge a gold standard for diagnosing EIA, but others feel that this overestimates bronchial hyperresponsiveness. When symptoms have been induced, spirometry or PEFR testing should be performed at 3- to 5-minute intervals for 20 to 30 minutes. Mild, moderate, or severe EIA is present when a reduction of 20 to 30%, 30 to 40%, or greater than 40% occurs, respectively (Level of Evidence C).[32]

Preventive strategies should precede medical treatment of athletes with EIA. Education of the athlete with EIA forms the cornerstone of effective treatment. Masks, modification of activity, or

changes of ambient conditions are helpful for sensitive athletes. Initial pharmacologic therapy is with a beta$_2$ agonist; an inhaled, short-acting medication is given 15 to 30 minutes prior to activity based on pre-exercise PEFR values and athlete symptoms. Athletes with classic EIA, without another complicating condition, may require only this preparticipation treatment (Level of Evidence C).[33] The majority of patients with EIA, however, have either allergic rhinitis or chronic asthma with exercise-induced exacerbations and require daily medical therapy. Acceptable agents for treating athletes with allergic rhinitis include intranasal corticosteroids and non-sedating antihistamines. Chronic asthma patients may benefit greatly from daily use of an inhaled corticosteroid, the most effective suppression agent, or a long-acting beta$_2$ agonist. Leukotriene-modifying agents, such as zafirlukast and montelukast, are also very effective as control agents when used daily. Older agents, such as necrodomil and cromolyn sodium, are useful but have largely been replaced by the newer, more potent medications mentioned previously. Oral and parenteral corticosteroids are reserved for treatment of refractory asthma exacerbations.

Non-traditional therapies are emerging with promising data from preliminary studies. Inhaled heparin has been used to evaluate the pathophysiology of EIA and has been shown to significantly block symptoms of EIA through its effect on mast cells. Inhaled furosemide has also been shown to attenuate the symptoms of EIA. Although evidence is inconclusive, furosemide appears to exert a local effect at the lung epithelium. The rheumatoid arthritis disease-modifying agent methotrexate has demonstrated effectiveness as a suppressive agent for chronic asthma. Inhaled indomethacin and vitamin C have both shown promising results in experiments using EIA patients.

Exercise-induced asthma is not a contraindication to athletic participation. Athletes can compete and succeed in their desired sport with proper monitoring and treatment. Historically, Olympic athletes with EIA have performed as well as or better than their peers. Educating athletes to self-monitor their symptoms and to adjust treatment regimens based on symptom severity, season, and demands of the sport are effective management strategies. Also, coaches can maximize athletic performance by modifying the training program to suit the athlete's symptoms. These tactics allow success for athletes with EIA.

SEIZURE DISORDERS

A seizure is defined as an abnormal electrical discharge of cortical neurons leading to sudden involuntary alterations in movement, perception, or behavior. Epilepsy refers to a disorder of recurrent seizures. Approximately 2 to 3% of the population suffers from epilepsy, with another 10% of the population having a seizure at some point in their lifetime. Seizure disorders are common in the overall population, including athletes. Although few strict guidelines exist for the participation of epileptics in athletics, some commonsense recommendations can allow for safe participation.

Seizures are classified according to their characteristics and are separated into two major categories: partial and generalized. Partial seizures localize to a specific part of the brain, leading to jerking or twitching of a limb; disturbances of sensation, smell, or vision; or psychological symptoms. Simple partial seizures cause no loss of consciousness, whereas complex partial seizures do ultimately impair consciousness. Both types of seizures can evolve into secondary generalized seizures.

Generalized seizures involve cortical discharge from both cerebral hemispheres leading to bilateral symptoms and loss of consciousness. The most common type of generalized seizure is the tonic-clonic, or grand mal seizure. Absence seizures are a subtype of generalized seizures consisting of blank staring spells with no associated memory of the seizure. Other generalized seizures are myoclonic, clonic, tonic, and atonic.

While most epilepsy in children is idiopathic, known causes of childhood epilepsy include birth and neonatal injury (58%), central nervous system (CNS) infection (15%), head trauma (12%), and, rarely, metabolic disorders and tumors. In adults, epilepsy generally results from vascular insults (60%), tumors (10%), and CNS infection (9%).

Regardless of the cause of epilepsy, certain inciting factors are known to decrease seizure threshold and increase seizure frequency. Some of these factors include fatigue, sleep deprivation, dehydration and overhydration, stress, hyperventilation, hypoxia, hyperthermia, and hypoglycemia. Although some of these factors occur with activity, exercise has not been shown to increase seizure frequency. Multiple studies have shown that seizures rarely occur during exercise and overall seizure control improves with exercise. How exercise decreases seizure activity is still debatable. Current theories are based on B-endorphin release, lowered blood pH with lactic acid

release, increased gamma-aminobutyric acid (GABA) concentration, and possibly increased mental alertness and attention. A few studies have shown that seizure activity can increase in the post-exercise rest period.

Although significant head trauma can lead to seizure activity, no data have shown that repetitive minor head injuries cause any deterioration of seizure control. In a study of 301 children including those with epilepsy, blunt head trauma was not shown to worsen seizure control in epileptics.

The treatment of epilepsy is limited to antiepileptic drugs (AEDs), such as phenytoin, carbamazipine, valproic acid, lamotrigine, gabapentin, and clonazepam. With AEDs, about one half of epileptics remain seizure free. AEDs unfortunately can have considerable side effects, such as fatigue, sedation, nystagmus, ataxia, confusion, and nausea. Many of these side effects are undesirable and can be dangerous for athletes.

Overall, few guidelines for epileptic participation in sports exist. In 1968, the American Medical Association (AMA) advised that individuals with a "convulsive disorder not completely controlled by medications" be banned from collision sports, contact sports, and even some non-contact sports such as tennis. The discovery of the beneficial effects of exercise on epilepsy led to the AMA changing their stance by explaining that epileptics with reasonable control of seizures should be allowed to play any sport not proven to cause chronic head trauma. In 1983, the American Academy of Pediatrics (AAP) went a step further, declaring that "epilepsy *per se* should not exclude a child from hockey, baseball, football, basketball, and wrestling" (Level of Evidence C).[25] Exercise is currently recommended for most well-controlled epileptics to improve overall fitness and self-esteem. Some common-sense guidelines should be used to determine if participation is safe. Specific sports that involve heavy blows to the head, such as boxing and martial arts, are contraindicated. High-risk sports that may pose risk of injury or death to epileptics or others, such as archery and riflery, are considered by many to be too dangerous.

Aviation sports, mountain climbing, and water sports should be considered only for very well controlled epileptics under adequate supervision. Studies show a fourfold higher risk of drowning in epileptics vs. the general population when water sports are unsupervised. Sports such as gymnastics and horseback riding, which have the potential for injury from a high fall, should also only be considered in well-controlled epileptics. If any of these activities are pursued by the epileptic, supervision must be present at all times.

Motor sports are contraindicated in epileptics who have at least one to two seizures per year; however, epileptics who are well controlled and seizure-free for at least 2 years are eligible for a driver's license and can be considered eligible to participate in motor sports. Eligibility should be based on excellent seizure control, the safety of the driver, and the comfort level of the physician, driver, and other participants.

To lessen the risk of participation, epileptics should be encouraged to use appropriate equipment and follow the rules of safe play. If during the sport major head trauma occurs or seizure frequency increases, the epileptic's participation should be discontinued. If a new seizure occurs in a participant involved in contact sports, that person should undergo a thorough work-up, observation period, and possibly treatment before returning to the sport.

A more important issue may be that few epileptics actually participate in exercise. One survey of 3000 NCAA Division I athletes discovered only one athlete, a gymnast, with epilepsy. Estimates suggest less that 5% of epileptics participate in a regular exercise program. Often coaches, parents, physicians, and epileptics themselves limit involvement in sport out of fear of uncontrolled seizures, embarrassment, or ignorance about the disease. Exercise should be encouraged to improve the health of epileptics. Because epileptics have a five-times higher risk of suicide, exercise may lessen the risk of depression and help boost self-esteem. Epileptics and physicians who follow common-sense guidelines can develop a plan to allow safe participation in sport.

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21 THE PHYSICALLY CHALLENGED ATHLETE

Richard B. Birrer

Let me win, but if I cannot win, let me be brave in the attempt.

—Special Olympics oath

HISTORY

The first World Games for the Deaf were held in Paris during 1924, the same year that saw the formation of the International Committee on Silent Sports. Twenty years later, Sir Ludwig Guttmann introduced wheelchair sports as part of rehabilitation of war veterans. The next three decades were witness to the formation of a number of international and national organizations and competitions for disabled athletes. Among the more notable were the National Wheelchair Basketball Association (1949), National Wheelchair Games and Athletic Association (1957), International Stoke Mandeville Games Federation (1957), Paralympics (1960), International Special Olympics (1968), the Olympiad for the Physically Disabled (1976), and the International Paralympic Games(1996).[1] The latter hosted 3500 athletes from 120 countries and over 500,000 spectators. In 1978, the Amateur Sports Act (PL95-606) renewed the commitment of the U.S. Olympic Committee to amateur athletes, particularly those with disabilities. Physically challenged athletes now enjoy opportunities in sporting activities and physical education unknown to their predecessors (see Table 21.4).[2,3] They participate in virtually every sport, including marathons, rock climbing, scuba diving, archery, kayaking, weight lifting, sky diving, golf, and the martial arts, and many have established world records. Most recently, basic and applied research has begun investigating the performance characteristics of these unique athletes.

PROFILE

The spectrum of physically challenged athletes includes any individual with a physical or mental

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impairment that substantially limits one or more of the major activities of life. Sport for the disabled athlete is therapeutic, recreational, and competitive. Self-respect, self-discipline, and a competitive spirit enhance the athlete's quality of life. In general, the physical fitness profiles of physically challenged athletes improve with training and age but are lower than a normative sample. In preparing for and achieving their maximum potential, however, such athletes can attain very high levels of fitness. Regular training can increase the aerobic capacity, strength, and flexibility of a physically challenged athlete above that of able-bodied, active individuals.[4,5] Anatomic and histologic changes occur in muscle fiber type and reflect the type of training (aerobic vs. anaerobic). Physically challenged athletes with a wide range of individual differences are able to compete against one another and achieve recognition as elite performers.[6] Performance benchmarks include a 100-meter dash by a blind athlete in 10.7 seconds; a 2.1-meter high jump (6'11") by a class 2 amputee; 1500 meters in 3 min, 57 sec in a wheelchair; a 263-kg (585-lb) bench press by a paraplegic weightlifter; a 2 hr, 23 min Boston marathon by a blind competitor; and the summiting of Mt. Rainier (14,410 ft) by an amputee, five blind and two deaf hikers, and a person with epilepsy.

PHYSICIAN'S ROLE

Physicians new to the sports world of the disabled tend to assume either that the candidate is very fragile, thus prohibiting any competition, or that the chosen sport is not too rigorous or risky. As a result, many of the physically challenged do not have close relationships with sports medicine figures and often do not seek medical assistance but rather choose to self-treat injuries.[7] The American Academy of Pediatrics "medical home" concept has direct application to working with the physically challenged (www.aap.org/advocacy/releases/medicalhome.htm). The primary care physician provides continuous, coordinated, comprehensive, accessible, accountable, affordable care that is compassionate and culturally competent. Such a physician is in an ideal position to deal with the prejudice, stereotyping, and stigmatization of the physically challenged athlete. The physician's role is one of evaluation, prescription, supervision, and support (Chapter 2).[7,8] This pivotal role requires careful coordination, creativity, and collaboration due to the unique challenges and complex decision making.

ASSESSMENT

The preparticipation physical evaluation (PPE) (chapter 3) includes a careful history and physical exam that is disability-specific (Table 21.1).[5,8] When clearance is provided by the sports medicine physician, the assessment is made in the context of a multidisciplinary team: family, school officials, athletic staff, and medical consultants (e.g., neurology, surgery, rehabilitation, etc.). A history of prior surgeries and injuries, co-morbid conditions (cardiac, genitourinary, and gastrointestinal), adaptive equipment needs, fitness and training level, risk factors, and current medications (prescription and over-the-counter) is essential (Level of Evidence C, consensus opinion).[9] As examples, poorly controlled seizures can complicate traumatic brain injury. Special problems such as having a single eye or kidney, ventriculoperitoneal (VP) shunt, or gastrostomy must be addressed. Medication dosage schedules should be reviewed and periodically changed using a flexible, highly individualized approach.[10] Some drugs (stimulants, sympathomimetic amines, antispasmodics, narcotic analgesics, anti-inflammatories, steroids, diuretics, beta blockers, peptide hormones, and their analogs) may be restricted or prohibited by the relevant governing body.

Atlantoaxial instability (AAI) places 10 to 20% of Down syndrome athletes at risk for subluxation/dislocation in contact sports. Cervical radiographs are of limited predictive value, but the neurologic examination is diagnostic. Significant findings (15% of AAI patients) include new onset neck pain, difficulty walking, abnormal gait, easy fatigability, clonus, positive Babinski sign, sensory changes, paresis, incoordination, clumsiness, torticollis, and limited neck mobility, although it may be insidious and chronic without significant associated pain. The evaluation of AAI includes flexion and extension lateral C-spine films. A distance of >5 mm (children) or 2.5 mm (adults) from the posterior aspect of the anterior arch of the atlas to the odontoid process is diagnostic. The American Academy of Pediatrics and the Special Olympics Committee recommend the restriction of activities with head or neck trauma risk if the atlantoaxial interval is ≥4.5 mm or ≥5 mm, respectively (Level of Evidence C, consensus opinion).[11] Other congenital cervical spine abnormalities include spondylolisthesis, dens abnormalities, and precocious arthritis. C-spine compression from AAI is rare and usually occurs before the age of 10 years; however, because Down syndrome patients have an increased risk of juvenile rheumatoid

TABLE 21.1
Preparticipation Assessment

Examination	Notes
Vital signs	Blood pressure
	Pulse
	Respiration rate
Skin	Bony prominences, prior breakdown site, insensate areas, catheter and ostomy sites in spinal cord injured (SCI) athletes, amputee and neuromuscular disease
Eye, ear, nose, and throat	Correctible visual acuity > 20/200 for non-visually-impaired athlete
	Visual fields
	Hearing acuity > 55 dB in the better ear in non-hearing-impaired athlete
Cerebrovascular system	Congenital heart disease, especially in developmentally disabled (Down's syndrome); cardiomyopathy in muscular dystrophy
Pulmonary	Diaphragmatic excursion, as measured by percussion along posterior thorax, >3 cm in females and >5 cm in males during respiratory cycle
	Pulmonary function tests for neuromuscular and scoliotic disabilities
Gastrointestinal/ genitourinary	Bowel control
	Continence
Neuro	Identification/confirmation of SCI level of muscle tone (Ashworth scale) and primitive reflexes; decreased cognitive ability, coordination and balance (both sitting and standing)
Musculoskeletal	Range of motion (goniometer); ligamentous laxity
	Strength testing, structural deformities
	Orthotics usage; sitting posture evaluation (head position, thoracic kyphosis, pelvic tilt)
Functional	Independence measures of transferring, ambulation/mobility and proficiency in using adaptive equipment
Neck	Atlantoaxial instability (AAI)

arthritis, AAI may occur at a later date in the setting of both conditions.

CLASSIFICATION

Historically, a number of classification systems have arisen based on the particular disability.[12] Examples include those of the Special Olympics International, U.S. Les Autres Sports Association, U.S. Cerebral Palsy Athletic Association, U.S. Association of Blind Athletes, Wheelchair Sports U.S.A., Disabled Sports U.S.A., Dwarf Athletic Association of America, International Stoke Mandeville Games Federation, and the American Athletic Association of the Deaf. Currently, the Functional Classification System (FCS) is applied to level the playing field (Table 21.2). The FCS consists of a medical and technical component. The latter is determined by a sports professional team that evaluates coordination, balance, range of motion sensorimotor function, and basic levels of functional ability using a point system. Live participation in the chosen sport constitutes the technical component. The examiner may modify the final numerical score by coupling the two components; the higher the number, the higher the functional ability. The letters T, F, S, etc. refer to the sport (track, field, swimming, etc.). The classification system identifies six categories: amputees, wheelchair athletes, Les Autres, visually impaired, cerebral palsy, and intellectual disability. Each category is subdivided into classes.

PRESCRIBING EXERCISE

Depending on the degree of disability and available resources, the exercise prescription should address activities of daily living, weight control, psychological well-being, and the fitness profile (e.g., range of motion, strength, and cardiopulmonary endurance).[13] The usual principles apply with the following exceptions. The target heart rate (THR), if not directly available from an exercise stress test (EST), may be lower than calculated by the standard formula due to highly reduced vascularity and muscle mass (Chapter

TABLE 21.2
Functional Classification Systems (FCS) Categories and Classes

Visually Impaired (Athletics)	
T10	No light perception; unable to recognize hand shapes
T11	2/60 and/or visual field of less than 5 degrees[a]
F11	
T12	2/60 to 6/60 and visual field of more than 5 degrees and less than 20 degrees[a]
F12	
Amputee	
T42	Single above-the-knee; combined lower and upper limb amputations; minimum disability
T43	Double below-the-knee; combined lower and upper limb amputations; normal function in throwing arm
T44	Single below-the-knee; combined lower and upper limb amputations; moderate reduced function in one or both limbs
T45	Double above-the-elbow; double below-the-knee
T46	Single above-the-elbow; single below-the-elbow; upper limb function in throwing arm
F40	Double above-the-knee; combined lower and upper limb amputations; severe problems when walking
F41	Standing athletes with no more than 70 points in the lower limbs[b]
F42	Single above-the-knee; combined lower and upper limb amputation; normal function in throwing arm
F43	Double below-the-knee; combined lower and upper limb amputations; normal function in throwing arm
F44	Single below-the-knee; combined lower and upper limb amputations; normal function in throwing arm
F45	Double above-the-elbow; double below-the-elbow
F46	Single above-the-elbow; single below-the-elbow; upper limb function in throwing arm
Cerebral Palsy	
T30	Severe to moderate involvement; uses one or two arms to push wheelchair; control is poor; affects both arms and legs
T31	Severe to moderate involvement; foot propelled wheelchair push; affects both arms and legs
T32	Limited control of movements; some throwing motion
T32 F32	Full strength in upper extremity; propels wheelchair independently; affects both arms and legs; or same side arm and leg
T33 F33	Good functional strength with minimal limitation or control problems in upper limbs and trunk; affects lower legs
T34	May use assistive devices; slight loss of balance; affects lower legs or both legs and one arm
F34	
T35	Walks or runs without assistive devices; balance and fine motor control problems
F35	
T36	Good functional ability in dominant side of body; affects arm and leg on same side of body
F36	
T37 F37	Minimal involvement; could be present in lower legs, arm and leg on same side of body, one leg, or demonstrate problems with balance
Wheelchair	
T50	Uses palms to push wheelchair; may have shoulder weakness
T51	Pushing power comes from elbow extension
T52	Normal upper limb function; no active trunk

TABLE 21.2 (CONTINUED)
Functional Classification Systems (FCS) Categories and Classes

T53	Backward movement of trunk; uses trunk to steer; double above-the-knee amputations
F50	No grip with non-throwing arm; may have shoulder weakness
F51	Difficulty gripping with non-throwing arm
F52	Nearly normal grip with non-throwing arm
F53	No sitting balance
F54	Fair to good sitting balance
F55	Good balance and movements backward and forward; good trunk rotation
F56	Good movements backward and forward; usually to one side (side to side movements)
F57	Standard muscle chart of all limbs must not exceed 70 points[b]
Functional (Swimming)	
S1	Unable to catch water; restricted range of motion; no trunk control; leg drag; assisted water start
S2	Unable to catch water; restricted range of motion; no trunk control; slight leg propulsion; unassisted water start
S3	Wrist control limited; limited arm propulsion; minimal trunk control; hips below water; water start
S4	Wrist control; arms not fully fluent; minimal trunk control; hips below water; better body position
S5	Full propulsion in catch phrase; limited arm movement; trunk function; leg propulsion; sit or stand starts
S6	Catch phrase present; arm movement efficient; trunk control; leg propulsion; push start, sit or stand
S7	Good hands; good arms; good trunk; hips level; stand or sit dive start
S8	Hand propulsion; arm cycle good; trunk good; hips and legs level; use of start blocks
S9	Full hand propulsion; full arm propulsion; full trunk control; propulsive kick; dive start from blocks
S10	Full hand and arm propulsion; full trunk control; strong leg kick; dive start and propulsion in turns
Visually Impaired (Cycling, Goalball, Judo and Swimming)	
B1	No light perception, unable to recognize hand shapes
B2	Visual acuity of 2/60 with less than 5 degrees field of vision
B3	Visual acuity of 2/60 to 6/60 and field of vision from 5 to 20 degrees

[a] Normal field of vision is approximately 120 to 180 degrees

[b] The extent of the disability is represented by an evaluation that tests function and strength of the muscle groups. Function and muscle strength are represented by point values for each class (i.e., less than 70 points equals an F41 or F57 athlete)

Source: Lai, A.M., Stanish, W.D., and Stanish, H.I., *Clin. Sports Med.,* 19(4), 793-819, 2000. With permission.

14). The program should progress more slowly (60 to 80%) than a comparable program for a non-disabled athlete in order to improve compliance and minimize injuries. Careful coordination with community exercise resources (YMCA, health clubs, municipal recreation departments, colleges, etc.) is essential. Finally, regular follow-up including examination of the physically impaired provides needed encouragement and motivation.

The prescription should be graded with respect to intensity, as a vigorous program can cause early fatigue with secondary depressive effects. Flexibility, strengthening, and proprioceptive exercises based on the sport and physical impairment should be individualized in conjunction with the preparticipation exam. Neglected muscle groups should be emphasized. Appropriate warm-up and cool-down program elements are important. Preventive taping and orthotics should be incorporated.

The choice of a sport should reflect the preference of the patient, the type and degree of the disability, and the person's motivation and determination (Table 21.3).[14,15] Horseback riding and ice-skating are two therapeutic modalities that have been shown to be very useful among the disabled population. Disabled riders can participate in social or organized riding clubs. They can

TABLE 21.3
Sport and Recreation Options by Disability

	Individual Sports																					
Disability	Archery	Bicycling	Bowling	Canoeing/Kayaking	Cross Country	Diving	Fishing	Goal Ball	Golf	Gymnastics	Horseback Riding	Power Lifting	Rifle Shooting	Road Racing	Sailing	Scuba Diving	Skating (Roller and Ice)	Skiing (Cross-Country, Nordic)	Skiing (Downhill)	Speed Skating	Swimming	Tricycling
Amputations																						
Upper extremity	RA	R	R	RA	—	R	R	—	RA	—	R	R	RA	R	R	R	R	R	R	—	R	R
Lower extremity AK	R	R	R	R	—	R	R	—	R	—	R	R	R	R	R	R	I	RA	RA	—	R	R
Lower extremity BK	R	R	R	R	R	R	R	—	R	—	R	R	R	R	R	R	R	R	R	R	R	R
Cerebral palsy																						
Amputation	R	R	R	R	R	R	R		R	—	R	R	R	R	R	I	R	RA	RA		R	R
Wheelchair	R	I	R	R	R	I	R		I	—	I	R	R	R	R	—	—	—	—	—	I	I
Neuromuscular disorders																						
Muscular dystrophy	RA	I	R	I	—	—	R	—	R	—	I	—	RA	—	R	I	I	I	I	—	R	R
Spinal muscular atrophy	RA	I	R	I	—	—	R	—	R	—	I	—	RA	—	R	I	I	I	I	—	R	R
Charcot–Marie–Tooth syndrome	R	R	R	R	—	—	R	—	R	—	R	R	R	—	R	R	R	R	R	—	R	R
Ataxias	R	R	R	I			R		I		I	I	R		R	I	I	R	I		R	I

Sensory Impaired																						
Blind	—	R	—	—	—	—	—	—	—	R	—	R	—	—	—	—	—	R	R	R	R	R
Deaf	—	R	R	—	—	R	—	—	—	—	—	—	R	R	—	—	—	R	R	R	R	R
Developmentally disabled	R	R	R	—	—	R		R		R	R	R	—	R	—	—	R	R	R	R	R	R
Spinal Cord Injury																						
Cervical	RA	—	RA	IA	—	—	R	—	—	—	X	—	RA	—	R	—	—	IA	IA	—	R	RA
High thoracic: T1 – T5	R	—	R	R	—	—	R	—	RA	—	I	—	R	R	R	R	—	IA	IA	—	R	R
Lower thoracolumbar: T6 – L3	R	—	R	R	—	—	R	—	RA	—	R	R	R	R	R	R	—	RA	RA	—	R	R
Lumbosacral: L4 sacral	R	R	R	R	—	R	R	—	R	—	R	R	R	R	R	I	R	R	R	—	R	R
Others																						
Osteogenesis imperfecta	R	I	R	R	—	—	R	—	I	—	I	I	R	—	R	I	I	R	I	—	R	R
Arthrogryposis	R	I	R	R	—	—	R	—	I	—	R	I	R	—	R	I	I	R	I	—	R	I
Juvenile rheumatoid arthritis	RA	I	RA	R	—	—	R	—	I	—	I	I	R	—	R	I	I	I	I	—	R	I
Hemophilia	RA	R	R	R	—	—	R	—	R	—	R	I	R	—	R	R	I	R	I	—	R	R
Skeletal dysplasias	R	R	R	R	—	—	R	—	R	—	R	I	R	—	R	R	R	R	RA	—	R	R

TABLE 21.3 (CONTINUED)
Sport and Recreation Options by Disability

	Team Sports																											
Disability	Baseball	Basketball	Basketball (Wheelchair)	Bocce	Fencing	Floor Hockey	Football (Tackle)	Football (Touch	Football (Wheelchair)	Handball	Ice Hockey	Poly Hockey	Racquetball	Slalom	Sledge Hockey	Soccer	Soccer (Wheelchair)	Softball	Table Tennis	Team Handball	Tennis	Tennis (Wheelchair)	Track	Track (Wheelchair)	Volleyball	Weight Lifting	Wheelchair Poling	Wrestling
Amputations																												
Upper extremity	R	R	—	—	R	—	R	R	—	—	R	—	I	I	—	R	—	R	R	—	R	—	R	—	R	R	—	—
Lower extremity AK	Ra	—	R	—	I	—	I	I	R	—	—	—	R	R	R	I	R	RA	R	—	I	R	RA	R	R	R	R	—
Lower extremity BK	R	R	I	—	R	—	R	R	I	—	I	—	R	R	I	R	I	R	R	—	R	I	R	I	R	R	—	—
Cerebral Palsy																												
Amputation	R	I		R	I	—	I	I	—	—	I	—	R	R	—	R	—	R	R	R	R	—	R		R	R	—	—
Wheelchair	I	—	R	R	I	I	—	—	R	—	I	—	—	R	I	—	R	I	R	RA	—	R	—	R	I	R	R	—
Neuromuscular Disorders																												
Muscular dystrophy	I	I	I	R	—	—	—	—	I	—	I	—	—	I	I	I	I	I	R	—	I	I	I	I	I	—	R	—
Spinal muscular atrophy	I	I	I	R	—	—	—	—	I	—	I	—	—	I	I	I	I	I	R	—	I	I	I	I	I	—	R	—
Charcot–Marie–Tooth syndrome	R	R	—	R	R	—	R	R	—	—	I	—	—	R		R	—	R	R	—	R	—	R	—	R	R	—	—
Ataxias	I	I	R	R				I	I		I			I	I	I	R	I	R		R	R	I	R	I	I	R	—

Sensory Impaired																											
Blind	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	R	—	—	R	—	R
Deaf	—	—	—	—	—	R	—	—	—	R	—	—	—	—	R	—	—	R	R	—	R	—	R		R	—	—	R
Developmentally disabled	—	R	—	—	R	R	—	—	—	—	—	R		R	—	—	—	R	R	R	R	—	R		R	—	—	—
Spinal Cord Injury																											
Cervical	RA	—	I	—	—	I	—	—	I	—	I	—	—	—	I	—	—	—	RA	—	—	IA	—	R	IA	—	I	—
High thoracic: T1 – T5	RA	—	R	—	RA	R	—	—	R	—	R	—	—	RA	I	—	R	RA	R	—	—	R	—	R	RA	R	R	—
Lower thoracolumbar: T6 – L3	R	—	R	—	RA	R	—	—	R	—	R	—	—	RA	I	—	R	RA	R	—	—	R	—	R	RA	R	R	—
Lumbosacral: L4 sacral	—	R	I	—	R	—	I	R	I	—	I	—	—	R	—	R	—	R	R	—	R	—	R	—	R	R	—	—
Others																											
Osteogenesis imperfecta	I	I	R	—	R	—	X	I	I	—	X	—	—	—	X	X	R	I	R	—	R	R	R	R	I	I	R	—
Arthrogryposis	R	R	—	—	I	—	X	R	I	—	I	—	—	—	—	I	—	R	R	—	R	—	R	X	R	I	X	—
Juvenile rheumatoid arthritis	I	I	I	—	I	—	I	I	I	—		—	—	—	—	I	I	I	R	—	I	I	I	I	I	I	I	—
Hemophilia	I	R	—	—	R	—	X	I	—	—	X	—	—	—	—	I	—	R	R	—	I	—	R	—	R	I	—	—
Skeletal dysplasias	R	R	—	R	R	—	I	R	—	—	R	—	—	—	—	R	—	R	R	—	R	—	R	—	R	I	—	—

Note: R = recommended, I = individualize.

easily assist at horse shows — for example, by judging. Carefully graded competitions for the disabled are also available. Ice-skating helps handicapped youngsters to gain confidence and self-control. Balance is essential, but less power and strength are required than for walking. A stable spine, good quadriceps, absence of knee flexion contractures, extension and abduction stability of the hips, and extension stability of the knee are required for participation. Additionally, relatively plantigrade feet, some dorsiflexion and plantar flexion stability of the ankle assisted by an orthosis, and motor power across the ankle joints are useful. Well-fitted, single-blade skates without toe picks are recommended. An outrigger skate aid of the Lofstrand type can be used to increase the support base. A Hein-A-Ken™ skate aid is useful with children who cannot propel themselves with reciprocal action. Children with minimal brain dysfunction and attention deficit disorders benefit through precision movement, obstacle completion, and kinesthetic awareness.

TYPES OF DISABILITIES

While some disabled athletes are deaf and blind, most have one of these sensations preserved.

Sensory Impairment: The Deaf Athlete

Although the oldest international organization (1924) for disabled sport was developed for the deaf athlete (Comité International des Sports des Sourds, or CISS), public interest in and support for athletic programs for the deaf have never been strong. Deaf athletes appear normal and are capable of playing all sports that are open to people with normal hearing. Fitness levels and motor development do not appear to be significantly different from normal peers. Sensorineural defects (e.g., congenital rubella, infections, congenital malformations, Rh incompatibility, and drug therapy), rather than conduction problems are the cause of significant deafness. While the severity of the hearing deficit can be classified, a hearing loss of 55 dB or greater in the better ear (three-frequency pure tone average at 500, 1000, and 2000 Hz) is required for purposes of competition qualification (eligibility standard for World Games). Concomitant damage to the vestibular apparatus, although relatively uncommon, affects balance and coordination. Activities with sharp turns, spins, or cuts (skiing, skating), or those demanding balance (gymnastics, diving) may not be possible. Communication is the major problem for the deaf athlete, and its lack may prevent participation in some sports. Colored lights and flags and light dimmers are used to facilitate play. The athlete must develop peripheral vision in order to recognize the field position of teammates. A full athletic program for deaf athletes is available at Gallaudet College, Washington, D.C., the largest liberal arts institution for deaf people in the world. Many deaf people have excelled at the amateur, professional and elite level.

Sensory Impairment: The Blind Athlete

Legal blindness is defined as 20/200, or 1/10 or less of normal vision in both eyes. Some blind athletes are legally blind; most (80%) have some useful vision, but others are completely blind. The underlying disorder may involve the retina, glaucoma, or cataract. Certainly, the athlete with partial sight has the ability to perceive light, dark, and shadows. The International Blind Sports and Recreation Association (IBSA) governs worldwide sport competition among blind athletes and utilizes functional classification as assessed by usable vision rather than movement capability. Unless there are other disabilities, the visually impaired are generally fit, although their movements may not be as free (shuffling gait, stiffness of posture and movement) as those of the non-visually impaired. A wide range of sports activities are available to the blind athlete: swimming, track and field events, weightlifting, rowing, wrestling, golf, skiing, tandem bicycling, beep baseball, roller or goal ball, and even archery and sky diving. A sighted companion calls signals, uses clap sticks, counts steps or strokes, or touches the runner/skier in order to guide the athlete. Blind bowlers utilize a portable, waist-high guide rail 12 feet (3.7 meters) long. A battery emits a beep in a regulation softball used in beep baseball. Athletes should wear protective gear (face masks and chest and groin protectors) in this and other contact sports.

Key legislation such as the Education Amendments to the Elementary and Secondary Act of 1965 (1972) (Section 904), Rehabilitation Act (1973) (Section 504), and Education for All Handicapped Children Act (1975) (PL94-142) provide the opportunity for physical education instruction in the least restrictive environment for most blind children. Nonetheless, blind children and youth are not socialized into sport in the same ways and by the same significant factors as sighted youngsters. Learned dependence and stereotypic behaviors are underlying. Acute visual loss may be associated with an adverse psychological reaction

and cognitive delays. While many blind people do compete in individual and team sports, it is estimated that for every blind individual who is given the opportunity to learn and engage in sport two blind individuals are not. It is, therefore, a lack of experience rather than a lack of ability that is responsible for fitness and motor delays.[7] The Lavelle School for the Blind in the Bronx, NY, is one example of a free school devoted to all blind individuals ages 3 through high school.

The Spinal Cord Injured Athlete

The wheelchair is the *modus operandi* for the spinal cord injured (SCI) athlete. Track and field, swimming, table tennis, pentathlon, archery, weight lifting, fencing, snooker, marathon, bowling, pool/billiards, slalom, road running, precision javelin, basketball, and darchery (darts and archery) are some common activities for these athletes.[16] SCI patients are introduced to these activities at regional centers before joining park or recreation department programs or the national wheelchair organizations.

Classification (Table 21.2) of wheelchair athletes is intended to enable those with even the most severe disability to compete in a fair manner with others of similar degrees of disability. The system is anatomically based, and manual muscle testing of upper and lower extremities and trunk balance must be performed to determine classification. Training affects the cooperation, performance, and quality of life of the athlete.[16] Factors such as spasticity, sensation, deformities, orthoses and surgical procedures, the type of equipment, and certain pathologies (such as the ataxia of multiple sclerosis, the spotty paralysis of polio, or the incomplete quadriplegia and proximal paresis of neuromuscular disease) mean that the two ends of the disability spectrum can present formidable classification problems.[17] One fair solution is split classification: classifying an athlete differently for each event. Thus, amputees and individuals with such wide-ranging neurologic or paralyzing disorders as meningomyelocele, osteogenesis imperfecta, or brain injury can compete in the National Wheelchair Athletic Association (NWAA) with a wheelchair.

The two basic types of wheelchair designs are for medical/regular or sports purposes. Over 20 different sports chairs are built for performance rather than comfort. Acceleration, turning, and maneuverability are optimized by the use of a roll-bar, anti-tip casters, rigid frame, lowered seat back, and adjustable axle plates. Additional wheelchair models include track and racing chairs and motorized chairs. The former utilize large cambered drive wheels, small handrims, and a lowered seat position. Motorized chairs use two 12-volt batteries and allow considerable independence for severely disabled persons for up to 8 hours (after an overnight charge) at speeds up to 5 mph and inclines of at least 10°.

Common problems include bowel and bladder dysfunction, urinary tract infection, osteoporosis, urinary calculi, muscle atrophy, decubital ulcers, difficulties in temperature regulation, and respiratory compromise, especially for SCI athletes.

The Amputee Athlete

Amputee sports are governed by the International Sports Organization for the Disabled (ISOD). Amputee athletes can participate in practically any sport or recreational activity (Table 21.3). Amputee games include swimming, skiing, slalom, archery, riflery, football-kicking, table tennis, bowling, and a variety of track and field events. Amputations may be congenital or acquired, but this factor is irrelevant to the classification system, although congenital amputations should suggest the possibility of other anomalies. Nine general, five track, and seven field classifications for amputees are used by the ISOD. The team physician should be familiar with the regulations concerning assistive devices, orthoses, and prostheses. For example, crutches and sticks are prohibited in all track events, while the wearing of a prosthesis is optional. While a wheelchair may be utilized in basketball and marathons, most amputee athletes compete with a prosthesis. Some skiers "three track" by using an outrigger that has a short ski tip and a swivel. Physical conditioning or training may decrease the metabolic cost of ambulation and the risk for heat-related illnesses for lower extremity amputees.[18]

Most injuries occur in running sports. Uneven surfaces, malalignment of the prosthesis and/or pylon, and lack of cushioning cause impact and rotatory forces to be transmitted almost completely to the residual limb.[12] Loss of a heel strike necessitates greater quadriceps strength and may predispose to early fatigue following above-the-knee amputation (AKA). The hop–skip running pattern is associated with decreased pelvic rotation and exaggerated truncal and asymmetric arm movements, and excessive vaulting off the sound limb occurs due to a prolonged swing phase of the prosthetic limb. Ischial bursitis and irritation of the ischial tuberosity, therefore, are common. Areas of minor irritation should be treated with protective dressings (DuoDERM®) before a pressure ulcer

develops. Socket alterations may be helpful in some cases. Strains and sprains of the sound limb, sacroiliac region, and lumbar spine are also secondary to increased mechanical stresses during running. Upper extremity and multiple coexisting amputations are more common in children, but complications such as phantom limb syndrome and skin breakdown are less frequent. Prostheses require repair and reconditioning more regularly in the younger amputee athlete due to increasing growth and expansion as well as the frequency of abuse and misuse.

The Cerebral Palsied Athlete

As a neuromuscular condition, the degree of muscular involvement in cerebral palsy (CP) can range from severe spasticity to slight speech impairment. Unlike SCI athletes and competitors whose disabilities affect the range of motion, balance, and strength of lower extremities, the CP athlete usually has to cope with associated upper motor neuron dysfunction such as perceptual–motor problems (60%), learning disabilities (>50%), seizures (30%), visual dysfunction (75%), deafness (20%), abnormal reflex activity (startle and asymmetric tonic neck) and muscle tone (50 to 85%), and other soft signs of neuron damage (e.g., impulsivity, hyperkinesis, and attention deficits). Of the general CP population, 85 to 90% have three or more disabilities. In addition, 30 to 70% of this population is mentally retarded, a diagnosis that excludes the individual from sports events by the standards of the National Association of Sports for Cerebral Palsy (NASCP). The latter uses a functional classification system of eight categories that is sport and disability specific. About 50% of all CP athletes compete in wheelchairs (i.e., they are functionally nonambulatory), and about half are ambulatory. Seizures are best controlled with carbamazepine, as it is the drug least likely to adversely affect the athlete's performance. Aerobic activity decreases seizure potential because the metabolic acidosis associated with lactic acid accumulation stabilizes neuromembranes.[19] If seizures occur with exercise, a stress test with an electroencephalogram (EEG) is recommended. Vigilance for missed dosages due to travel and ataxia and nystagmus due to adjusted dosage schedules is paramount. Diazepam suppositories should be available at athletic events for intractable seizures.

Les Autres Athlete

A French term for "the others," *les autres* represents athletes with locomotor disabilities exclusive of cerebral palsy. Examples include muscular dystrophy, multiple sclerosis, organic brain syndrome, osteogenesis imperfecta, arthrogryposis, short stature, and ataxia. Some of these disabilities are not static; they may have a variable course, and some may be progressive; therefore, periodic medical reviews are appropriate. While the ISOD governs competition for this group internationally, the NASCP regulates the group in the United States. The classification may not always be appropriate for the associated dysfunctions and tremendous individual differences of this group of athletes. Spina bifida athletes generally tolerate sports well. A neurology or neurosurgical consultation is appropriate if tethering is a concern. Damage to a VP shunt is uncommon. Athletes with an Arnold–Chiari malformation should be managed like a Down syndrome patient with AAI. Because cognitive and learning disabilities are common, rule adaptation may be appropriate. Injury to the lower extremities is possible due to osteoporosis and sensorimotor problems. Wheelchair modification and orthotics should be considered. Bowel and bladder dysfunction are usually not problematic but may be an inconvenience.

The Developmentally Disabled and Special Olympics

The Special Olympics is the world's largest program of sport training and competition for mentally retarded individuals 8 years and older (over 1,000,000 participants, 500,000 volunteers, and 100,000 coaches in over 150 countries and all 50 states). Aquatics, gymnastics, bowling, figure and speed skating, alpine and cross-country skiing, soccer, softball, volleyball, floor and poly hockey, bowling, basketball, and track and field events occur at thousands of local, area, chapter, and national settings. Contact sports are not allowed. The International Special Olympics is held every 4 years. The Special Olympics has established a motor activities training program to introduce severely and profoundly mentally retarded people to sport activities and physical fitness. Classification of athletes is based on gender, age, and prior sport performances and is commensurate with the official rules of national sport-governing organizations. 3.5% will require medical attention for injury or illness. Down syndrome athletes are injured 3.2 times more commonly than athletes with other forms of mental retardation, presumably because of generalized joint laxity. Most injuries are minor and easily treated on site. Down syndrome athletes require attention to ligamentous

laxity, hypotonia, pes planus, congenital heart disease, AAI, and obesity. While AAI may lessen with maturity, serial neurologic examinations are recommended annually. Neurologic symptoms mandate activity restriction and further evaluation. Magnetic resonance imaging (MRI) is the imaging method of choice for suspected AAI.[11]

MEDICAL PROBLEMS

Autonomic dysreflexia (AD) is characterized by piloerection, paroxysmal hypertension and bradyarryhthmia, hyperhydrosis, and headache secondary to a noxious stimulus (e.g., pressure sore, distended bowel/bladder, infection, fracture, tight clothes) below the SCI level (most problematic at or above T6). AD has been observed in 80% of high-level athletes with paraplegia and is frequently diagnosed.[7,19] Hyperthermia (50% of track competitors) and hypothermia (9% of swimmers) disorders have been noted in the quadriplegic population (secondary to AD) and in the Special Olympics (decreased perception of problem). Management of AD includes removal of the offending stimulus, if possible, and treatment of the hypertension (nitroprusside, phentolamine, labetalol). "Boosting" or self-induced AD by an SCI competitor to enhance performance has been reported and should be diligently screened for in the preparticipation exam.[5]

Bladder infections have been reported in 22% of pediatric disabled athletes.[5] Although overuse injuries occur with slightly more frequency than acute ones, functional deterioration, hypoactivity, and fear of accident cause most of the damage to these children. Preventive strategies emphasizing environmental control and strength and conditioning plus orthotics and similar devices to correct structural deformities and reduce biomechanical stress are important.[20] No empirical data exist for injury patterns in the sensory impaired.

Spinal cord injury athletes participating in swimming, endurance races, and cold-weather activities may experience hypothermia due to loss of muscle mass below the lesion level, loss of sweat, lack of vasomotor and neural control, and decreased sensation. While preventive education is important, intra- and post-competition monitoring is critical. Loss of sudomotor and vasomotor reflexes cuts both ways and may also be responsible for hyperthermia, a condition potentially worse in the disabled athlete. In the 1990 U.S. Junior Wheelchair Games, 49% of the participants suffered from hyperthermia or heat-related illness.[7,19] If the athlete's axillae are hot, one should seriously consider the possibility of heat illness. The concomitant administration of anticholinergics or alpha blockers make this even more likely. Both conditions require emergency treatment (Chapter 35).

Pressure sores afflict wheelchair athletes, amputees, and all insensate athletes with improperly fitted prostheses, and occurs in those sports that involve high amounts of directional change and lateral movement (e.g., basketball, tennis). Anatomical areas that require attention are the greater trochanter of the hip, sacrum, and ischium. Pressure loads are further exaggerated by specially designed sports wheelchairs in which the athlete's knees are higher than the seat. Proper positioning, custom-fitted cushioning, correct fit of prostheses and equipment, regular repositioning, and minimization of skin shear and moisture are important management strategies.

Premature osteoporosis and osteopenia secondary to paraplegia can lead to fractures in the paralyzed extremities during contact sports. These should be suspected in the setting of AD, pain, angular deformity, and minimal mechanical force. Heterotopic ossification also occurs in SCI or traumatic brain injured athletes, usually around major joints. While sports participation does not increase the risk of ectopic bone formation, it should be considered a stimulus to AD. Limitation of exercise capacity (decreased cardiac output), venous blood pooling, and the potential for deep venous thrombosis are additional problems in the SCI athlete. Management strategies include abdominal binders, positive pressure garments (stockings), and lower extremity functional electrical stimulation.

Spasticity is a potential problem for any athlete with a central nervous system (CNS) injury. Removal of irritant stimuli, flexibility training, appropriate bowel and bladder programs, and regular posture/position changes are appropriate. Awkward gait patterns are extremely exhausting, as is the use of wheelchairs and orthotics. Extra energy is required. Obesity prevention is a particular challenge for those individuals who use crutches to ambulate or who bear weight on weakened extremities. Individuals with muscular dystrophy should not exercise to exhaustion due to the danger of muscle damage (weakness within 30 minutes following exercise, excessive muscle soreness 24 to 48 hours following exercise, severe muscle cramping, extremity heaviness, prolonged shortness of breath).

Psychological problems (depression, anxiety) can occur in any physically challenged athlete but are more common in the childhood and adolescent

Table 21.4
Major Organizations for Physically Challenged Athletes

National Sports Center for the Disabled
P.O. Box 1290
Winter Park, CO 80482
303-293-5711
www.nscd.org/nscd

United States Amputee Association (USAA)
Route 2, County Line
Fairview, TN 37062
615-670-5453

United States Association for Blind Athletes (USABA)
33 N. Institute Street
Brown Hall, Suite 015
Colorado Springs, CO 80903
719-630-0422
www.usaba.org

American Athletic Association of the Deaf (AAAD)
3607 Washington Blvd., Suite 4
Ogden, UT 84403-1737
801-393-7916 (telecommunication device for the deaf)
801-393-8710 (voice)
www.aaad.org

Dwarf Athletic Association of America
418 Willow Way
Lewisville, TX 75067
214-317-8299
www.daaa.org

National Association of Sports for Cerebral Palsy (NASCP)
66 E. 34th Street
New York, NY 10016
212-481-6359

United States Cerebral Palsy Athletic Association
200 Harrison Ave.
Newport, RI 02840
410-848-2460
www.uscpaa.org

National Handicapped Sports and Recreation Association (NHSRA)
Capitol Hill Station
P.O. Box 18664
Denver, CO 80218
303-232-4575

United States Organization for Disabled Athletes
147 California Ave.
Uniondale, NY 11553-1131
516-485-3701
www.wws.net/usoda

Special Olympics, Inc. (SO)
1356 New York Avenue, N.W., Suite 500
Washington, D.C. 20005

Special Olympics International
1325 G Street, NW, Suite 500
Washington, D.C. 20005
202-628-3630
www.specialolympics.org

Disabled Sports USA
451 Hungerford Dr., Suite 100
Rockville, MD 20850
301-217-0960
www.dsusa.org

Wheelchair Sports USA
3595 E. Fountain Blvd., Suite L-1
Colorado Springs, CO 80910
719-574-1150
www.wsusa.org

Amputees in Motion
St. Benedict's Manor
3000 Polk, #419
Ogden, UT 88403
801-731-3971
www.usinter.net

The United States Olympic Committee
Disabled Sports Services Dept.
One Olympic Plaza
Colorado Springs, CO 80909-5760
719-578-4958

Amputees in Motion
P.O. Box 2703
Escondido, CA 92025
619-454-9300

age category. Parental overprotection, unrealistic expectations, inadequately trained coaches, lack of medical support, inadequate transportation and toileting facilities, social isolation, and peer pressure may negatively impact improved self-confidence, self-sufficiency, and self-actualization.

MUSCULOSKELETAL INJURIES

Injury rates are approximately the same as those of athletes without a disability in similar sport activities.[21] They are usually minor and of low grade. Soft tissue (e.g., sprains, bursitis, tendinitis, and strains, especially of the shoulder, arm and elbow) and skin problems (e.g., abrasions [71% are wheelburns], pressure sores, blisters [77%]) occur in up to 97% of wheelchair athletes during training and competition in track events.[7,21] Shoulder pain is the most common complaint in wheelchair athletes.[12] The differential diagnosis should include bicipital and rotator cuff pathology as well as osteonecrosis. In 14 to 16% elbow pain is secondary to forearm extensor tendinitis, olecranon bursitis, triceps tendinitis, epicondylitis, and nerve entrapment (carpal tunnel [50–73%], ulnar [50%], radial [16%]). The point of hand–wheel contact at the proximal carpal tunnel is the site of impingement in carpal tunnel, whereas Guyon's canal is the ulnar entrapement site. Ganglion cysts of the long flexors or extensors are secondary to repetitive injury to the wrist joint capsule. Avascular necrosis of the lunate (Kienbock's disease), dorsal compartment tenosynovitis, and deQuervain's disease are additional repetitive use injuries. Track, road racing, and basketball also have the highest percentage of such injuries. Hand and wrist fractures are the most commonly encountered fractures in the wheelchair athlete, particularly during collision sports. Associated osteopenia may impair the healing process. In the childhood athlete, the epiphyseal plate is involved. Skiing by athletes with disabilities is relatively safe. The knee is injured more commonly in CP athletes. Overuse injuries to the wrists, hands, and rotator cuff are common in the wheelchair population, whereas lower extremity overuse problems (tendinopathy, apophysitis) are very unusual. Cross-training in another sport (e.g., swimming) for a short period may be necessary. No empirical data exists for injury type or rate for athletes with sensory impairments.

SUMMARY

Regular physical activity for the physically challenged in the form of sports and recreational exercise improves their fitness profile, increases muscular function, and builds courage and self-esteem. Regular participation moves these individuals beyond their specific disability, focuses on ability and achievement, and enables them to enter the mainstream of society. A proactive physician role is essential because physicians are rarely reported to be primary motivators for competitive or recreational athletic involvement.

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22 THE RUNNER*

Chad Asplund and Suzanne M. Tanner

INTRODUCTION

A popular form of physical activity, running is a relatively simple method of increasing overall fitness and reaping the health benefits of regular exercise. The number of runners in the United States is estimated to be over 30 million. More than 10 million of these athletes run on more than 100 days of the year, and about 1 million enter competitive races each year.[1] The increase in participation and competition has been accompanied by an increase in running-related injuries. The competent primary care sports physician must be familiar with the etiology, pathogenesis, evaluation, diagnosis, treatment, and prevention of these injuries and common medical problems in runners.

EPIDEMIOLOGY OF RUNNING INJURIES

It is not surprising that injuries occur during running when one considers that the lower extremity is loaded with 1.2 to 2.1 times body weight at heel strike, and 2.5 times body weight at toe-off,[2] and that each foot strikes the ground an average of 800 times per mile.[3] Compared with walking, these forces must be attenuated by the body in roughly one third the time, due to the decreased period that the foot is in contact with the ground.[3] The annual incidence of running injuries ranges from about 37 to 56%.[4] Jacobs and Berson[5] found that 210 (47%) of 451 runners competing in a New York 10-km race had sustained at least one previous injury that required them to reduce their weekly mileage during the 2 years prior to the race. When running, the force at the ankle and hip is twice as great as when walking on a level surface; at the knee, it is six times as great.[6] No wonder the knee is the site most commonly injured (25 to 45% of injuries).[3,5] Other sites that may suffer from acute trauma or chronic overload include the low back, hips, hamstring, anterior lower leg, Achilles tendon, ankle, and heel.[3,5] The patellofemoral pain syndrome is the most common injury, followed by iliotibial band friction syndrome, plantar fasciitis, meniscal injuries of the knee, and tibial stress syndrome (Level of Evidence B, retrospective study).[7] About 60% of these injuries appear to result from training errors such as excessive interval training or speed work, running on poor surfaces, rapid mileage increase, ignoring a previous injury, failure to recognize physical limitations due to a biomechanical problem, poor flexibility training, and wearing improper or worn-out footwear.[4]

* In the second edition, this chapter was authored by Suzanne M. Tanner.

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CAUSES OF RUNNING INJURIES

Etiologic factors associated with running injury include previous injury, lack of running experience, running to compete, and excessive weekly running distance (Level of Evidence B, systematic review).[4] Identifying and altering characteristics of the runner, training methods, and running environment may help prevent running injuries.[8] Possible factors that may contribute to musculoskeletal damage during running are discussed below.

Characteristics of the Runner

Age

In the majority of studies, no significant relation has been found between age and running injury rate.[9] Brunet et al.,[3] however, found that an increasing age of runners paralleled a slight increase in the self-reported incidence of hip, back, and foot pain. These complaints, however, typically increase with age in the general population, irrespective of running. Taunton et al.[7] also found that age less than 34 years conferred increased risk for patellofemoral pain syndrome. Similarly, Yzerman and van Mechelen found that runners at a younger age sustained more injuries than older runners.[4] Thus, a relative, not absolute, relationship may exist between injuries and extremes of age.

Gender

It was previously thought that gender *per se* does not seem to be an important risk factor for running injuries.[7] Several retrospective studies have shown no difference between men and women in the rate of running injuries;[5,8,10] however, a large 15-year prospective longitudinal study of over 3000 high school cross-country runners showed that girls sustain running-related injuries more often than boys (Level of Evidence B, clinical cohort study).[11] Females with poor quadriceps strength and an increased Q angle* may be predisposed to patellofemoral pain. Stress fractures also tend to be more common in female runners than male runners. In younger age groups, female runners tend to be more prone to running injuries than males, although the exact etiology is unknown.

Structural Abnormalities

Although the role of anatomical factors in running injuries has not been well documented in epidemiological studies, the hypothesis that structural abnormalities are a risk factor for running injuries is too reasonable to deny.[7] Leg length inequality is seen frequently but generally is not considered functionally significant unless greater than 13 mm (0.5 in.).[12] Up to a 20- to 30-mm leg length discrepancy may be acceptable in a low demand population, but in the repetitive high-loading environment of running, many suggest compensating for a discrepancy as small as 5 mm.[13] Wen et al.[14] found that lower extremity alignment was not a major risk factor for injury in a low-mileage group, but further study in high-mileage runners is warranted. Genu valgum, varum, and recurvatum; patella alta; and pes cavus and planus have been blamed as causes of running injuries,[7] but data from various studies are conflicting.

Body Build

Powell[8] examined body mass index (BMI; weight in kilograms divided by height in meters squared) and found it was unrelated to injury rate. Taunton et al.[7] found that women with a BMI of <21 kg/m^2 were at increased risk for tibial stress fractures and spinal injuries. Low BMI, especially in the context of a lower bone density and higher level of bone density required in active athletic female runners, may predispose to stress-related injuries.[15]

Past Injury

Runners with a previous injury may more likely be reinjured because: (1) the injury may have not healed completely, (2) rehabilitation of surrounding muscles may be incomplete, (3) inherent individual tissue susceptibility to injury may remain (e.g., structural abnormality), and (4) repaired tissue may not function as well.[7] Many studies have supported and identified previous injury as an important independent risk factor for running injuries; however, the exact mechanism still requires clarification.[4]

Characteristics of Running

Distance

Weekly running distance is a strong determinant of running injuries.[4] The average number of miles run per week is clearly associated with the incidence of running injuries.[7] The incidence of stress fractures, for example, increases with more miles run per week. In a study by Brunet et al.[2] of 375 women, 4% sustained a stress fracture while running 11 to 20 miles per week; 32% of the women sustained a stress fracture while running more than 50 miles a week. Newell and Bramwell[16] estimate that running between 50 and 70 miles per week results in a 50% chance of knee injury.

* Angle of the shaft of the femur and the shaft of the tibia. Normal in girls is under 20°; normal in boys is under 15°.

Marti and Willick showed that, although the absolute risk of injury increases with increasing mileage, the relative risk (per unit distance) decreases with increasing mileage.[17] Existing literature does not support running contributing to osteoarthritis, but some evidence may suggest an association of high mileage running with an increase in osteoarthritis.[17] No specific mileage can be identified below which one can run without risk of injury nor a mileage above which the risk of injury abruptly increases.[7] A linear relation appears to exist between injury rate and mileage run per week.[8]

Speed

Speed of running does not appear to be related to the risk of injury when the number of miles run per week is taken into account.[7] In the younger runner, however, a significant association exists between training speed and running injuries.[4] Although, in general, it has not been shown that running speed causes injuries, impact forces increase as speed increases, so that during time of recovery, it would be prudent to advise runners to reduce training speed as a means to lower impact.[18]

Stability of Running Habits

A sudden change in weekly mileage is a frequently mentioned factor contributing to running injuries cited in clinical studies. Over 60% of running injuries have been attributed to training errors, most notably rapid increases in mileage and intensity of training.[18] An abrupt increase in the stress on the musculoskeletal system is likely to produce an injury because of lack of tissue ability to adapt to the sudden change.[4] Limiting weekly increases in mileage to 10% maximum may be a safe guideline, but the actual magnitude of the increase needed to elevate the risk of injury is unknown.[7]

Stretching

Theoretically, improved flexibility increases the range of motion around a joint and thereby reduces stress placed on the tissues around the joints during running.[7] Surprisingly, though, no association between performing stretching exercises and injury prevention has yet been found in epidemiological studies.[5,7] In fact, many studies cite stretching as a cause of increased running injuries.[4] Stretching following injury has been demonstrated to prevent reinjury, and chronic and overuse injuries may benefit from a regular stretching program.[19] Overall, stretching and its relation to injuries is controversial, but improved muscle flexibility aids in recovery from and prevention of reinjury.

Characteristics of the Running Environment

Terrain

No association has been found between terrain and rate of injury, suggesting that persons who regularly run up and down hills have no more or fewer injuries than persons who regularly run on level ground.[7] Runners who frequently run on roads with cambered surfaces have been shown to have left-sided injuries, probably related to unequal load in the legs.[4]

Surfaces

In men, no difference in the incidence of injury between those who usually run on an artificial surface and those who usually run on the street has been found.[2,5] In women, however, it has been found that running on hard concrete surfaces leads to an increase in running injuries.[20] Running on ice and snow, of course, may lead to falls.

Shoes

Many studies show that well-designed and properly fitted running shoes should assist in the reduction of overuse injuries, and proper shoe selection and fit may compensate for biomechanical abnormalities and decrease the rate of injury.[21,22] Excessively worn running shoes may also lead to injury. Yzerman and van Mechelen found a significant correlation between infrequent change of running shoes and injury. Shoes with poor heel counters may cause overpronation and result in internal tibial or femoral torsion, which can increase the risk of injury.[3]

Other Potential Causes

Other potential, but inadequately studied, factors that may be associated with running injuries include running form, strengthening exercises, warm-up prior to running, and time of day during which running is performed.[7]

EVALUATING THE INJURED RUNNER

A detailed evaluation (Table 22.1) must be carried out, as a number of distinct etiologies are possible (Table 22.2). After a thorough history, which should include a healthy amount of listening, the athlete (dressed in shorts and barefoot) should be examined standing, sitting, prone, and supine. Anatomy (e.g., landmarks, length, size, shape, etc.) and biomechanics (e.g., range of motion, strength) should be assessed on the injured as well as the normal side. The running shoes should

TABLE 22.1
Evaluation Checklist

1. History

Pre-existing medical/surgical illnesses, including medications and allergies

Previous injury and outcome

Symptom

- Onset
- Mechanism of injury
- Change over time
- Treatment and results change over time

Training habits

- Distance
- Frequency
- Time of training
- Intensity/pace/speed
- Footwear
- Style
- Warm-up/cool-down
- Terrain/surface

2. Exam

Carefully check the following sites for structural and functional abnormalities:

- Abdomen
- Back
- Hips
- Thighs
- Knees
- Legs
- Ankles
- Feet/footwear

3. Laboratory

As indicated for underlying medical illness

4. Radiology

Indications include suspicion of osteoarthritis, osteochondritis dissecans, and loose bodies

5. Special studies

Stress films — may be positive when plain films are normal

Technetium-99 bone study — high sensitivity, moderate specificity for suspected stress fracture (positive 5 to 7 days after injury)

Computed tomography (CT) scan — superb for cortical detail

Magnetic resonance imaging (MRI) — excellent for soft tissue detail

Treadmill analysis of running/gait — useful for evaluating effectiveness of orthotic devices and determining whether malalignment or training technique is predisposing to injury

TABLE 22.2
Running Injuries

Site	Condition	Predisposing Conditions
Back	Lumbar strain	Weak back and abdomen
	Herniated disc	
	Sacroiliac joint irritation	Leg length discrepancy
	Femoral anteversion	
Hip	Greater trochanteric bursitis	
	Osteitis pubis	
	Piriformis syndrome	
	Ischial bursitis	
	Gluteus medius tendinitis	
	Anterior iliac crest apophysitis	Children only
	Posterior iliac crest apophysitis	Children only
Thigh	Quadricep strain	Inflexibility
	Hamstring strain	Inflexibility
	Adductor strain	Inflexibility
	Abductor strain	Inflexibility
Knee	Quadriceps tendinitis	
	Chondromalacia/patellofemoral pain	Females, widened Q angle
	Irritation of the plica	
	Patellar tendinitis	
	Prepatellar bursitis	
	Osgood–Schlatter's disease	Teenager
	Iliotibial band friction	Inflexibility
	Popliteus tendinitis	
	Pes anserinus bursitis	
Lower leg	Gastrocnemius strain	Inflexibility
	Periostitis ("shin splints")	
	Anterior tibialis tendinitis	Inflexibility of Achilles tendon
	Anterior compartment syndrome	
Ankle	Achilles tendinitis	Inflexibility, hyperpronation
	Sever's disease	Young teenager or child
	Retrocalcaneal bursitis	Tight shoe heelcup
	Peroneal tendinitis/subluxation/	Inflexibility, cavus foot
	Posterior tibialis tendinitis	Hyperpronation
Sprains	Lateral ankle ligaments	Inadequate rehabilitation
	Deltoid (medial) ligament	After previous sprain
Foot	Plantar fasciitis/heel spur	Adult, pes cavus, pes planus
	Accessory navicular bone	
	Metatarsalgia	Shoewear, inflexibility of Achilles tendon

TABLE 22.2 (CONTINUED)
Running Injuries

Site	Condition	Predisposing Conditions
	Sesamoiditis	Hyperpronation, cavus foot
	Bunions	Hyperpronation, tight shoewear
	Blisters	Shoewear, pes cavus
	Nerve entrapment (tarsal tunnel syndrome)	
	Toe problems (ingrown toenails, subungual hematoma)	
All sites	Stress fracture	
	Aggravation of preexisting arthritis	

also be examined for wear. Wear patterns can indicate biomechanical tendencies, which may help identify causes of injuries. While acute injuries are more evident, chronic overuse injuries are more common in runners; often, injuries may be mixed.

Medical Problems

The runner may be affected by a number of medical problems that must be distinguished from more significant underlying illnesses (Table 22.3). These problems are discussed at length in the medical section of the text.

TABLE 22.3
Medical Problems in Runners

Problem Area	Specific Problem
Thermoregulation	Heat exhaustion/heat stroke
	Hypothermia
	Frostbite
Skin	Runner's nipple
	Athlete's foot (fungal infection)
	Sunburn
Gastrointestinal	Cramping pain
	Diarrhea, with or without blood
Urinary tract	Hematuria
	Myoglobinuria
Hematologic	Iron-deficiency anemia
Reproductive	Oligomenorrhea/amenorrhea
Psychological	Addiction to running
General	Dehydration

Psychological Aspects

"Runner's high" has been described as euphoria or a sense of well-being accompanying long-distance runs. Experienced runners who regularly train long distances and who seek mental dissociation while running most frequently report this high. Mental diversions may permit escape from pain. The rhythmic pace and controlled breathing accompanying running has been likened to meditation. Perhaps endorphins, released from the pituitary gland and other sites during running, have a morphine-like effect, masking pain and producing euphoria.[23] Psychological aspects are also vital in the recovery from injury. Robinson et al.[24] suggest that "rehabilitation is 75% psychological and 25% physical." The runner's actions, emotions, and cognitions can work either for or against the rehabilitation behaviors,[25] so it is especially important to address the psychological state of the injured runner.

Shoe Selection

The four main categories of running shoes are stability, motion control, cushioned, and lightweight:

- *Stability* shoes have a good mix of cushioning, medial support, and durability. These shoes are generally semicurved and have a medial post or multidensity midsole. Stability shoes are well suited to the midweight, neutral runner with medium to low arches.
- *Motion control* shoes are the most rigid, with the goal of limiting overpronation. These shoes tend to be the heaviest and most durable (also most expensive) of the running shoes. Typically, they have medial post, multidensity midsole, possibly a polyurethane midsole, and a carbon rubber outsole. Many are built on a straight last, which offers stability and

TABLE 22.4
Shoe Fitting

Place old shoes on a firm surface; look for any tilt of the shoe either medially or laterally.

Inspect the wear pattern on the sole of the shoe.

Have the patient stand barefoot with feet shoulder width apart and inspect arch height.

Have patient dip feet into basin of water and make footprints on dark paper; evaluate the arch pattern.

Have patient walk 15 to 20 feet barefoot and observe gait for touchdown, carriage, and push-off.

From this, determine the foot type and biomechanical pattern:

Low Arch/Overpronator	Normal Arch	High Arch/Underpronator
Motion control or stability (firm midsole)	Stability or cushioned	Cushioned (flexible midsole)

maximum medial support. Motion control shoes are best geared to the heavyweight overpronator with flat arches.

- *Cushioned* shoes have the softest midsole and the least medial support. They are usually built on a curved or semicurved last. These are most appropriate for light to midweight underpronators with high arches.
- *Lightweight* shoes are generally cushioned and may be used for training or racing. High-performance, biomechanically sound runners generally use lightweight shoes, because they may offer a kinematic advantage.[26,27]

The running shoe protects the body from injury by absorbing and distributing the force of impact.[3] A difference of approximately 33% in initial shock absorption has been observed among 40 different shoe models, but no long-term differences in shock absorption characteristics have been found to be based on either price or manufacturer.[4] Shock absorption of running shoes decreases as a function of miles run. Shoes that exhibit superior initial shock absorption capability often have more rapid degradation of this property.[3] Shoes lose approximately 30% of their initial shock absorption after 500 miles of running,[3] or after approximately 3 months of weight-bearing exercise. Thus, prudent advice may be to suggest that runners purchase new shoes every 400 to 500 miles or 3 to 6 months of regular wear. It has also been shown that a wet midsole has 40 to 50% less shock-absorbing capability.[28]

In a relatively short amount of time in the office, the physician can assist a runner in proper shoe fit (Table 22.4) First, have the patient stand with feet shoulder width apart and observe their arches. Next, perform a wet test; have the patient get their feet wet and observe the footprint left on a piece of dark paper. If the patient has a high arch, the foot will then tend to underpronate, as described previously, and will benefit from a cushioned shoe with a curved last. These runners should avoid motion control or stability shoes, as these will tend to minimize foot mobility. A flat-arched foot predisposes to overpronation. Runners with low arches tend to overpronate and are best fit with motion control or stability shoes with firm midsoles and straight to semicurved lasts. They should avoid cushioned, curved last shoes. A runner with a normal arch can wear either a cushioned or stability shoe. Patients with normal arches and no biomechanical abnormalities could use a cushioned shoe. If they have some mild to moderate pronation, a stability shoe is probably a better choice. Lightweight runners who have no biomechanical problems could train in cushioned, lightweight trainers, but this should be discouraged due to the decreased shock-absorbing capability and a higher potential for injury.

Orthotic Devices

Orthotic devices are preformed or custom-made rubber or plastic shoe inserts. By supporting the foot in a neutral position, pronation or supination in a symptomatic runner is corrected. Asymmetry, such as a leg-length discrepancy, can also be corrected. Although orthotics have been used for years, little scientific or clinical evidence has been published in the literature to support their use.[29] The effectiveness of the orthotic device, however, is dependent upon the skill of the medical personnel molding and adjusting the orthotic. The fabrication of orthotic foot devices for runners

requires both skill and experience. Significant variance can be observed among personnel constructing orthotic devices using the same molding system. Runners should have orthotics created by a person experienced with sports orthotics, such as a sports podiatrist. Patients should gradually increase the duration of wearing orthotics from several hours per day to full-time usage over several weeks. Adjustments of the orthotic inserts are required if pain reduction does not occur after 1 to 2 weeks. If a runner who wears orthotics becomes injured, the orthotic should be inspected for wear or breakdown. Because biomechanical malalignment is often well tolerated or compensated for by the body's own adjustments, orthotic devices should not be prescribed as a preventive measure in an asymptomatic runner.

THE YOUNG RUNNER

Recently, the popularity of distance running among children, including participation in marathons, has surged. It is difficult to determine if the aerobic fitness benefits of distance running in prepubescent children outweigh the risks. Reports of potential damage have not been evaluated with proper epidemiological scrutiny. It is not known whether the risk is greater for children than for adults. Until further data are available concerning the relative risk of endurance running at different ages, the American Academy of Pediatrics (AAP) recommends that, "If children enjoy distance running and are asymptomatic, there is no reason to preclude them from training for and participating in distance running" (Level of Evidence C, consensus opinion).[30] The AAP, however, advises medical personnel who counsel families to warn the families of potential risks. Training for long-distance running by prepubescent children may induce thermoregulatory, hematologic, endocrine, psychosocial, and musculoskeletal problems (see Chapter 18). The incidence of these disorders, however, is not well documented.

A child's ability to maintain thermal homeostasis during prolonged running is less efficient than an adult's, particularly during extremes of heat and cold (Chapters 10 and 18). Children absorb more heat than adults due to their higher ratio of body surface area to mass. They have a less efficient sweating mechanism and must rely on more nonevaporative cooling mechanisms.[31] Thus, children may be more predisposed to dehydration, heat stroke, and hypothermia than adults during prolonged running in extreme conditions.

Iron depletion, manifested by low serum ferritin concentration even with a normal hemoglobin concentration, is not uncommon among adolescent runners of both sexes. In one study, 80% of young female athletes were iron deficient.[32] An iron-poor diet, possible foot-strike hemolysis, hematuria, mild occult gastrointestinal bleeding, and potential loss from menstruation may be contributing factors. Studies of the incidence and etiology of iron depletion, however, have not been performed in prepubertal children.

Delayed menarche often occurs in female distance runners. Menarche will usually occur several months after cessation of or reduction in training. When menarche is delayed, a medical etiology should be explored, rather than assuming that it is caused by exercise alone. Amenorrhea is an important clinical symptom in young runners due to the potential of endocrinologic consequences, as peak bone mass is gained between 16 and 30 years, with 48% being accrued in the adolescent years.[31]

Psychologic and social problems for the child runner may result from spending long hours training and striving for unrealistic goals. A runner's self concept may become tied to athletic success, with loss in competition producing a decreased feeling of self worth. To minimize this risk, the AAP recommends that prepubertal children should be allowed to participate for the enjoyment of running, without fear of parental or peer rejection or pressure.[30]

Overuse injuries, particularly involving the apophyseal growth plate, are common in children (see Table 22.2). Recent data indicate that 30 to 50% of all pediatric sports injuries are due to overuse.[33] The American College of Sports Medicine estimates that 50% of overuse injuries in children are preventable.[34] In an effort to prevent overuse injuries, running programs should emphasize general fitness and avoid excessive volume.

THE OLDER RUNNER

The older runner is at a higher risk of injury because of age-related changes, including prior joint injuries, loss of flexibility, and strength deficits. Thus, it is important for the primary care sports provider to be knowledgeable about the demands of the sport and the physiologic changes seen in the elderly when treating injuries in this population.[35] The most notable changes observed with aging are those involving the musculoskelatal system. Lean body mass decreases, interstitial fat content increases, joint motion diminishes, and muscle strength and endurance decrease. Progressive loss of bone mass is also a concern.[36]

While running does not appear to cause osteoarthritis in persons with normal weight-bearing joints,[37,38] impact from running tends to accelerate articular cartilage damage in persons with preexisting joint damage. Thus, running may potentiate the development and severity of osteoarthritis in persons with a previous total meniscectomy, ligamentous instability, and preexisting arthritis.[6]

Many elderly people take medications that can trigger adverse effects while they exercise,[36] so healthcare providers must be cognizant of what medications are taken and educate the patient regarding the potential risks with running.

Sudden death in runners over age 30 years is almost invariably due to severe atherosclerotic coronary artery disease. Before beginning a vigorous exercise program, an exercise stress test should be considered after a patient is carefully risk stratified.

Injuries to the older athlete can be thought to be a combination of training effects and changes to the body secondary to aging. As with other runners, proper training techniques are important in order to avoid injury in older runners.

SUMMARY

The incidence of running injuries may be reduced by employing proper training methods, correcting anatomic deficits, and using the proper equipment. Mileage and intensity should be increased gradually, and injuries should be properly healed and rehabilitated before resuming high-level training. Leg length discrepancies, foot and arch abnormalities, and excessive motion may contribute to injuries, and correction of these deficits may decrease frequency of injuries. Properly fitted shoes can help minimize excessive lower extremity motion, can reduce shock, and may help prevent injuries. Running is a simple and effective way to enjoy the health benefits of regular exercise and with attention to these factors may be performed safely in all age groups

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23 THE SWIMMER

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INTRODUCTION

Swimming is the most popular participation sport in the United States, involving 120 million athletes.[1] Men and women of all ages swim for fitness benefits, leisurely pursuit, camaraderie, or competition. Swim teams across the United States recruit children as young as 6 and master's programs attract swimmers into their 90s. Although the overall number of injuries is small compared to the large participation number, invariably the sports medicine physician will see and treat many swimming injuries. The majority of swimming injuries are found in competitive swimmers, and most of these injuries are related to overuse.[3] A successful competitive swimmer trains 5 to 7 days a week for 10 to 11 months of the year. Many engage in two workouts a day, often averaging 8000 to 20,000 yards per day. This equals over a half million arm movements a year and could be compared with running 45 miles per day.[4] It is understandable why shoulder pain is one of the major problems swimmers develop. Swimmers need to recognize the difference between soreness and the true pain that signals an injury and seek appropriate attention in a timely manner; however, competitive swimmers will often

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Figure 23.1 Freestyle stroke is broken down into two parts: pull-through and recovery.

continue to swim for weeks, worsening their injury. The coach, trainer, and sports physician must work as a team to provide aggressive management of injuries in order to return the swimmer to the water quickly. Education to prevent recurrence of injuries is also important.

BIOMECHANICS

Competitive swimming consists of four strokes: breaststroke, backstroke, butterfly, and crawl (freestyle). In the backstroke, butterfly, and crawl, the majority of propulsion comes from the arms. Thus, it is not surprising that the majority of injuries in these swimmers involve the upper extremity. All three have overhead strokes that involve similar shoulder motion, and therefore result in similar injury patterns. The majority of training, regardless of the swimmer's chosen stroke, is done freestyle. Proper freestyle technique decreases the likelihood of injury to the shoulder. During the out-of-water phase (recovery), the torso rotates as the shoulder exits the water in an abducted and externally rotated position. The elbow should remain above the hand until the hand enters the water in front and just outside the line of the shoulder. To keep the elbow high, the swimmer must roll the body 70 to 100 degrees. During the underwater phase (pull-through), the shoulder internally rotates and adducts to propel the body forward. The upper trapezius, rhomboids, supraspinatus, and deltoid all function in combination to position the scapula and humerus for hand entry and exit during freestyle (Figure 23.1).[5]

Breakdowns in freestyle mechanics, whether secondary to pain or fatigue, can lead to aggravation of the shoulder. A fatigued swimmer may drop the elbow during the recovery phase which may result in impingement of the rotator cuff muscles. In lieu of changing to a different stroke or resting completely, the swimmer can continue swimming the freestyle stroke with a modification that places less strain on the shoulder; the swimmer's arm exits at the iliac crest instead of at the thigh, resulting in decreased tendon irritation

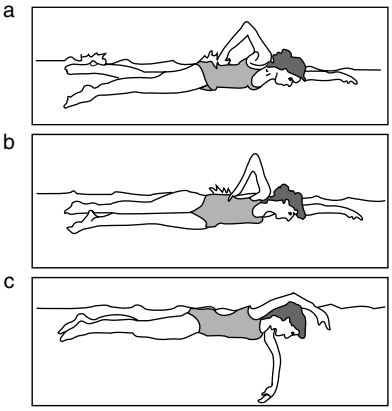

Figure 23.2 Reduce freestyle injuries by pulling the hand out of the water at the iliac crest. (From Kammer, L.T.C., *Phys. Sportsmed.*, 27(4), 53, 1999. With permission.)

(Figure 23.2).[6] The breaststroke is unique in that it relies heavily on the legs for propulsion and less on the arms.[7] A whip kick is utilized during this stroke which places tremendous valgus stress at the knee and ankle (see Figure 23.3). Consequently breaststrokers are more often bothered by lower-extremity injuries.

UPPER EXTREMITY INJURIES

Swimmer's Shoulder

Shoulder pain is the most common complaint among competitive swimmers. It is generally related to the repetitive overuse of the swimmer's stroke as well as to problems with stroke technique. Classic swimmer's shoulder refers to an impingement syndrome caused by repeated rubbing of the rotator cuff muscles and biceps tendon against the under-surface of the acromion, resulting in chronic inflammation.[8] This condition is seen to some extent in up to 40 to 60% of competitive swimmers.[9] Typical symptoms include pain about the acromion that is initially present only during a workout and maximal at the beginning of the pull phase of the stroke.

Figure 23.3 The whip kick utilized during the breaststroke. (From Richardson, A.R., *Clin. Sports Med.*, 1986, 5(1), 104. With permission.)

With time, pain can become constant and prevent the swimmer from competing. Eventually, chronic impingement syndrome can lead to tears of the rotator cuff; however, this is rare in the younger swimmer.

Initial treatment includes relative rest, icing, and nonsteroidal anti-inflammatory medications. In addition to decreasing the total weekly mileage, the swimmer should use varying stroke styles. One option is to use swim fins that will allow the swimmer to train at a pre-injury pace with less stress on the shoulder.[1] In cases where pain is persistent, the swimmer should limit training to kickboard workouts to maintain cardiovascular and lower extremity fitness while minimizing strain on the shoulder. The kickboard should be held with the affected arm slightly flexed at the elbow and shoulder, as the standard kickboard position with the arm straight out essentially reproduces the impingement test maneuver and may aggravate symptoms.[1] In addition, use of an upper arm counterforce brace (tennis elbow strap) placed high on the arm over the biceps tendon while training may help reduce symptoms.[10]

If conservative management fails, corticosteroid injections can be effective in helping to reduce inflammation.[9] Other therapeutic modalities such as electrogalvanic stimulation (EGS), hot packs, and ultrasound may also be tried. Acromioplasty, removal of the bursae, or removal of the acromioclavicular ligament is reserved for recalcitrant cases.

Preventing future shoulder injuries requires a combination of stroke technique education, stretching, and strengthening. The swimmer should have his stroke analyzed by an experienced swim coach to correct any stroke mechanics problem. In addition, swimmers should avoid overstretching the anterior shoulder and focus on the posterior shoulder, which is often tight.[11] Due to the freestyle mechanics, most swimmers have stronger internal rotators than external rotators.[22] To counteract this imbalance, the swimmer should focus on strengthening the rotator cuff muscles with specific attention placed on the external rotators. Some swimmers will need to focus on the scapular stabilizers, especially the serratus anterior. A strengthening program should be created with a physical therapist to focus on balancing antagonist and agonist muscle groups.

Shoulder Instability

In recent years, instability of the shoulder has been recognized as a major cause of shoulder pain in competitive swimmers. This is due in part to vigorous flexibility programs as well as the repetitive forceful stretching of the joint capsule caused by shoulder circumduction during swimming. The result is recurrent anterior or posterior subluxation at the glenohumeral joint with corresponding anterior or posterior shoulder pain. Backstroke swimmers are particularly prone to isolated anterior instability, termed *apprehension shoulder*.[12] Repetitive anterior subluxation may result in labral tears.

The pain of glenohumeral instability can be difficult to distinguish from impingement, and frequently the two co-exist.[1] The pain tends to be maximal midway through the pull phase of the stroke and is frequently associated with clicking felt during certain motions. Often the swimmer can relate episodes of the shoulder "going out."[13] On exam the apprehension test should be done in two directions: anterior and posterior. Inferior instability can be demonstrated with the sulcus sign, done by pulling downward on the arm while the athlete sits on the examining table. Inferior instability in combination with anterior and/or posterior instability is defined as multidirectional instability. Swimmers with multidirectional instability are at greater risk for labral tears, Hill–Sachs deformity, or Bankart lesions. It is important to view axillary x-rays to rule out the

latter two problems. If a labral tear is suspected, a CT-arthrogram or magnetic resonance imaging (MRI) can be ordered to confirm this diagnosis.

Initial treatment is conservative with relative rest, ice, and nonsteroidal anti-inflammatory medicines (NSAIDs). Central to conservative treatment is a strengthening program to compensate for shoulder laxity. In general, such a program focuses on internal rotators for anterior instability, external rotators for posterior instability, shoulder abductors for inferior instability, and a balanced strengthening program of the rotator cuff when multidirectional instability is present. With return to swimming, stroke modification may be necessary to prevent recurrence. In swimmers with persistent instability despite a one-year trial of conservative therapy, a surgical procedure to tighten the glenohumeral joint may be indicated.[1] Swimmers must be counseled that a surgical procedure may limit their ability to swim at the same competitive level as prior to surgery due to a reduction in joint mobility.

Hand/Elbow Injuries

Although the majority of upper extremity injuries involve the shoulder, the elbow can be susceptible to overuse injuries as well. The repetitive full extension achieved during the backstroke may cause a triceps tendinitis. During freestyle, the high position of the elbow during recovery may aggravate the ulnar collateral ligament. For both these conditions, conservative management is appropriate. The stroke techniques should be modified to correct any poor mechanics.

LOWER EXTREMITY INJURIES

Breaststroker's Knee

Knee pain in swimmers is most commonly seen in those who do the breaststroke due to the tremendous valgus forces placed on the leg during the whip kick. This kick uses the entire inner surface of the calf to generate forward propulsion, resulting in stress on the medial collateral ligament.[14] Frequently, improper technique plays a role as well. Invariably, the result is a grade I sprain of the medial collateral ligament (MCL), producing pain along the course of the MCL that is increased with valgus stress. Rarely does this injury produce instability. Treatment is entirely symptomatic with icing, NSAIDs, and avoidance of the whip kick. Attention should be paid to correction of poor kick technique. This most often involves incorrectly abducting the hips during the recovery phase of the kick and rapidly extending the knees with the legs apart during the propulsion phase of the kick.[15,16] To reduce knee strain, the swimmer needs to limit hip abduction so the knee goes no further than the hip.

Patellofemoral Pain

Patellofemoral pain is a common cause of anterior knee pain in swimmers and may frequently get lumped into the category of *breaststroker's knee*. In all strokes, but especially the breaststroke, the swimmer pushes the legs against resistance while bending the knee. Combined with pushing off the wall, the stress on the patellofemoral joint can be considerable.[7] The clinical findings and treatment do not differ in the swimmer. Improper execution of kicks is often a factor that can be corrected. Treatment is symptomatic and includes rest, icing, NSAIDs, and quadriceps exercises. In order to avoid the offending kick, the swimmer can train solely with the arms for a short period of time using the aid of a foam buoy. A patella-stabilizing brace made of waterproof neoprene may also be used during workouts.

Foot and Ankle Problems

The most common cause of foot and ankle problems in swimmers is extensor tendinitis due to the repetitive neutral to plantar flexion done in the flutter kick. Symptoms include pain and crepitus over the extensor tendons during flexion and extension. The inflammation of the extensor tendon is best treated symptomatically. Stretching of the extensor tendons may also be helpful. In severe cases, a local cortisone injection around the tendon sheath may be helpful.[1] The swimmer can usually continue workouts with less vigorous or no kicking using a foam buoy. A swimmer may strike the pool wall or gutter during flip-turns and cause a heel contusion. This usually does not limit the swimmer but pain may linger for weeks. Rarely a calcaneus fracture occurs. The governing body for competitive swimming, United States Swimming, prohibits gutters at competitions to reduce foot injuries. Skin lacerations or abrasions on the overlying skin also commonly occur related to injury during flip turns.

BACK INJURIES

Low Back Strain

A variety of swimming movements including flip turns, dive starts, and body rolls in freestyle and backstroke place excess stress on the lower back and can result in pain. Tremendous stresses are also placed on the lower back to maintain body position and motion with the butterfly and breaststroke. The resultant low back strain is treated

symptomatically with rest, heat, and NSAIDs. Muscle relaxants may also be beneficial in the initial stages. The swimmer should institute a back stretching and strengthening rehab program during treatment and continue this routine to prevent recurrences. Abdominal strengthening exercises may also be helpful.

Scoliosis/Scheuermann's Disease

Swimming has been implicated in the development of both scoliosis and Scheuermann's kyphosis. Considerable controversy exists as to whether competitive swimming may actually worsen the scoliotic curve.[17–19] Becker et al showed a small increase in the incidence of scoliosis in adolescent/preadolescent swimmers, suggesting that direct external forces may affect skeletal growth in young athletes.[17] However, over the past few decades, no studies have demonstrated that sport participation directly worsens scoliosis or kyphosis beyond the expected natural history of the disease.[18] Wood and his colleagues[18] encourage swimming and other sports for the benefit of strength, flexibility, and endurance.

Spondylolysis/Spondylolisthesis

The hyperextension required during a variety of strokes, particularly the butterfly, predisposes the swimmer to spondylolysis. It can be unilateral or bilateral and is most commonly found at the L-5 vertebrae. Pain typically occurs at the belt line and is worsened by starts and flip turns.[9] Treatment starts with rest and avoidance of starts and flip turns. The butterfly and breaststroke should also be avoided. Spondylolysis rarely progresses to spondylolisthesis. Depending on the severity of symptoms, age of the athlete, duration of symptoms, and response to conservative treatment, a swimmer with progression may require a brace or surgical correction.[20]

COMMON MEDICAL PROBLEMS SEEN IN SWIMMERS

Respiratory Issues

Exercise-Induced Asthma

Athletes with exercise-induced asthma (EIA) often gravitate to swimming activities because their symptoms are not as severe in the warm, humid conditions of enclosed pools. Coaches need to be aware of the asthmatic swimmer and assure that appropriate medication is poolside.

Infections

Conjunctivitis

Infectious conjunctivitis is rarely transmitted by pool water, as most germs that cause pink eye are killed by chlorine.[21] A true bacterial conjunctivitis may be difficult to differentiate from chemical conjunctivitis, which will also present as a red eye. Repeated exposure to chlorinated water results in corneal edema and some loss of corneal epithelial cells that can cause mild blurring of vision and a halo effect around lights, along with redness and photophobia. Symptoms improve without treatment over several hours. Goggles may help prevent this problem.

Otitis Externa

Otitis externa, or swimmer's ear, is a common problem in swimmers due to the hours spent submersed in water. The infection is typically due to Gram-negative organisms, fungus, or irritants. Treatment with topical antimicrobials or antifungals is effective. Casual swimmers can return to the pool in 7 to 10 days, while the competitive swimmer may return in 2 to 3 days. To prevent recurrences, swimmers should be advised to dry the ear with a drying agent after swimming using either VoSol otic drops or a mixture of 50% vinegar and 50% alcohol.

Otitis Media

Some young swimmers may have tympanostomy tubes in place for recurrent middle-ear infections. The need for ear plugs in these swimmers is controversial. While Becker et al.[22] demonstrated that swimming without ear plugs did not increase the incidence of otitis media, it is unclear whether competitive swimmers have any increased risk of infection due to the increased time they spend in training. Swimmers with tympanostomy tubes who swim only occasionally should probably use antibiotic drops after swimming, but use on a chronic basis should be avoided due to potential ototoxicity.[23] If the swimmer will be swimming outside of the chlorinated pool environment, ear protection is advised.[24]

Dermatologic Concerns

Sun Damage

Many swimmers train in open water, leaving them susceptible to damaging ultraviolet light. Approximately 20 to 30 minutes before swimming, a waterproof sunscreen with sun protection factor (SPF) of 15 or greater should be applied. Reapplying sunscreen at least every 2 to 3 hours is recommended, more often for continuous water

exposure. Studies to define risk factors for melanoma conclude that the greatest risk is when exposure to intermittent ultraviolet light occurs before the age of 15.[25] Multiple studies have assessed the relationship between melanoma and swimming, ultimately concluding that swimming itself poses no threat.[26]

Athlete's Foot

Tinea pedis may be transmitted from pool decks and showers to a swimmer's bare foot. Swimmers may present with typical lesions, but a recent study showed that 36% of swimmers who tested positive for tinea did not have visible lesions.[27] All swimmers should be encouraged to wear sandals to reduce the chance of spread. Treatment includes topical or oral antifungals.

Swimmer's Xerosis

The body's natural sebum is diluted when a swimmer spends hours in the pool. The outer skin layers become dehydrated, leading to the dryness that most swimmer's experience. A few preventive measures can help reduce swimmer's xerosis. Swimmer's should be advised to reduce the time in the shower after swimming and use warm instead of hot water. After lightly patting the skin dry with a towel, the swimmer should apply bath oil or lotion to help keep the skin hydrated.[28]

Aquagenic Acne

Some swimmers develop oily skin instead of extreme dryness. Treatment involves using mild cleansers and antibiotic acne creams. Oral medication may be necessary.

Green Hair

The pool water contains a combination of chemicals including chlorine and copper. The chlorine may bleach the hair but it is the copper ions that are responsible for the green tint some light-haired swimmers develop.[29] Although only a cosmetic problem, this color change can cause anxiety in swimmers. To remove the green tint, 2 to 3% hydrogen peroxide can be applied and then rinsed out after 30 minutes.

Seabather's eruption

In ocean swimmers, a vesiculopapular pruritic rash can develop several hours after swimming in water containing larvae of the organisms *Edwardsiella lineata* and *Linuche unguiculata*. This seabather's eruption is usually found on areas of the skin covered by the bathing suit. The rash and pruritis may last several days but sometimes up to a few weeks. Treatment is symptomatic with antihistamine, antipruritics, and topical steroid creams.[30]

PROBLEMS OF THE SENIOR MASTER'S SWIMMER

Few sports can attract athletes into their golden years like swimming. The U.S. Masters Swimming (USMS) organization has nearly 34,500 members. The first age group is 19 to 24 and other groups follow in 5-year increments. Unlike swimmers on college teams, these swimmers are swimming without the pressure to win; rather, they seek stress relief, health benefits, camaraderie, and sometimes rehabilitation. Master's programs across the United States sponsor competitions for teams with participants well into the 90s. In contrast to collegiate swimmers, master's swimmers may not place as much emphasis on strength and flexibility training, thus setting themselves up for potential injury. A study comparing shoulder injuries in collegiate vs. master's swimmers concluded that 50% in each group reported having shoulder pain lasting 3 weeks or more despite the fact that the collegiate swimmers trained much more intensely.[31] Degenerative changes, muscle weakness, stiffness, and deconditioning may all contribute to the higher injury rate among older swimmers. The sports physician must also keep in mind various other causes of pain in older swimmers. Degenerative cervical spine may cause referred pain to the shoulder. Arthritis may increase joint pain in the overused shoulder. Cancer, heart disease, and other more serious disease processes should also be included in the differential diagnosis of pain.

ISSUES IN ENDURANCE EVENTS

Triathlons

In 1974, 46 athletes participated in the first known triathlon. A few years later, in 1978, 12 athletes completed the first Ironman, which consisted of a 2.4-mile swim, 112-mile bike, and 26.2-mile run.[32] Since that time, the popularity of triathlons has dramatically increased on an annual basis. The Hawaiian Ironman recently attracted 1500 participants and turned away many thousands more. USA Triathlon, the governing body of triathlons, has over 29,000 members. Triathletes may develop overuse injuries, traumatic injuries, and problems due to open-water challenges. As the ultimate cross-trainers, triathletes generally have the ability to rest from the offending activity if an injury occurs. A study of Seafair triathlon participants in 1986 showed that 49% suffered a training

injury that caused them to stop training for at least one day, take medications, or attain medical assistance. Running was the cause of 70% of these injuries.[33]

Open-Water Swim Events

Open-water events are defined by FINA (Fédération Internationale de Natation Amateur) as any swimming event that takes place in rivers, lakes, and oceans. Various mental, physical, and environmental challenges must be overcome to successfully complete an open-water event. Famous open-water swims include crossing the Catalina Channel, the Bering Strait, and the English Channel. Athletes face the possibility of ingesting contaminated water causing gastroenteritis, becoming hypothermic or dehydrated, being kicked by another swimmer, or developing fatigue that could lead to drowning. Swimming in ocean water presents the problem of marine bites or stings, resulting in localized pain and rarely leading to anaphylaxis.[34] Swimmers may inadvertently ingest water contaminated with *Giardia*, *Salmonella*, *Shigella*, *Leptospira*, or *Crypotosporidium*. All but *Leptospira* cause gastrointestinal distress, while leptospirosis is a febrile illness with abdominal pain, myalgias, and chills. In 1998, the largest outbreak of leptospirosis occurred in Springfield, IL, affecting 12% of 876 triathletes who were contacted.[35]

Factors that increase the risk of a swimmer becoming hypothermic include the temperature of the water, wind chill, duration of exposure, fatigue, and the swimmer's ability to conserve body heat. The minimum water temperature suitable for safe competition is defined by FINA as 14°C.[36] A swimmer with mild hypothermia (core body temperature 32 to 35°C) will have tachycardia, peripheral vasoconstriction, and increased shivering which may not become evident in the water. However, the moderately hypothermic swimmer may show signs of altered mental status. Supervising personnel should monitor the water for swimmers changing course or not responding appropriately to directions. Severe hypothermia can occur quickly and put the swimmer at high risk of drowning.[37]

OTHER AQUATIC SPORT INJURIES

Water Polo

Water polo has many different types of injuries due to the aggressive nature of the sport. Like swimming, most of the injuries involve the upper extremity. The water polo player moves the length of the pool in quick swim sprints, following the course of the ball. The overhead throw is similar to that of a baseball player, yet the water polo player lacks the advantage of firm ground and the ability to use the legs and torso to help generate power. It is not uncommon for the water polo player to develop swimmer's shoulder, rotator cuff injury, or traumatic dislocations.[38] According to Colville and Markman,[38] based on 15 years of experience with the U.S. National and Olympic Water Polo Teams, the incidence of shoulder pain in elite water polo players approaches 80%.[38]

The rules of water polo allow opponents to treat the arm as part of the ball and pull, grab, and twist the arm or hand to free the ball. This invariably leads to injuries. To block a ball, players spread their fingers as wide as possible. Consequently, many traumatic injuries involve the metacarpals and phalanges, including dislocations, fractures, and lacerations. Players may also sprain or rupture the collateral ligament of the elbow or the thumb. The most common lower extremity problem among water polo players is medial knee pain. The "eggbeater" kick places a valgus stress along the medial collateral ligament which can lead to sprains and sometimes meniscal tears.

The close contact among players leads to frequent collision injuries. Players are at risk for facial lacerations, conjunctival lacerations, ruptured tympanic membrane, head trauma, and spine trauma.[39] Spine immobilization in the water is challenging because the medical staff must rely on the pool participants to bring the injured player to the poolside in the event of a serious injury.

Synchronized Swimming

Synchronized swimming has evolved from a Hollywood performance to a competitive, complex sport recognized in Olympic competition since 1984. The typical elite synchronized swimmer trains between 3 to 5 hours, up to 6 days a week, incorporating aerobic workouts, weight training, flexibility programs, and performance skills training. The most common injuries seen are lumbar strain, patellofemoral syndrome, and shoulder instability.[40] Muscle strains involving the quadriceps, hamstring, and groin occur due to the quick movements and thrusts during routines. Due to the aesthetic nature of this sport, it is important for the clinician to monitor for signs of eating disorders in these athletes.

SUMMARY

The majority of swimming injuries are due to overuse of the shoulder, knee, and back. Treatment is generally conservative and coupled with an aggressive rehabilitation program. The coach, trainer, and physician work as a team to return the swimmer to the aquatic environment. Prevention of recurrent injury often involves stroke modifications.

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24 THE THROWER

Robert E. Sallis and Anne S. Boyd

INTRODUCTION

Throwing is a central activity in many sports and can involve different techniques depending on the sport. The javelin thrower utilizes a straight arm throw, football and baseball players utilize the rotation of their shoulders to propel a projectile throw, the hammer thrower spins and utilizes the centrifugally induced throw, and the shotputter uses an explosive push from the shoulder to propel the shot. All of these throws involve the extremes of motion and subject the arm to a variety of stresses that can result in both acute and chronic overuse injuries. The shoulder and elbow are subjected to the greatest amount of stress with throwing and therefore are most frequently injured. It has been said that the margin between throwing hard enough to get a major league batter out and not so hard as to cause injury is very small.[1] Thus, with almost every pitch, a major league baseball pitcher pushes the limits of his arm to near the maximum tolerable stress. When one considers the number of pitches thrown during the career of an elite pitcher, it is amazing how resilient the throwing arm can be.

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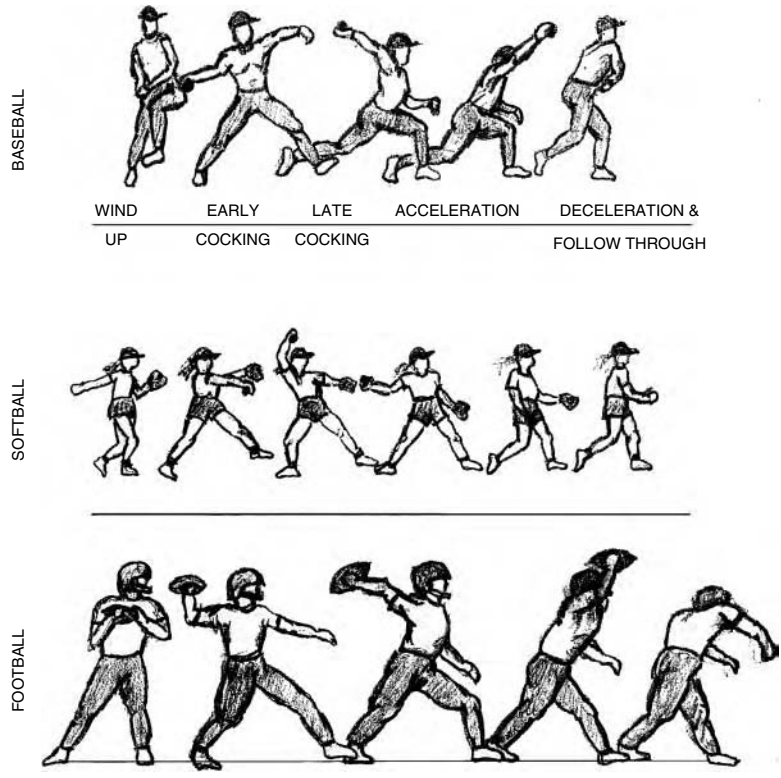

Figure 24.1 Phases of throwing for various sports.

BIOMECHANICS OF THROWING INJURIES

The act of throwing is a complex motion that involves the entire body in various degrees. It requires the generation of kinetic energy in order to propel the ball with velocity. This energy is generated mainly by muscles in the lower extremities and torso and released through the throwing motion. After release of the ball, the remaining energy must be dissipated. Injury can occur if the muscular generation of kinetic energy is done improperly, through incorrect transfer of energy through the shoulder and elbow to the ball or by inappropriate dissipation of remaining energy following release. The maintenance of correct biomechanics during the throw helps protect the thrower from injury.[2] For this reason, a basic understanding of the biomechanics of throwing is extremely helpful in the diagnosis of the injured throwing athlete. The throwing motion can be broken down into four basic phases: cocking, acceleration, release/deceleration, and follow through. Isolating symptoms to one of these four phases can be helpful in making the correct diagnosis in the injured throwing athlete. A fifth phase (wind-up) occurs in baseball pitching and is mentioned for completeness (Figure 24.1).

Wind-Up Phase

During wind-up, the pitcher gathers his arms close to the chest, draws one leg inward, turns his chest away from the plate and prepares for the throw. This phase is vital to "coil" the body and produce the energy that will be released in the throw. This phase is rarely the cause of injury.

Cocking Phase

Cocking begins at the end of wind-up and is completed when the throwing arm is at its most extreme external rotation. This phase can be divided into early and late stages. Early cocking is where the ball is brought back with arm abduction and external rotation. In late cocking, the shoulders begin to rotate forward to face the batter, the contralateral "stride" leg is planted, and the arm progresses to maximal external rotation. The combination of abduction and external rotation results in anterior translocation of the humeral head on the glenoid, and the subsequent rotation of the torso results in shearing forces

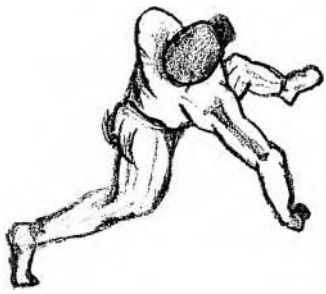

Figure 24.2 Overhead view of throwing motion: axis of the arm in relation to the body.

across the anterior shoulder. These motions may cause stretching of the joint capsule and rotator cuff tendons and lead to anterior subluxation and anterior cuff tendinitis. Over time, repeated subluxation can also damage the anterior labrum and capsule (Bankart lesion) as well as the underlying glenoid fossa (bony Bankart lesion) and can occasionally lead to the "dead arm" syndrome.[3] The long head of the biceps tendon is also placed under extreme tension during the cocking phase which may result in tendinitis and even tendon subluxation.

Acceleration Phase

This phase begins after maximal humeral rotation and ends with ball release. Because of the tremendous force generated through momentum transfer, this phase seems to account for the most injuries. During this phase, the trunk and shoulders are brought forward as the arm lags behind (Figure 24.2). The triceps, pectoralis major, serratus anterior, and latissimus dorsi all fire maximally during the acceleration phase. Tears at the insertions of these structures (particularly the latissimus and pectoralis major) are not uncommon and may produce local pain at this phase of the throw. The supraspinatus tendon is frequently irritated by the undersurface of the coracoacromial arch during rapid internal rotation and may lead to the impingement syndrome. Microtrauma with internal rotation may also cause grinding of the labrum. Acceleration also imparts a combination of rapid extension and valgus strain to the elbow. The resultant load is transmitted as a distracting force to the medial structures of the elbow (primarily the ulnar collateral ligament) and as a compressive force to the lateral elbow (radial–capitellar joint). This may predispose the thrower's elbow to strains or tears of the ulnar collateral ligament (Figure 24.3). Forearm flexor muscles pulling at the medial epicondyle can cause epicondylitis or avulsion. At the lateral elbow, the compression forces can cause osteochondritis of the anterolateral capitellum or Panner's disease in the young athlete and eventually cause loose bodies and arthritis.[5]

Release and Deceleration Phase

This phase begins with ball release (at the critical point of maximal energy transfer from trunk to hand), immediately followed by deceleration of the humerus through powerful contraction of the rotator cuff muscles. Deceleration is the most violent phase of throwing, with forces as much as twice those in the acceleration phase acting to prevent glenohumeral dislocation.[3] Greatest stress is placed on the posterior shoulder, although the stride leg aids in deceleration.[4] The posterior rotator cuff muscles and capsule can be inflamed as the humeral head is forced backward, leading to a posterior cuff tendinitis, impingement, and eventual tears in the posterior rotator cuff muscles (supraspinatus, infraspinatus, and teres minor).[6] Some throwers may develop posterior subluxation, which is associated with throwing discomfort and soreness. Also, the scapulothoracic muscles are very important during deceleration because they stabilize the scapula, control scapula protraction, and provide a stable base for the rotator cuff to decelerate the arm. During this phase, the elbow is also rapidly decelerated by the biceps, brachialis, and brachioradialis.[5] Inflammation and pain can result from repeated stress to these muscles, particularly with a weak rotator cuff.

Improper technique during a pitcher's release can accentuate the stress of throwing and directly lead to injury. Common errors resulting from pitching mechanics include opening up too soon, opening up too late, and overthrowing. During a pitch, the planted foot should always be facing home plate. A player who opens up too soon places the planted foot toward the first-base side (for a right-handed pitcher), allowing upper body rotation too early in the throw. This forces the arm to remain behind the pitcher for a prolonged time, necessitating a whip-like motion and creating increased stress across the anterior shoulder and elbow. Conversely, opening up too late occurs when the pitcher places his foot toward third base, slowing down the rotation of the torso. This effectively eliminates much of the body momentum provided to the pitch and forces the pitch to be thrown entirely by the arm, often stressing the posterior shoulder. Overthrowing or a "bullwhip throw is seen often in pitchers who

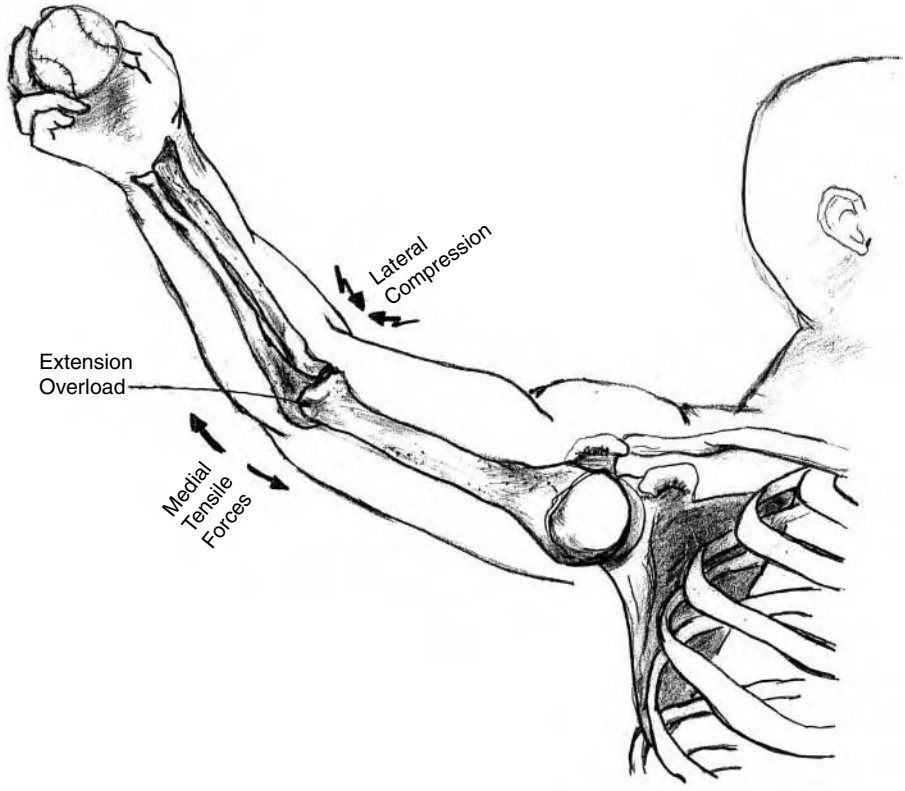

Figure 24.3 Forces on the elbow.

are trying to get more velocity on a pitch. Here, the player plants the stride leg landing on the heel and causes hyperextension of the knee. This causes a sudden deceleration of the body that considerably stresses the throwing arm, particularly the anterior shoulder.

Follow Through

During the follow-through phase, the body moves forward with the arm to dissipate any remaining kinetic energy from the arm back through the body and into the trailing leg. The follow through adds repeated stretch to the posterior shoulder and to the elbow, as the elbow is pushed into extension with pronation of the forearm. Shoulder pain during the follow-through phase is most often the result of inflammation of the teres minor, the long head of the triceps, or the posterior shoulder joint capsule (Figure 24.4). Elbow pain is most often related to irritation of the olecranon fossa caused by forced elbow extension.

SHOULDER INJURIES IN THROWING

Rotator Cuff Injuries

Injuries to the rotator cuff are common in the throwing athlete, occurring during all phases of the throwing motion. Rotator cuff tendinitis typically begins with pain while throwing and may be accompanied by complaints of early fatigue or loss of pitching control.[6] Changes in the thrower's release may also play a role, as can the presence of glenohumeral subluxation.

Local inflammation of rotator cuff tendons and nearby structures, with concomitant edema, can decrease the space under the coracoacromial arch.[7] As these structures get pinched between the arch and the humeral head, pain occurs with overhead shoulder motion (i.e., impingement syndrome). A rotator cuff tear may result from chronic impingement, beginning as a partial cuff tear and progressing to a complete thickness tear.[2] Pain tends to be a persistent dull ache that may bother the athlete even at night.[8]

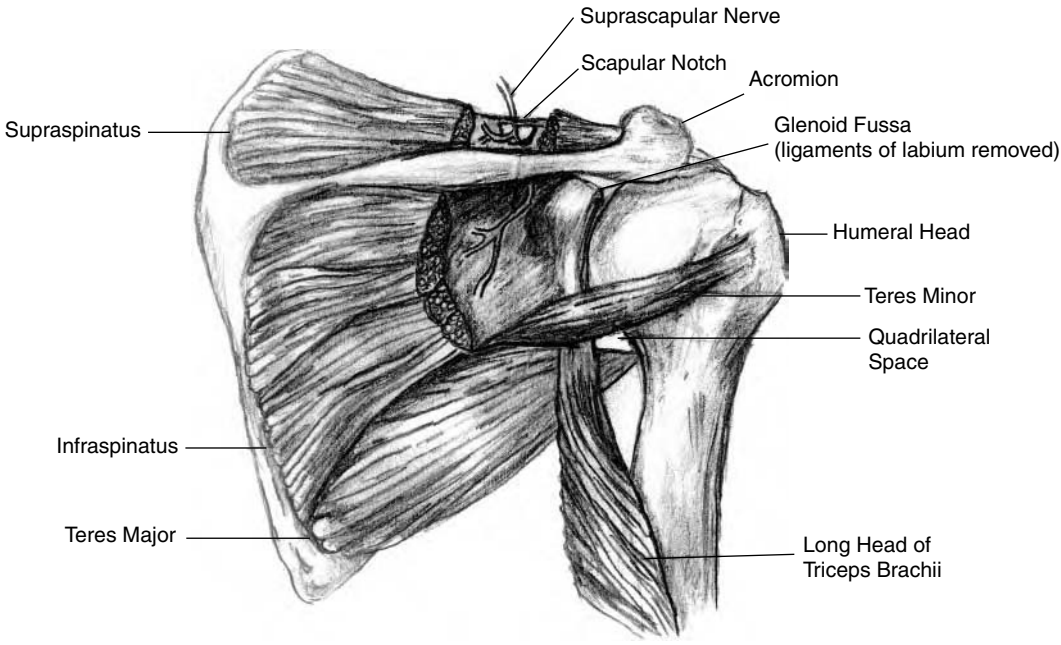

Figure 24.4 Shoulder: posterior view.

The exam will show pain and weakness with resisted rotator cuff motion in patients with tendinitis. Impingement tests (Neers and Hawkins tests) will provoke pain or limit motion when positive. X-rays of the shoulder include an outlet view to evaluate for subacromial bony spurs or a type III (hooked) acromion causing impingement of the supraspinatous tendon. Chronic rotator cuff tendinitis often results in calcification within the tendon that is seen on x-ray. An injected MRI can help confirm a suspected rotator cuff tear.

Treatment of rotator cuff injuries involves a period of rest until pain resolves, followed by a structured rehabilitation program designed to address the athlete's specific needs.[6] The initial rest period can be accompanied by icing and nonsteroidal anti-inflammatory drugs (NSAIDs), along with the judicious use of injectable steroids if needed.[9] Other physical therapy modalities such as ultrasound and electrical stimulation have been used but are of questionable benefit.

Therapy should begin with reinstituting the normal range of motion. Problems with flexibility should be addressed through a proper stretching program, followed by a strengthening program tailored to correct specific rotator cuff weakness.[10] Strengthening exercises are essential in cases of recurrent subluxation.

As pain resolves and flexibility is restored, the athlete should begin a graduated throwing program within the limits of pain. Throwing mechanics should be examined at this time and problems corrected. Surgery should be considered in those with chronic impingement or rotator cuff tears only after a 3- to 6-month trial of conservative treatment.[11]

Glenoid Labrum Injuries

Injury of the glenoid labrum can occur in the throwing athlete but is much less common than rotator cuff injury. Recurrent anterior and posterior humeral head translation during the throwing motion (such as with subluxation) can result in degenerative changes and eventually tears of the labrum.[12,22] Tears may also occur around the upper quadrant of the labrum secondary to forceful biceps contraction (the so-called superior labrum anterior and posterior, or SLAP lesion).[12] Typical symptoms of labrum injury include pain with overhead activity, often associated with a sense of clicking, catching, or locking about the shoulder.[12] "Dead arm" syndrome describes paralyzing pain and numbness, brought on with forceful cocking, that prevents forward throwing motion.[2] Such symptoms suggests anterior subluxation, often associated with a torn anterior labrum.[2,3] X-rays in a patient with a glenoid labrum injury generally are negative. Suspected

labral injury can be confirmed with magnetic resonance imaging (MRI). If MRI is not definitive and labral injury is highly suspect, a second study to be considered would be a computed tomography (CT) arthrogram, which may demonstrate subtle injuries missed by MRI. Initial treatment of labrum injuries should be conservative. Strengthening exercises are performed to reduce recurrent instability. If the athletes' symptoms fail to respond to conservative treatment, surgery may be indicated. Frequently this can be done via the arthroscope and involves repair or debridement of the labrum to a stable peripheral rim.[12]

Glenohumeral Joint Instability

Instability of the glenohumeral joint is a common cause of pain and dysfunction in the thrower's shoulder.[13] Shoulder instability is due to a stretching of the shoulder capsule and glenohumeral ligaments and can lead to excessive displacement of the humeral head as it relates to the glenoid. This may be a congenital condition, the result of repetitive throwing, or due to a traumatic dislocation.[7] Repeated humeral head subluxation can irritate the rotator cuff and frequently coexists with the impingement syndrome. It may also result in glenoid labrum injury from repeated microtrauma. The diagnosis may be missed, as recurrent subluxation tends to present concomitantly with other problems. Most frequently these athletes have pain that is worse during the cocking phase with anterior subluxation and worse during the deceleration phase with posterior subluxation. Tests for shoulder instability (apprehension and relocation tests) should be done on all athletes presenting with shoulder pain. X-rays should include axillary and notch views to look for Bankart and Hill–Sachs lesions.[13] Management frequently involves treating associated tendinitis or impingement as well as attempting to decrease joint instability. A tailored program of rotator cuff strengthening and flexibility is recommended. Those with anterior subluxation frequently have weakness of the internal rotators and a loss of external rotation, while those with posterior subluxation frequently have weakness of the external rotators and a loss of internal rotation.

Biceps Tendinitis, Subluxation, and Tears

Injury to the biceps tendon can also result from repetitive throwing. During the follow-through phase, the biceps must forcefully decelerate elbow extension and forearm pronation. This causes a significant amount of stress on the proximal tendon, which may become irritated and painful, particularly during the follow-through phase of the athlete's throw. True primary biceps tendinitis (acute inflammation without associated rotator cuff pathology) is rare; however, a close association of biceps tendinitis with impingement syndrome has been observed. Although not formally part of the rotator cuff, the biceps tendon is subject to the same mechanical wear beneath the coracoacromial arch as the rotator cuff, the most common cause of tendinosis (tendon degeneration without inflammation) or tenosynovitis, and may lead to biceps tendon rupture. Additionally, because the rotator cuff soft tissues maintain the bicep tendon in its boney canal on the superior humerus, repeated irritation of the rotator cuff can promote subluxation or dislocation of the biceps tendon. [21] This displacement causes wear on the biceps tendon and may cause anterior shoulder pain.

Patients with tendinitis or instability usually present with pain at the bicipital groove that often radiates toward the deltoid insertion and may be worse at night. In this respect, it is often difficult to distinguish it from impingement or rotator cuff tendinitis. An audible or palpable snapping sensation over the biceps tendon as the athlete goes through the arc of motion may indicate biceps instability. On exam, patients will demonstrate point tenderness over the bicipital groove, and provocative tests (Yergason test and Speed test) may help to elicit biceps pathology. X-rays will be negative in primary tendinitis but may show changes indicative of secondary pathology such as a subacromial spur. Interestingly, ultrasound has been shown to be useful in evaluation of both biceps and rotator cuff pathology. Finally, MRI is an excellent, noninvasive method for evaluating the biceps tendon and labral complex.[21]

Treatment for primary and secondary tendinitis is conservative and includes rest, ice, and NSAIDs. When symptoms improve, physical therapy and gentle range of motion exercises may be initiated. If impingement is involved, general recommendations for treatment of impingement are followed. Injection of steroids in or around the bicipital insertion zone is controversial, and more often a subacromial injection is used.[21]

Little League Shoulder

Little League shoulder is a term that refers to stress fracture through the proximal humeral physis. It is caused by degeneration of this growth plate from repetitive throwing. This entity has been reported exclusively in throwers, primarily

between the ages of 11 and 16. Because the proximal humeral physis contributes more to the length of its bone than any other growth plate, damage here puts the athlete at risk for cessation of bone growth and deformity.[7] The young thrower with Little League shoulder usually complains of the insidious onset of pain that is exacerbated by throwing. Diagnosis is made by noting tenderness to deep palpation at the proximal humerus and x-rays that show widening of and sclerosis along the margins of the physis compared to the opposite shoulder. Treatment for this injury is conservative. Extended rest from throwing (at least 6 weeks, and up to 6 months) should be observed until no residual pain is noted and the exam and radiographs have returned to normal. Shoulder strengthening exercises may be undertaken if strength deficits exist, but heavy lifting is avoided.[7]

Effort Thrombosis (Paget–Schroetter Syndrome)

Subclavian vein thrombosis may develop in throwers as the vein is mechanically obstructed at the thoracic outlet.[14] Swelling of the arm, cyanosis, and superficial vein prominence are often only present when the athlete throws and resolve with rest. Diagnosis can be confirmed with Doppler flow ultrasonography or venography.[7] Treatment involves rest, heat, and elevation, often with a 3- to 6-month course of Coumadin®. A thrombolytic agent, such as urokinase, may be useful to lyse clots.[14] Frequently, problems with recurrence arise and may require surgical resection of the first rib or clavicle, which is often a source of compression on the subclavian vein.

ELBOW INJURIES IN THROWING

Medial Injuries

Elbow injuries are common in the throwing athlete. Medial injury due to tensile stress is caused by tremendous valgus strain that occurs during the acceleration phase of throwing (Figure 24.3).

Ulnar Collateral Ligament Injury

The ulnar collateral ligament of the elbow is made up of three bands that make it well suited to withstand strong tensile forces.[5] However, repetitive throwing can cause microscopic tears within the ligament that can lead to eventual destruction. Factors that increase valgus overload at the elbow, such as opening up too soon on release, may hasten this degenerative process. This may lead to partial tears of the ligament or even complete rupture that is often accompanied by an audible "pop" during a throw.[5] Laxity of the ligament or pain can be demonstrated by doing a valgus stress test with the elbow flexed to about 30° to unlock the olecranon, or by using the milking maneuver. Partial tears will result in pain during exam testing, while larger or complete tears will demonstrate valgus laxity. Treatment includes rest, icing, and NSAIDs. As pain resolves, a range of motion and flexibility program should be started to return full motion, followed by progressive resistance exercises to strengthen the musculature about the elbow. Once strength has returned, the athlete should begin a graduated throwing program leading to a full return to throwing.[10] An assessment of throwing mechanics should also be made and errors corrected to help prevent recurrence. Surgery may be indicated in those who fail to respond to conservative therapy or in those with evidence of valgus instability on stress testing.[22]

Medial Epicondylitis

In addition to the ulnar collateral ligament, the flexor muscles of the wrist attach at the medial epicondyle of the elbow, making medial epicondylitis a common injury in throwers. Traction in this area may even lead to avulsion fractures at the epicondyle, a common injury in the adolescent athlete whose secondary ossification center has not fused. Traction may also cause tendinitis within the wrist flexor–pronator muscle tendons that attach at the medial epicondyle.[5] Treatment is similar to that outlined above for ulnar collateral ligament injury. Steroid injection may be used with caution to help reduce inflammation around the epicondyle (Chapter 27). Care must be taken to avoid injection into a ligament or tendon, as this may result in further degeneration and rupture. The close proximity of the ulnar nerve to the medial epicondyle may lead to radiating numbness in the ulnar distribution after lidocaine injection. A regimen of no more than three injections at least 1 month apart may be used in conjunction with rest and rehabilitation exercises.[5]

Ulnar Neuritis

Due to the proximity of the ulnar groove to other medial elbow structures, ulnar nerve injury may also be a source of symptoms in the throwing athlete. Mechanical compromise of the nerve medially from traction, friction, or compression may lead to ulnar neuritis with resultant shooting pain, tingling, and numbness down the forearm to the fourth and fifth fingers.[5] Treatment remains

conservative although patients with persistent ulnar nerve irritation may need surgical decompression or transposition.[5]

Lateral Injuries

Lateral elbow injuries related to valgus-generated compressive forces also occur in the throwing athlete. Symptoms occur primarily during the acceleration phase of throwing when valgus torque is maximal. Osteochondritis dissecans may occur on the anterolateral surface of the capitellum. It is most likely caused by repeated impingement of the radial head against the capitellum during the throwing motion.[15] With time, this leads to breakdown of the surface of the capitellum along with hypertrophy of the radial head. Eventually fragments of the capitellum may dislodge and form loose bodies within the joint, leading to the early development of arthritis.[7,20] X-rays may show cystic changes within the capitellum, irregularity along the radial head, or loose bodies. Clinically the athlete may have minimal pain in the beginning that can progress over years. Eventually, swelling, lack of extension, catching, and even locking may occur if fragments are loose within the joint. Treatment should begin with the standard RICE (rest, ice, compression, elevation) regimen. A period of splinting may be helpful. This should be followed by flexibility and strengthening programs and then a gradual return to throwing. Surgery may be needed for intractable symptoms or when symptomatic loose bodies are present.[5,7] Osteochondrosis of the capitellum (Panner's disease) tends to occur in children ages 6 to 12 and is found almost exclusively in baseball pitchers. It is thought to be due to compressive stresses at a time when the humeral blood supply is limited, causing an avascular necrosis of the capitellum.[7] Panner's disease presents with lateral elbow pain, swelling, effusion, loss of elbow extension, and tenderness at the joint. X-rays demonstrate irregularity of the capitellum and overgrowth of the radial head. Management is primarily conservative. After a period of rest, the involved area tends to revascularize and remodel, and this disorder generally has a good prognosis.

Posterior Injuries

During the follow-through phase of the throwing motion, the elbow is forced into full extension as the forearm is pronated. This results in the olecranon being jammed into the olecranon fossa, causing inflammation and bony breakdown that can lead to osteochondritis dissecans. Loose bodies may eventually form with repeated trauma.[25] Treatment is again conservative and similar to medial and lateral elbow problems. Surgery may be needed to remove loose bodies or spurs that typically occur at the tip of the olecranon.

Little League Elbow

The term *Little League elbow* classically refers to separation and fragmentation of the medial epicondyle ossification center. While the term has been used to describe almost all causes of elbow pain in the young throwing athlete, it is typically seen in Little League baseball pitchers, although it has been described in tennis, javelin throwing, and gymnastics.[16] The young thrower is particularly susceptible to elbow problems during the acceleration phase because muscles and ligaments attach at the medial epicondyle near a growth center. The medial apophysis is weaker and more likely to be injured by the tensile stress associated with throwing.

In the young thrower, traction forces can result in overgrowth of the medial epicondyle and fragmentation of the epicondylar apophysis (Figure 24.3). During adolescence, avulsion fractures through the epiphysis or of the entire apophysis can occur, as well as stretching and inflammation of the ulnar nerve, medial collateral ligament, and flexor pronator muscle group. Much less commonly, compressive forces on the lateral side of the joint can cause osteochondritis of the capitellum and radial head, degenerative changes of the articular surfaces of the radiocapitellar joint, loose body formation, overgrowth of the radial head, and proximal radial epiphyseal closure. Osteochondritis is generally a serious injury, often resulting in chronic pain and disability.[7,15,20]

Little League elbow frequently starts out as an insidious pain that goes unreported because the young athlete is worried about being held from play. Pain and tenderness are generally localized over the medial epicondyle. It is important that x-rays be done of both elbows to determine if an avulsion of the epiphysis has occurred. Treatment is similar to elbow injuries in adult throwers, with an emphasis on adequate rest. A gradual return-to-throwing program should also be strictly followed. Special attention should be given to throwing mechanics and avoidance of breaking pitches (curves and sliders). Prevention of Little League elbow is of prime importance. Pitchers should be limited to six innings of pitching per week, and coaches should encourage their young players to report elbow soreness at once. Proper pregame warm up should be stressed, along with icing after pitching.

HAND INJURIES

Blisters on fingertips are the most common hand injury in the throwing athlete. Repeated curve balls may lead to blister formation on the pitcher's thumb, while a fastball more typically leads to blisters on the fingertips. Blisters are best treated with rest and avoidance of repeated irritation (Chapter 56). If large, they may be aspirated with a sterile needle and covered with a protective bandage. Rare cases of fingertip ulcers have been reported in baseball pitchers. These are probably related to circulatory disturbances, being frequently associated with cold and cyanotic fingers.[17,19] Treatment is initially symptomatic but may require surgical correction.

THROWING INJURY PREVENTION AND REHABILITATION

Conditioning

Effective injury prevention techniques can be extremely valuable in terms of performance and avoidance of injury in the throwing athlete. The first step is a well-rounded conditioning program that includes a cardiovascular as well as a general strengthening program. Special focus should be placed on the shoulder and arm muscles used in the throwing motion. Such a program should be balanced and include flexibility exercises focused on these same areas.

Warm Up

Throwing athletes should be encouraged to take time to properly warm up before beginning to throw. Warm up must include exercises for the cardiovascular system, muscles, and connective tissue.[18] Examples include slow jogging followed by light tossing and flexibility drills before engaging in hard throwing.

Graduated Throwing Program

A graduated throwing program is used when returning a thrower to action after a shoulder or elbow injury. This allows full range of motion with minimal stress.[1] The athlete should start in the outfield and loft the ball gently toward home, getting it there with three or four bounces. This should be continued for 15 minutes or until pain occurs. The next day he should move in a little closer and throw a little harder, with only one to two bounces. This can be advanced each day, provided no excessive pain or stiffness is reported, over several weeks until the player reaches the pitching mound. The athlete can then start throwing with a wind-up at about half speed and then gradually advance to full speed as tolerated.

SUMMARY

The act of throwing subjects both shoulder and elbow to tremendous stress. Understanding the different phases of the throwing motion can be very helpful in pinpointing the cause of pain in the throwing athlete. Nearly all problems in the throwing athlete are initially treated with rest, ice, and nonsteroidal anti-inflammatory medicines, followed with specific flexibility and strengthening exercises as pain resolves. Next, a program is used to slowly return the athlete to throwing. The vast majority of throwing injuries respond to conservative treatment.

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25 THE OUTDOOR ATHLETE*

Sean W. Mulvaney and Janus D. Butcher

INTRODUCTION

The popularity of outdoor sports continues to grow, and they account for the fastest growing segment of recreational life in the United States. These activities span a wide spectrum of difficulty and commitment — from multiday adventure racers pushing the body to the limits of human endurance to a family hike in familiar woods. The reason for pursuing these activities goes deeper than just strengthening the body. The need to get away from crowds or to use strength and wit to move over water, rock, snow, and earth has a strong pull in this fluorescently lit, temperature-controlled world. Because of the many unusual hazards that confront these athletes, the primary care physician advising them must be prepared to address a broad range of concerns.

PREPARATION

Even an experienced sea kayaker in familiar waters can end up in the water. The time to think about thermal protection and immersion hypothermia is not after the kayaker is in the water, with rapidly failing hands that can no longer manipulate the distress flare, an unfavorable current, darkness falling, and no itinerary left at home

* In the second edition, this chapter was authored by Janus D. Butcher.

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with the paramour. It may seem like common sense, but the best time to think about misadventure or accident is before it happens. Doing even a cursory risk assessment and forming a contingency plan before heading out the door can mean the difference between life and death in harsh or seemingly benign environments. While many outdoor athletes compete as part of a small team (two- to five-person) or go on outings as members of a larger party, most train, and even compete, solo. Contingency planning is critical for the solo athlete in the outdoor setting.

Careful pre-event planning involves doing a risk assessment on the specific hazards of the chosen activity and environment. This step is critical for both safety and enjoyment. Both the most likely adverse events (such as heat in the desert or rock-fall on cliffs) and the less likely but potentially serious events, (an injury or illness that prevents ambulation or is life threatening) should be addressed. Leaving an itinerary with a responsible friend is always a good idea. Deciding what contingency equipment to bring along should be discussed with a responsible, experienced practitioner of that activity. A realistic assessment of one's own skill level is critical. A mobile phone can provide a margin of safety in areas with service, but a party large enough for self-rescue is better. Information on climate, terrain, hazardous plant and animal life, endemic diseases, immunizations required, and closest medical care should be researched prior to the event. Several sources for such information can be found in Table 25.1.

TABLE 25.1
Information Sources for Outdoor Travel Risks and Injuries

International Travel Health Guide, Rose, S.R., Travel Medicine, 2001

Medicine for Mountaineering and Other Wilderness Activities, Wilkerson, J.A., Ed., Mountaineers Books, 2001.

Wilderness Medicine, 4th ed., Auerbach, P.S., Mosby, 2001.

Health Information for International Travel 2001–2002, Centers for Disease Control and Prevention, 2001.

Wilderness Medical Society Practice Guidelines, Forgey, W.W., Ed., Globe Pequot Press, 2001.

www.cdc.gov

www.travmed.com

Members of the expedition should be well seasoned in the chosen method of travel or experience should be gained during the preparation phases. Many great trips and expeditions have failed to achieve their goals due to lack of realistic training and physical preparation. Having properly fitting, broken-in gear, especially boots, is one of the most important aspects of preparation. Starting a rigorous trip without breaking in footgear is a set-up for failure. The time to find out that a new pack rubs your neck raw is not on the second day of a 10-day trek.

If remote destinations or extended travel is planned, the medical histories of the team should be reviewed to determine if significant health or safety limitations exist. Specifically, any history of asthma, hymenoptera venom or other allergies, diabetes, heart disease, or other potentially life-threatening medical conditions should be reviewed to ensure that appropriate medical capabilities exist within the group. While few medical conditions exclude the participation of these athletes, careful planning for emergency care may save lives in the field. Basic immunizations should be up to date, including a current tetanus toxoid booster. Special immunizations may be recommended for certain high-risk activities, such as rabies vaccine for cave explorers or travelers to high-risk areas. Current recommendations for immunization and infectious disease chemoprophylaxis should be reviewed if foreign travel is planned (see Table 25.1).

Medical Kits

No medical kit can handle all situations. Medical kits should be tailored to the remoteness of the activity, size of party, special needs, specific environmental threats, and medical skill level available. A realistic and important factor that many physician-advisors fail to account for when recommending a medical kit is the weight and space limitations of small teams. Team members should make hard, well-thought-out choices based on weight limitations before setting out. They should know why each item is being carried and should be able to differentiate between what is really needed and what is merely nice to have. Team members need to realistically evaluate the amount of medical gear carried by the team. Recommendations on the contents of a medical kit are highly subjective. The recommendations in Table 25.2 focus on lightweight, versatile equipment, medication, and supplies. Additional critical personal prescription medications should be carried in the kit in case of loss or damage. Team members

TABLE 25.2
Wilderness Medical Kit

Note: The following would be appropriate for a 3- to 4-person team for up to 6 days moving through taxing terrain in most environments. The emphasis is on lightweight and routinely encountered problems. These recommendations are the result of corroboration with multiple physician and non-physician outdoor athletes, both civilian and military, but the final list is only an opinion, a starting point from which to construct a medical kit suited to the activity. The basic kit recommended will fit in a 10- by 6-inch resealable plastic bag (without the phone or flare).

Item	Notes
Drugs	
Acetaminophen tablets	10 325-mg tablets
Ibuprofen tablets	12 800-mg tablets
Iodine	8 per day per person (for water purification; varies on water source/needs)
Metronidazole	15 500-mg tablets (for giardiasis, amebiasis)
Ciprofloxacin	12 500-mg tablets (for bacterial dysentery)
Amoxicillin/clavulinic acid	8 875-mg tablets (for skin infections, animal bites or significant wounds)
Azithromycin (if member is penicillin allergic)	—
Antacid	12 tablets
Imodium	12 tablets (for symptoms of diarrhea)
Antimalaria prophylaxis	As needed
Ciprofloxacin ophthalmic drops	For anyone wearing contact lenses to treat corneal ulcer
Anaphylaxis kit (epinephrine autoinjector, diphenhydramine)	As needed for allergic team members
Note: Each member is responsible for sunscreen and personal prescriptions with backup of vital prescriptions (i.e., albuterol). Trips of longer duration should include doxycyline to cover rickettsial infections.	
Medical Equipment	
Tincture of benzoin ampoules (or Krazy®Glue; skin preparation for blister treatment)	4
Rolled length of duct tape	20 feet (for blisters, rigging improvised splints/slings)
Moleskin	6 4 × 4-inch pieces.
Elastic wrap	3-inch wide
Steri-strips	1/2 inch
Petroleum gauze	4 × 4-inch or 3 × 18-inch
10% Povidone–iodine (Betadine®)	20 mL; dilute 15:1 with purified water to irrigate wounds after initial irrigation and debridement
Space blanket, lightweight mummy-bag style	For shock, exposure
Small LED head lamp	
Signaling Equipment (Environment Dependent)	
Signal mirror	
One lightweight signal flare (Skyblazer XLT flare)	
Whistle	
Mobile phone/radio transceiver	

TABLE 25.2 (CONTINUED)
Wilderness Medical Kit

Note: This is a pared-down list and is lightweight. Put the pills in small, polyethylene Nalgene® bottles sold at outdoor shops. Clearly label the contents. Put the small bottles in a small, tough waterproof bag. Place the remaining gear in another small waterproof bag. Keep the medicines accessible on your pack so you can treat on the go. Do not underestimate the value of signaling equipment; it can get you out of a situation that the most sophisticated first-aid kit cannot handle.

Item	Notes
Supplemental Supplies	
If space is available, these are highly useful (some might argue essential) items:	
Medicines	—
Nonsedating antihistamine	6 24-hour capsules
1% hydrocortisone cream	1 tube
Toradol	60 mg ampoule (for severe pain)
Diflucan®	1 150-mg capsule (for female team members)
Supplemental Equipment	
High-quality set of tweezers designed for splinters	For splinter and tick removal
Small scissor	—
14-gauge catheter needle	For reduction of tension pneumothorax
Nasopharyngeal airway	—
Triangular bandage	—
Adhesive bandages	4 (200 if young children in party)
Medium trauma dressings	2
Tincture of benzoin capsules	4 additional
Iodine swabs	
Scalpel blade (#10)	
Syringe	5- or 10-cc syringe with one 18-gauge and two 25-gauge 1.5-inch needles
Gauze bandages	3 (4 × 4-inch)
Petroleum gauze	2 additional 4 × 4-inch or 3 × 18-inch

need to be familiar with potential reactions and adverse effects of each drug.

NUTRITION

Travel in the wilderness is associated with very high-energy demands. On 3- to 5-day trips of moderate intensity, nutritional demands will not be critical. Shortfalls in energy will be met by a healthy person's non-vital adipose stores. For higher intensity efforts, efforts at altitude or in cold environments, or longer duration trips, failure to meet energy needs will result in degraded performance and potentially critical depletion of bodily reserves. Energy itself is the key element of the diet, and a lack of sufficient calories results in a catabolic state where the body uses its own lean body mass and fat to sustain itself.

Fluid balance and the need to remain hydrated surpass all nutrition needs. In many outdoor pursuits, potable water is not available and cannot be carried in sufficient quantities for the duration of the trip. The physician should advise the athlete on the importance of having a plan to obtain potable water, by using iodine, filtering, or boiling. The sooner athletes accept that the clearest mountain stream needs to be purified, the sooner they will save themselves the pleasantries of explosive diarrhea and other

more serious waterborne illnesses. Hydration bladders encourage use on the fly and can aid the effort against dehydration.

Wide variations in intensity and technique make predictions of energy expenditure difficult. Personal experience is the best teacher. Some estimates may be found in Chapter 14 (Table 14.6). These give general guidelines that may be helpful in planning for specific energy needs. From human studies at altitude, it has been estimated that the daily net energy deficit in climbers is 2000 kJ/day in men.[1] The energy deficit is secondary to altitude-induced anorexia and increased energy needs of acute high-altitude exposure. The medical literature contains no definitive data regarding advantages of a particular diet composition for either the outdoor or altitude athlete. The diet suggested by the Food and Nutrition Board for minimizing health risks (25 to 30% energy from fat, 12 to 20% energy from protein, and the remainder of energy as carbohydrate[2]) is a reasonable guideline.

Athletes must find foods that are appealing, nutrient dense, and compatible with their environment. The wide variety of quality energy bars and gel packets offers an easy, space-efficient, and energy-dense supplement to the diet. Although energy bars are handy, most of the diet should consist of "real" food. It is critical to test out food items during training to ensure compatibility both with the individual and the activity.

A particular challenge arises in conditions of high-altitude mountaineering, where the energy demands are great but the altitude effects on appetite and cognitive function often result in low caloric intake. In these circumstances, pre-expedition determination of caloric and fluid needs with specific daily menu planning may prevent high-altitude cachexia.[3]

TRAINING

All too often, thrill seekers depart from a sedentary lifestyle and embark on a physically demanding venture without adequate conditioning, which can be very painful and potentially dangerous. Determining when to begin the specific pre-expedition training program is determined by both the level of fitness of the individuals and the magnitude of the trip planned. In order to prevent injuries due to poor conditioning, a complete exercise program should be initiated that has both cardiovascular and strength components. A comprehensive training calendar can help focus the athlete's efforts, beginning with general endurance workouts in the preseason and sport-specific activities later. For example, the recreational telemark skier who plans an extended backcountry trip should emphasize a lower body workout to increase both strength and endurance (with squats, lunges, circuit weight training, and running) in those muscle groups used most (i.e., the quadriceps, lower back). When the conditions permit, the athlete could progress to short routes on skis. The diversity of outdoor activities precludes listing training plans for each sport. Assistance in developing a specific program can be found in periodicals or books pertaining to the chosen sport.

SPECIFIC HAZARDS OF WILDERNESS TRAVEL

Bites and Stings

Snake Bite

Few animals raise as many fears or are associated with as many legends as the snake, which has led to considerable misunderstanding in the lay medical community on appropriate treatment of venomous snake bites. The two families of poisonous snakes found in North America are the Elapidae (coral snakes) and the Crotalidae, or pit vipers. The American Association of the Poison Control Centers documented 1507 venomous snake bites in the United States. Of these, 57% were by rattlesnakes, 33% by copperheads, 6% by cottonmouths, 4% by coral snakes, and less than 1% by unspecified cotalids. During that year, 7% of bites were deemed life threatening, with one fatality.[4] Less than 1% of snake bites in this country are lethal. In the last 30 years, 95% of snake bite fatalities have been secondary to rattlesnake envenomations. In approximately 25% of all poisonous snake bites, no venom is injected (a dry bite).

Snakes generally tend to avoid people and do not attack unless cornered or disturbed. As a result, most bites result from intrusions into the snake's immediate area. More than 50% of victims are 17 to 27 years old. In one report, alcohol consumption was involved in nearly 30% of bites.[5] Many snake bites occur below the ankle, and hiking boots offer some protection.

Coral snakes are indigenous to the southern United States and account for approximately 20 to 25 bites each year. Coral snakes are not aggressive and usually only bite if being handled. They have small jaws and small teeth so that they must chew the bite site to envenomate. In a case of possible eastern coral snake (*Micrurus fulvius*) envenomation, elapide anitvenin should be administered. The effects of elapide venom may

be delayed and fatal. In reference to the stripes on a snake, the adage "red then yellow — kill a fellow; red then black — venom lack" helps when differentiating a coral snake from other nonvenomous snakes, but becomes less accurate south of the U.S. border.

The pit vipers (including rattlesnakes, water moccasins, and copperheads) are widely distributed across most of the continental United States and account for the majority of poisonous snake bites. Crotalidae venom is composed of several protein toxins, which affect multiple organ systems. The extent of systemic injury depends on the health and size of the victim and the degree of envenomation (the amount of venom injected), which is influenced by the age and sex of the snake, the number of bites, and time of attachment. Local injury includes both mechanical trauma from the fangs and the effects of venom toxins. Early development of edema at the bite site is common. Edema may progress up the extremity and in severe cases can lead to a compartment syndrome. Myonecrosis and neuropathies (related to venom toxin) develop frequently. Systemic symptoms of weakness, nausea, vomiting, pain, and paresthesias often occur in the first 12 hours and may be enhanced by anxiety. Development of a coagulopathy is indicated by the formation of hemorrhagic blebs near the bite wound. More significant coagulation disorders may cause conjunctival hemorrhages or bleeding elsewhere. Rarely, frank disseminated intravascular coagulation occurs. Severe venom reactions can result in cardiovascular collapse and death due to hypovolemic shock.

Treatment of Snake Bites

The most important consideration following a venomous snake bite is *evacuation as soon as possible* to a facility where definitive care can be obtained. In the field, the victim should be moved away from the snake and quieted. Hysteria should be dealt with by being calm and reassuring. At a safe distance, attempt to identify the snake. The bitten extremity should be loosely immobilized and the victim transported as soon as feasible. *Incision and drainage, capturing the snake, oral suction, ice, tourniquets, constriction bands, and splinting will increase morbidity and should not be used.*[4] First-aid measures have never been proved to decrease morbidity or mortality from crotalid envenomations. Research has found a twofold increase in necrotic and infectious complications that required surgery in patients who received first-aid treatments such as cryotherapy (ice), cutting and sucking, tourniquets, or superficial constriction bands.[4] The use of a venom extractor is controversial. Walking increases venom transit time, so if at all possible the victim should be carried from the site.

Polyvalent crotalidae antivenin (Wyeth Labs) has been standard therapy and should be administered immediately upon diagnosing significant envenomation by any Western hemisphere crotalid. The Wyeth polyvalent crotalid antivenin is produced from equine serum and is associated with systemic reactions, including serum sickness and anaphylaxis. Because large amounts of antivenin may be required, the potential for significant reactions is relatively high. If available, a newer antivenin, Crotalidae Polyvalent Immune Fab (Ovine, CroFab™; Protherics, London), is effective with less associated adverse effects. CroFab is a sheep-derived antigen-binding fragment ovine, which is less immunogenic. Prompt use of either antivenin will decrease systemic absorption of the venom. Other treatment considerations for all snake bites include local wound care, prophylactic antibiotics, and tetanus immunization.

Venomous Insects (Hymenoptera)

Bees, wasps, and fire ants produce venoms with multiple protein and polypeptide components, including histamine, serotonin, dopamine, kinins, and enzymatic proteins. The sting or bite produces a local reaction with instant pain, erythema, and wheal formation. Multiple stings are common, especially with fire ants and the Africanized honey bee ("killer bee"). Large local reactions spreading more than 15 cm radially from the sting site and lasting over 24 hours are common. In sensitized individuals, a generalized reaction with hives and pruritus may occur. Anaphylaxis is an extreme systemic reaction, characterized by airway edema, hypotension, and loss of consciousness. It is estimated that 0.4% of the U.S. population is sensitive to hymenoptera venom, with 40 to 50 deaths occurring each year from anaphylaxis.[6]

Treatment of hymenoptera stings involves immediate removal of the stinger by any method: pulling, pinching, or scraping. Recent literature supports the fact that the faster the stinger is removed the better; the method of removal does not matter.[7] Local care with ice will lessen the immediate symptoms. Oral antihistamines also may help relieve symptoms in severe local reactions. Antibiotics (e.g., erythromycin or first-generation cephalosporins for 7 to 10 days) should only be used if a secondary infection develops,

as manifested by increased erythema, warmth, and fluctuation 48 hours after the sting.

Treat anaphylactic reactions by standard protocols. Individuals with a history of severe reactions or anaphylaxis should carry an epinephrine autoinjector. Immunotherapy aimed at desensitizing these individuals can be effective in preventing life-threatening reactions. Immunotherapy should be considered in cases involving children and other individuals with severe fire ant allergy. In highly endemic areas, fire ants sting over 50% of the population each year and are the most common cause of insect-sting anaphylaxis.[8]

Nonvenomous Insect Bites

Mosquitoes, ticks, fleas, and biting flies are a few of the commonly encountered blood-feeding arthropods. Arthropod vectors transmit life-threatening illness, such as malaria, Dengue fever, rickettsial infections, leishmaniasis, and plague. In areas where these illnesses are endemic, travelers should be familiar with the signs and symptoms of infection. Specific prophylactic measures may be recommended and should be strictly followed when traveling in endemic areas.

Prevention of attack by arthropods involves three components: avoidance, physical barriers, and chemical barriers. Avoidance of swampy areas will reduce insect exposure. Avoidance is not always possible in the pursuit of adventure. The physical barriers most commonly employed are netting, screens, and clothing. Mosquito netting is very effective and should cover sleeping areas. Clothing should fit snugly at the ankles and wrists and be loose everywhere else. Long sleeves should be worn. Light colors are less attractive to biting flies.

Insect repellents are the mainstay of deterrence. According to the Centers for Disease Control, the most effective repellents contain the ingredient *N*,*N*-diethyl-3-methyl benzamide (DEET) at a concentration of less than 35%. Products with higher concentrations of DEET should be avoided, particularly in children.[9] The deterrent effects of DEET application should last 4 hours, but some formulations can last longer than 8 hours. Rarely, children exposed to DEET have toxic encephalopathy. The possibility of adverse reactions to DEET will be minimized if the following precautions are taken: apply sparingly, avoid high-concentration products, and avoid applying repellents to portions of children's hands that have contact with eyes or mouth.[9] In areas with arthropod-borne illnesses, the risk from disease outweighs the risk of a rare adverse effect of DEET use.

Permethrin is an insecticide that can be impregnated into clothing, netting, and screens. This is very effective, and treated garments are not toxic to humans. A properly impregnated garment will retain its effectiveness for over 8 washings. Permethrin should never be applied directly to skin, only to clothing or netting not being worn at the time of application. Do not wear treated clothing until completely dry.

Ticks are vectors for many illnesses, including Lyme disease, ehrlichiosis, Rocky Mountain spotted fever, Colorado tick fever, tick paralysis, tularemis, babesiosis, relapsing fever, and many other illnesses. These illnesses can be largely avoided if the tick is removed shortly after attachment. Inspecting the body twice a day facilitates timely removal. Grasp the attached tick as close to the skin as possible with tweezers (or with fingers if tweezers are not available.) Pull the tick out with smooth, steady pressure; do not squeeze the body of the tick, as this may inject infective organisms.

Rabies

Rabies is a lethal infection resulting from contact with saliva from an infected animal. This usually occurs from a bite but has been reported in individuals licked by an infected animal. Rabies is predominantly a disease of wild and domestic carnivores and is endemic in raccoons, foxes, skunks, cats, and bats in the United States. The vaccination program has all but eliminated rabies in U.S. domestic dogs; however, for an unprovoked attack by an unknown dog the possibility of rabies should be considered during medical treatment. Rabies is extremely rare in small rodents and rabbits. In third-world countries, rabies is a more serious health risk. After establishing a presence in Afghanistan in 2002, the U.S. Army reported 4 new cases of rabies per day among the Afghani residents in Kabul.[10] In India, estimates of annual human rabies cases range from 15,000 to 25,000.[11]

Rabies is an almost uniformly fatal infection once symptoms have developed, so pre- or postexposure prophylaxis is critical. The CDC recommends preexposure rabies immunization for persons living in or visiting (for more than 30 days) areas of the world where rabies is a constant threat. This recommendation may be broadened to include travelers to high-risk areas for even shorter periods who will not have access to medical facilities with sterile needles and safe antiserum. Spelunkers can be unknowingly exposed to

aerosolized rabies virus in moist caves from infected bat populations. Preexposure immunization consists of three 1-mL intramuscular injections of rabies vaccine, given on days 0, 7, and 21.

Postexposure care involves thoroughly washing the wound with large quantities of soap and water, then administration *as soon as possible* of rabies antiserum and rabies vaccination. Washing the wound must not be delayed. Immunization can be delayed 1 to 2 days. If the biting animal cannot be killed or captured and diagnosed, the animal must be assumed to be rabid. U.S. citizens traveling in third-world countries exposed to rabies should go immediately to the nearest American embassy.

General Trauma and Injuries

Simple skin wounds and lacerations occur while moving through the wilds. The potential for infection is increased even in minor wounds. An up-to-date tetanus immunization is important before starting an outdoor event. Consider prophylactic broad-spectrum antibiotics (such as amoxicillin/clavulanate) early in cases of a deep wound, large-area abrasions, wounds to the hand, or any puncture wound.

Lacerations should be thoroughly cleaned; large or deep wounds should be irrigated with a syringe using Betadine® diluted in clean water in a 1:15 ratio. Apply a clean dressing and protect the wound from further injury. Primary closure with sutures in a field setting has a very high rate of infection and should not be done. A wound sutured in a field is not able to drain, heals at a much slower rate, and develops greater scars.

Minor orthopedic injuries such as sprains, strains, and overuse syndromes are frequent side effects of rigorous outdoor travel. Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) are effective in relieving musculoskeletal pain. For 24-hour or multiday endurance events, strongly consider not using NSAIDs for analgesia. Acute gastritis with mild gastric bleeding is not uncommon in long, hard events; this condition is exacerbated by NSAID use (use of COX-1 or COX-2 inhibitors is preferred).

Compression dressings (ace wrap) are useful for minor sprains, but more serious sprains and fractures should be splinted and the patient evacuated to a treatment facility. Splinting materials, such as wire mesh splints or inflatable splints, may be carried in large parties, but small parties should leave that weight in the car and improvise splinting materials as needed. Almost anything can be fashioned into a splint, including backpacks, backpack stays, sleeping pads, sticks, paddles, etc. Duct tape is handy for securing improvised splints. Remember to splint the joint above and below the injury and check distal perfusion at least every 20 minutes as the injury swells.

Minor burns are common in wilderness travel. Candles, cooking stoves, and hot pots all afford burn opportunities. Significant burns resulting in severe injuries and death can result from the use of combustible liquids for starting fires. Wild fires also pose a risk to backcountry travelers. Staying aware of fire conditions, active fires, wind direction, and terrain features such as rivers or blind canyons is important when traveling in the backcountry.

Treatment of minor burns involves immediate removal of the heat source and cooling of the burned area with cool water. Wash the area with plain soap and water. Obvious dead skin should be cut away with sharp scissors. Dress with an antibiotic ointment or cream such as silver sulfadiazine 1% cream. Silver sulfadiazine should not be used in pregnant women in the third trimester or those with sulfa allergy. Cover with fine-mesh gauze or petroleum gauze or occlusive dressing. Watch for signs of infection and consider antibiotic therapy. Change dressings daily.

Carbon monoxide poisoning occurs mainly from the use of combustion heating or cooking devices in a tent, trailer, or ice-fishing house. This colorless, tasteless gas displaces oxygen on the hemoglobin molecule. At 20% carboxyhemoglobin people complain of headache, nausea, and vomiting. When this level rises to 30%, confusion, lethargy, and an inability or lack of interest to self rescue occurs. Carbon monoxide poisoning is a regular killer of tent-bound mountaineers. Gas heaters and stoves should not be used at all in tents and should be used only in structures with proper ventilation.

Hunting Injuries

Hunting is a popular activity, with an estimated 20 million recreational hunters in North America. In this activity, there is potential for many different types of injuries, from twisted ankles to fatal gunshot wounds. Often hunters enter very remote areas and hunt alone, making search and evacuation efforts difficult. A relatively high incidence of cardiovascular deaths occurs among hunters, often attributed to a general lack of prior conditioning.

Gunshot Wounds

The most serious accidents involve firearms. Between 1983 and 1986, 860 fatal hunting accidents were due to gunshot wounds reported nationwide. Additionally, 6132 nonfatal firearm injuries were recorded in recreational hunters.[12] The most common factor involved the victim being mistaken for game or inadvertently entering the field of fire of another hunter.[12] Self-inflicted accidental injuries occur less frequently but are usually related to falls, dropping the firearm, or catching the trigger mechanism, causing the weapon to discharge. These self-inflicted injuries have a high association with tree-stand use.[17] Improper safety procedures or horseplay are also found in many cases. A large proportion (approximately 30%) of the victims are under 20 years of age, which may reflect inexperience or poor judgment in this age group. Malfunction of the firearm is rarely found to be the cause for accidental injury. Other contributing factors are alcohol consumption, hunting from a vehicle, and hunting near residential areas. Accidental injury from improperly stored firearms in the home is a tragic and all-too-common occurrence. The gun owner must ensure that these weapons are unloaded and stored with trigger locks, but preferably in a locked gun safe. Education of children and other people in the house on firearm safety can avoid a tragedy if passive measures fail. Because gunshot wounds are so devastating, prevention is particularly important hunting. Several preventive measures are listed in Table 25.3.

Definitive management of serious gunshot wounds or other significant penetrating wounds requires a multispecialty trauma team. Commonly used hunting weapons include rifles, shotguns, handguns (in some states), and bows. Shotguns account for over half of all hunting-related firearm injuries. This weapon is devastating because of the broad entrance wound generated by the shot spread. At close range, the shotgun causes mainly a crush-type injury with significant disruption of the blood supply, which can result in large tissue losses. The injuries caused by rifles and handguns produce both a crush injury from the penetrating missile and a stretch injury from the temporary cavity formation. Field measures focus on stabilization of the victim by controlling blood loss with stair-stepped measures of direct pressure, elevation, pressure points, and tourniquet and by evacuating the victim to a medical facility. *Look for both the entrance and the exit wounds to control bleeding.* Treat for shock. Evacuate the casualty as soon as possible.

TABLE 25.3
Prevention of Accidental Firearm Injuries

Wear blaze-orange clothing.
Require adult supervision of young hunters under the age of 18.
Require firearm and hunter safety courses for all participants.
Avoid any alcohol consumption while hunting.
Unload weapons before entering a vehicle or house, when mounting a tree stand, or when walking up a steep or slippery surface.
Store all weapons unloaded and locked away from children.
Other safety issues for hunters include:
Older hunters should have a complete medical evaluation before embarking on a rigorous trip.
Develop a preseason conditioning program.
Carry an appropriate medical kit and survival supplies when entering remote areas.
Do not hunt alone.
Leave a copy of the travel route with local authorities if remote destinations are planned.

COMMON DERMATOLOGIC PROBLEMS IN THE OUTDOOR ATHLETE

Plant-Induced Dermatitis

Contact dermatitis, also referred to as Rhus dermatitis, results from contact with leaves of plants of the genus *Rhus* in sensitized individuals (poison ivy, oak, and sumac). These plants contain the allergenic resin urushiol, which produces a type IV, delayed hypersensitivity reaction. The rash is characterized by vesicles in a linear pattern (from brushing by the plant) over a pruritic, erythematous base. The initial reaction may occur as early as 2 days or as late as 24 days after exposure. Contrary to popular lore, it does not spread by scratching, but past lesions may become activated. For effective prophylaxis, wear clothing to protect from contact or wash the exposed area with warm soapy water within several hours after contact. Untreated, the lesions last 3 to 6 weeks. Topical low-potency steroids may be supplemented with antihistamines to treat the associated pruritus. More significant outbreaks (extensive rash on the face, hands, or genital area) can be treated with a 20-day taper of oral prednisone, starting at 40 to 60 mg per day.

Acute Sun Exposure

Overexposure to solar radiation is a common occurrence despite the overwhelming association with permanent skin damage, cataracts, and cancer. While it may be difficult to convince some people of these long-term problems, the acute effects are often undeniable to the victim. Acute phototrauma results from unprotected exposure to sunlight, either direct or reflected. While all ultraviolet wavelengths may cause skin injury, ultraviolet-A (UVA) and -B (UVB) are the most important. UVB radiation penetrates into the epidermal layers and is responsible for the erythema of sunburn. UVA radiation penetrates into the dermis and may pose a risk for long-term injury.[14] Ultraviolet radiation from sun exposure is the number one modifiable risk factor for skin cancer, including melanoma. Melanoma incidence rates increased an average of 2.7% per year in the United States from 1992 through 1998.[15] Factors associated with increased odds of sunburn include greater sun sensitivity, white race, age younger than 16 years, more hours spent outdoors, and highly valuing a tan.[16]

Prevention of phototrauma is accomplished by the use of both physical and chemical barriers and by sun avoidance. Clothing constructed of tightly woven material such as cotton twill can reflect nearly 100% of the UV radiation, but a typical white cotton T-shirt has a sun protective factor (SPF) of only about 5 to 9. Hats or bandannas can protect head and neck areas. Chemical barriers (sunscreens) provide significant protection. These compounds are readily available, practical in most environments, inexpensive, and well tolerated by most people. Sunblocks are graded by their SPF, which indicates the degree of protection offered. The SPF should be at least 15 in a waterproof formulation as a minimum recommendation. The most common components are benzophenones, cinnamates, octocrylene, and PABA esters, all of which are effective in blocking UVB radiation. Some sunscreens use avobenzone, which provides UVA blockage. Use of DEET-containing insect repellent decreases the effectiveness of sunscreen by 34%.[17] Avoiding midday sun (10:00 a.m. to 2:00 p.m.) when practical offers a significant reduction in UV radiation exposure. Education of patients, especially parents of infants and children, on the importance of sun protection and the sequelae of sun exposure, is key in promoting a healthy lifestyle. Increased altitude is associated with a 4% increase in effective UV radiation for every 1000 feet of elevation gain. Weather affects the exposure in obvious ways, but cloud cover may be deceptive; high, thin clouds may reduce UV radiation by a relatively small amount (20 to 40%).

Acutely in phototrauma, vasodilatation results from the release of inflammatory mediators from damaged cells. These chemicals, including histamine and cytokines, are also the cause of the associated pain and itching. Symptoms begin 2 to 6 hours following exposure and peak at 15 to 24 hours. The pain and itching resolve over 2 to 5 days, during which time epidermal peeling may occur. Treatment of mild sunburn generally centers on symptomatic relief. Skin moisturizers are soothing and will protect against the drying effects of sunburn. Oral NSAIDs such as aspirin or ibuprofen will help to reduce the pain and inflammation associated with acute sunburn.

Other sun-related eruptions include photosensitivity reactions, most often related to medications, and the polymorphic light reaction. The polymorphic light reaction, often called sun poisoning or allergy, involves the development of pruritic papules on areas of exposed skin. This often follows the first exposure of the season and not all areas of exposure are affected. These lesions resolve over 1 to 2 weeks. No therapy is usually required. In symptomatic cases, oral antihistamines may be given.

Ultraviolet keratitis (snowblindness) results from prolonged unprotected exposure to bright glaring sunlight. This condition can be very painful and temporarily blinding. The discomfort starts 6 to 10 hours after exposure. Treatment consists of bacitracin or erythromycin ophthalmic ointment or flouroquinolone drops applied to the eye four times a day, analgesia, and dark sunglasses. Symptoms are usually relieved by the second day and treatment can be stopped. Chronic UV exposure can result in cataract formation. Both of these conditions can be prevented with the use of UV-blocking lenses, especially in high-risk situations such as glacier travel.

Chiggers

This common summer dermatosis causes a great deal of suffering in outdoor enthusiasts, especially in the southeastern United States. Larvae of soil-dwelling mites of the family Trombiclidae cause the reaction. In the U.S., the most important species is *Eutrombicula alfreddugesi* (red bug, chigger, harvest mite). The mite larvae are contacted on plants where they climb in search of a host. They attach themselves to the host by a hooked mouthpart or stylosome, through which they feed on blood. The larvae usually only attach for a

short length of time before falling off. The lesions are most commonly seen where the clothing is tight against the skin, such as sock tops and waist line. The mites do not burrow into the skin, contrary to popular lore.

The host's allergic response is initiated by the larva's saliva and stylosome. The characteristic skin eruption is intensely pruritic and initially appears as a weal, often with a central red spot (the mite). Over the course of several hours, papules will form which tend to vesiculate. Often this will be followed by nodule formation that persists for several days. Untreated, symptoms will usually resolve in 5 to 14 days. Treatment includes limiting the extent of initial infestation by immediately removing the mites. Washing or rubbing the exposed skin with alcohol at the onset of symptoms removes mites. Antiparasitic agents such as lindane are effective but are rarely required. Treatment of the eruption is largely symptomatic, employing antipruritic agents such as calamine lotion or oral antihistamines. In more extensive reactions, oral corticosteroids may be required. Chiggaway® is a commercial product used to treat the associated pruritus. Unproven home remedies include painting the lesions with clear nail polish.

Infestation can be prevented by the use of clothing that limits contact with the mite. Clothing should fit snug at the wrists, ankles, and neck and should be thoroughly laundered after use. Insect repellents are also effective in preventing infestations.

Swimmer's Itch (Cercarial Dermatitis)

Exposure to cercaria, a parasitic schistosomal larvae, results in a local inflammatory reaction associated with intense pruritus. The offending larvae are found in both fresh and saltwater, with snails and birds as the intermediate hosts.[18] This reaction is most commonly seen in freshwater lakes, near the shoreline where aquatic vegetation grows. Itching and erythema occur as these parasites penetrate the skin, often immediately upon leaving the water. Symptoms gradually worsen, with swelling and urticaria frequently reported. More severe reactions may cause blistering. Subsequent exposures result in more intense type IV hypersensitivity reactions.

Treatment involves immediate removal of the cercariae by vigorously toweling off prior to their penetration of the epidermis. This will limit the number of larvae involved. The reaction is generally self-limited, resolving in 1 to 2 weeks, with the pruritus resolving in the first 24 to 48 hours. More severe reactions are treated like contact dermatitis.

Public health measures to prevent outbreaks involve posting or closing beaches where these larvae have been reported. In areas endemic with human pathogenic schistosomiasis (fresh waters in tropical latitudes, especially Africa), avoid freshwater lakes and seek medical treatment when exposed. Praziquantel is curative for these types of schistosomiasis.

FOOT CARE

Moving on foot over significant distance can put stresses on the feet not routinely encountered. Even among conditioned athletes, pain from blisters on the feet can result in the cessation of the outdoor activity and the blisters put the athlete at risk for infection.

Footgear

The first step in preventing blisters is having properly sized, good-quality, broken-in footgear. Properly sized footgear is large enough to accommodate the appropriate socks and account for swelling of the feet from prolonged walking, climbing, or running. Unless experience has shown otherwise, allow for a full size larger boot/shoe to account for foot swelling when purchasing footwear. A boot too small causes the toes to jam forward into the toe box of the boot, especially when going downhill. This results in trauma, blisters, and loss of toenails. Snug boots also reduce circulation and contribute to cold feet in cool environments. In order to break in footgear, wear them around the house and train in them. The breaking-in period can be somewhat shortened by wearing footgear after soaking them in water and by using saddle-soap on leather boots. The sock setup for boots consists of a thin inner sock of a polypropylene type fabric with a thick, quality merino wool outer sock. This setup provides an outstanding combination of wickablity and cushioning that is relatively protective. Keep toenails short and trim them square. Keeping the feet dry with regular sock changes has been shown to reduce blisters, but often dry socks may not be available in the quantity needed.

BLISTERS

Friction blisters occur when the skin rubs against another surface. Initially a reddened hot-spot may develop. If the friction continues, a split in the epidermis results, causing a pale area to develop around the hot-spot and a stinging sensation to

occur. Over the next few hours, the cleft fills with exudative fluid and forms a blister that raises the overlying skin.[19] Group members must be educated to stop and treat any hot-spot as soon as they feel it developing. Pressing on through that initial awareness will lead to increased pathology that will more profoundly slow the group. The heel and great toe are common areas to develop a hot-spot. On the trail, a blister represents a failure to pay attention to earlier warning signs. Experienced athletes should check the feet of younger or less experienced group members. If a hot-spot develops, dry the affected area, apply tincture of benzoin to the area and the surrounding skin, then apply a patch of moleskin or duct tape. Be generous with the size of the patch and trim it so it has curved edges. Do not unroof blisters. Clean the area, and drain the blister by inserting a sterilized needle into a lower margin of the blister, then gently express the fluid. Treat the drained blister as a hot-spot. If the blister is unroofed, treat as an abrasion wound. A doughnut patch around the blister may be helpful.

ALTITUDE-RELATED ACTIVITIES

Hazards related to altitude are discussed in Chapter 10.

WATER SPORTS

Boating, canoeing, sailing, swimming, jet skiing, and diving are a few of the water activities commonly enjoyed. While most of these activities are safe, carelessness and reckless boating operation results in many deaths and injuries every year. U.S. Coast Guard data from 1987 reported 1036 recreational boating fatalities and 3501 boating-associated injuries. Over half of the fatalities were associated with ethanol consumption. Another major contributor to boating deaths is improper use (or lack) of personal flotation devices (PFD). The available information suggests that injuries are most likely to result from collisions between vessels, whereas death is associated with the victim being thrown into the water. Approximately 90% of the deaths in water sports are due to drowning, although trauma is also involved in many of these cases.[20]

Trauma in Water Sports

Aquatic activities are associated with a broad range of injuries, many of which are unique to these sports. Water skiing and diving are associated with the most devastating injuries. Scuba diving has many unique concerns related to barotrauma and nitrogen absorption. Water skiing injuries range from relatively minor sprains and bruises to devastating collisions and propeller trauma. The "skier's enema" or "skier's douche" describes a high-velocity water stream on the perineum, which can result in significant rectal or gynecologic trauma. These injuries are preventable with the use of a neoprene suit. Rope injuries are common and range from burns to amputations. Cervical spine injuries are also reported in water skiers. Extensive soft-tissue and bony injury resulting in partial or complete amputations can result from contact with a propeller. Severe head injuries most often occur when the skier is being retrieved and visual contact is lost.[21] This can be avoided with the use of a spotter in the boat (other than the driver). Spinal cord and head injuries from aquatic sports are devastating and usually preventable. All people, especially youngsters, should be specifically counseled about general water safety, including diving safety. Whitewater kayakers and rafters should be advised to wear helmets. Table 25.4 lists safety measures that can reduce the injuries and deaths associated with water sports.

TABLE 25.4
Prevention of Injuries and Deaths in Water Sports

Learn to swim or tread water.
Do not mix alcohol and water sports.
Do not swim alone; use the buddy system.
Use a U.S. Coast Guard approved personal flotation device (PFD).
Use an approved helmet for whitewater canoeing, kayaking, and rafting.
Take a water safety course before the operation of any water craft.
Use propeller guards on all power boats.
Be aware that water distances are not easily judged and that only good swimmers can swim 200 yards.

Drowning

Over 6500 drowning deaths occur every year, making it the second leading cause of accidental death in this country.[22] The age distribution of these deaths is bimodal, with the highest rates occurring among children under the age of 5 and a second peak in adolescents and young adults. In this latter group, alcohol is involved in over 50% of the cases. Resuscitation measures in the drowning or near-drowning victim include immediate initiation of rescue breathing, started in the water, provided that this does not endanger the

rescuer. The airway should be maintained and the rescue breathing continued while the victim is brought to land. Chest compressions are ineffective in the water and should not be instituted until the victim is placed on a firm surface. Near-drowning victims usually aspirate very little fluid. These patients should be transported to the emergency room as soon as possible, *even if they appear to have recovered*. Significant acute pulmonary edema or infection can occur after a near-drowning episode. If cervical spine injury is suspected, care must be taken to stabilize the neck.

Immersion Hypothermia

Water conducts heat away from the body approximately 27 times faster than air. Accidental cold-water immersion can be rapidly fatal (e.g., 15 minutes or less in 32 to 40°F water) without the use of survival gear and prompt rescue. Survival time can be significantly extended by a few simple safety measures. The basic element in aquatic survival is the use of an approved PFD. These allow reduced movement to keep the head out of the water, which conserves heat. The victim should also assume a heat-escape-limiting posture (HELP): The arms are brought across the chest with the elbows flexed, the knees are brought to the abdomen with the legs crossed, and the neck is flexed forward. This position reduces both activity and free flow of cold water across the body, resulting in slower core-temperature drop and increased survival time.[23] Water athletes, (kayakers, sailboarders, water skiers, etc.) should use thermal protection in the form of a wetsuit or a drysuit in cool water conditions. The use of a survival suit providing flotation and environmental protection is recommended in high-risk environments such as operations on rough seas. In coldwater rescues, in which the victim may be hypothermic and the mammalian diving reflex may be active (e.g., children), resuscitation efforts should be continued until the victim's core temperature approaches normal before considering the efforts futile.

SUMMARY

With millions of Americans involved in outdoor sports and recreational activities, a broad spectrum of associated health problems may confront the primary care physician. Environmental issues include inadvertent plant and animal contact, sunburn, heat exhaustion, hypothermia, near-drowning, and death. The counseling physician should be ready to offer preventive strategies including nutritional advice, pre-trip preparation, foot care, recommended appropriate training and conditioning programs, and creation of a backcountry medical kit.

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SECTION TWO
CLINICAL SPORTS MEDICINE PRACTICE

PART I
SPORTS INJURIES

26 EPIDEMIOLOGY OF SPORTS AND RECREATIONAL ACTIVITY

Richard B. Birrer

INTRODUCTION

The epidemiology of sports injuries entered the scientific era less than three decades ago. Although still a relatively unexplored frontier, athletic injury research has provided not only descriptive information of existing injury patterns but also insights into risk, causality, and potential intervention techniques. At least 215 million Americans participate in one or more sport or recreational activity. In order of preference, the three favorite pastimes are walking, swimming, and bowling.[1] Of the 42 million Americans who are 18 years of age or younger, 7.5 million participate in school sports. The mean beginning participation age is 11 years (range 3 to 18 years). While much of this activity at the adult level occurs sporadically (e.g., during picnics and family outings), structured programs run the spectrum of community-sponsored softball and bowling teams to fun runs and amateur sports teams' activity.

In 1999, 65% of adolescents engaged in the recommended amount of physical activity (compared to only 15% of adults in 1997);[2] 40% of adults engaged in no leisure-time physical activity. Women, individuals with lower socioeconomic

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status, Hispanics and African-Americans, adults from northeastern and southern states, the disabled, and elderly have lower rates of physical activity. Lack of time, unsafe environment, and inaccessibility to a convenient facility are the major barriers sited for decreased physical activity.[2] No accurate annual counts of morbidity and mortality rates for athletic or recreational activities are available, but crude estimates have placed the number of deaths at 5000 to 7000 and the number of injuries at 10 to 12 million yearly.[3] Of these injuries, 30% are treated by the medical profession. Although fragmentary, the data reveal a substantial public health problem.

REPORTING SYSTEMS

At least 20 national or regional sports injury reporting systems are currently operating (Appendix 5). Each has inherent strengths and weaknesses. Definition of terms, sample selection, study design, data collection, and statistical methods differ among studies.[4–7] Specific problems include: (1) the nature of specific problems related to injuries may not be identified; (2) statistically valid injury rates often cannot be established because the number of exposures is unknown; (3) elements of the accident sequence are unclear; (4) severity ratings are arbitrary and long-term effects of trauma are not recorded; and (5) non-emergency-room injuries are not included. Hence, comparability, reliability, and generalizability of these databases are difficult.

INJURY DATA

Table 26.1 identifies common sport and recreational activities in the United States, the estimated number of participants, gender ratio, injury incidence, and prevalence, severity, and peak age group of injury.[4] The majority of injuries occur to children, adolescents, and young adults (5 to 24 years). Children 5 to 14 years of age sustain 30 to 32% of sports-related injuries, with the highest rates being observed in tetherball and gymnastics. Catastrophic pediatric injuries involve not only baseball, basketball, gymnastics, cheerleading, football and hockey but also softball, track, tennis, swimming, and water polo.[8] Sports with augmented speed, collision, and contact (skateboarding, bicycling, all-terrain vehicles, sleds, gymnastics, etc.) are generally associated with more severe injuries. Fall has higher rates of catastrophic injuries compared to winter and spring.[8] Competitive sports have double the average rate of injuries of all sports combined: 16 per 1000 hours of activity. Sprains and strains are the most frequent type of injury over all age groups. Contusions, abrasions, and lacerations account for almost an equal share of injuries in the 5 to 14 age group. In the 5 to 24 age group, 45% of injuries occur during group sports and 55% during individual activities.[9] Basketball (17%) and cycling (16%) are most common in each group, respectively.

The head is injured most frequently in individuals less than 6 years old; upper extremities, particularly the fingers, in the 5 to 14 age group; and the lower extremity, in those over 15 years of age. For all groups, head injury incidence was 258/100,000 (0.22 to 0.52 catastrophic) for basketball, 233/100,000 for bicycle riding, 174/100,000 (0.38 to 0.56 catastrophic) for baseball, 167/100,000 (1.56 catastrophic) for football, 2.78 for ice hockey, 0.61 for track and field, and 102/100,000 for playground equipment.[10,11] No catastrophic injuries were reported for wrestling, tennis, lacrosse, or cross-country at the high school and college level during the same period. Catastrophic neck injuries have an incidence of 0.38/100,000 at the high school level with no fatalities.[12] Peripheral nerve injuries occur in many sports, most commonly football, weightlifting, wrestling, baseball/softball, basketball, biking, downhill skiing, equestrian events, and running.[13] The upper extremity is involved sixfold more frequently than the lower extremity and involves most commonly the median, axillary, and ulnar nerves. Burners, cervical radiculopathy (C-7 most commonly), and peroneal injury follow. Hand and wrist injuries are more common in adolescents than adults;[14] 14.8% of adolescents injure their upper extremity (16% hand, 9% wrist) and 80% of the injuries involve males. Football accounts for the highest number of injuries as well as fractures (52%), dislocations (61%), and sprains (38%). At the Olympic level, injuries to the wrist and hand occur as follows: 25%, boxing; 6%, judo; 13%, weightlifting; 17%, basketball; 17%, volleyball; 12%, ice hockey; and 25%, baseball, with sprains being most common (49%) followed by contusions (15%) and fractures (10%). Severe eye injuries occurring in lacrosse, wrestling, baseball, softball, hockey, racquet sports, indoor cricket, and full-contact martial arts can be reduced by 90% using polycarbonate protective eyewear.[8] The injury rate to the orofacial area is 1.4%; this type of injury is most frequent in the 5- to 14-year-old group and has a 3:1 male to female ratio.[15] 64% involve baseball, basketball, soccer, rugby and hockey. The most common

cause of mandibular fracture is sports-related trauma.

Less than 2% of all treated victims require hospitalization. It is unknown what percentages of injuries are acute, chronic, or due to overuse; however, reinjury rates of the same limb or body part appear to be 2 to 3 times higher than the risk of initial injury. Stress fractures are more common in weight-bearing activities, especially those involving a jumping or running component. The stress fracture rate is 8.3 to 84.9% in track and field activities, 22 to 67.5% for ballet dancing, 21% for figure skating, and 14.3 to 27% for gymnastics, with a twofold higher rate for females.[16] Caucasians have a 2.3 to 24.8% relative risk for stress fracture over African-Americans. The tibia followed by the metatarsal, fibula, navicular, femur, and pelvis are fractured. The riskiest sports are track, jogging/running, dance, Australian football, racquet sports, field events, rowing/canoeing, triathalon, basketball, cricket, and aerobics. Unusual stress fractures involve the coracoid process of the scapula in trapshooting, scapula when running with hand-held weights, first rib from pitching/throwing, second through tenth ribs from rowing/kayaking, pubic ramus in running and ballet, and the pars interarticularis in ballet, weightlifting, gymnastics, volleyball, cricket, and springboard diving.[16]

Risk of injury may not be insignificant for individuals cross-training or participating in other sports for off-season conditioning. Incidence rates range from 50 to 200 per 1000. Unsupervised recreational activities (skateboarding, sledding, sandlot sports, etc.) are associated with significantly higher rates of injury than organized sports events and games.[6] Injury patterns for the physically challenged are similar to those in athletes without disabilities.[17]

GENDER

Since the 1970s, female participation in sports has increased 600 to 700%, whereas male participation has increased a modest 20 to 30%. With the passage of Title IX (1972) forbidding sex discrimination in schools that receive federal funding, 69% of American women now participate in sports or fitness activities. Women, who predominate in skating, gymnastics, dancing, swimming, calisthenics, aerobics, exercise walking, biking, and exercising with equipment, comprise nearly half of new golfers and the majority of new participants in weight training, running, cycling, and basketball. Despite inherent biological and anatomical differences between the sexes, with males excelling in speed and power and females in grace and balance, equal training produces no differences in capacity for improvement in fitness and neuromuscular coordination. Injury profiles for a large number of sports indicate no gender-unique injuries. Certain sports (e.g., basketball, volleyball, gymnastics) are associated with higher injury rates and severity, with injuries to certain anatomical areas (e.g., the knee), and with specific injury types (e.g., sprains, especially of the anterior cruciate ligament, and dislocations in females, and fractures, strains, and contusions in males).[18,19] The rates of serious and catastrophic injury in females is 1/10 to 1/20 of that in males.[10] The rates of injury are higher for females in many sports. Presumably, the differences are due to specific sports, coaching techniques, and the athlete's readiness to participate. Improved evaluation, conditioning, and coaching methods are expected to be preventive.

SPECIFIC SPORTS

Football

Of all injuries, 10.3% occur in football. The number of fatal and catastrophic injuries has significantly declined over the last two decades (1968, 3.4/100,000; 1988, 0.4/100,000). The number of football fatalities outnumbered the fatalities of all other organized sports combined from 1973 to 1980.[4] The number of head (68%) and neck (17%) football injuries is still the highest for any sport. In 1995, the fatality rate for head injuries in high school football was 0.25/100,000 players. The leading cause, subdural hematoma, occurred in 74% of head-injury cases (0.13/100,000) and is associated with permanent cerebral injury.[11] The concussion rate is 5.4 to 6.2 per 100 players, with 10% experiencing a grade 2 or 3 injury. The professional level is associated with a seven- to ninefold increased risk for fatality.[10] Defensive positions are associated with a fourfold to fivefold greater risk of injury frequency and severity; however, the prohibition of primary and initial contact with the head in tackling and blocking and improvements made in protective equipment (e.g., neck rolls), coaching methods, training programs, and medical care are largely responsible for the decrease of catastrophic injuries over the last two decades. The risk of injury rises with age, peaking in young adulthood, and is lower in teams with more experienced coaches and assistant coaches. High school athletes have the highest injury rates. While 50% of injuries involve the lower extremity (knee 36%), 30% involve the upper extremity. Types of injuries include sprains

TABLE 26.1
Most Popular Sport/Recreational Activities in the United States, Listed by Incidence of Injury

Sport	Estimated Incidence per 1000 Participants	Estimated Number of Participants (millions)	Estimated Prevalence[a]	Estimated Mean Severity[b]	Participant Male:Female Ratio	Peak Age Group of Injury (years)	Coefficient of Variation[c]
1. Football	250 (55–810)	6	136	2.3	5:1	5–14	0.05
2. Basketball	230 (40–913)	11	192	1.7	5.7:1	15–24	0.04
3. Baseball	90 (17–125)	20	143	2.3	3:1	5–14	0.06
4. Wrestling	90 (18–270)	2	15	2.7	26:1	15–24	0.08
5. Track and field events	68 (30–170)	2	6	2.1	2:1	5–14	0.11
6. Gymnastics	45 (7–2000)	3	14	2	1:2	5–14	0.09
7. Bicycling	28	20	231	6	2.5:1	5–14	0.06
8. Hockey (all kinds)	28 (19–41)	2	15	2.4	3.6:1	15–24	0.20
9. Swimming/pools	27 (4–57)	120	44	15.8	1.6:1	5–14	0.24
10. Volleyball	25 (1–110)	8	38	1.6	1	15–24	0.08
11. Ice/roller/inline skating	25 (3–80)	4	37	2.2	1:1.7	5–14	0.11
12. Soccer	25 (1–110)	18	40	2.1	2.9:1	5–14	0.09
13. Snowskiing	16 (2–3)	20	20	2.1	1.6:1	5–14	—
14. Martial arts	10	2	8	1.0	4.5:1	25–64	0.08
15. Racquet sports	10 (1–90)	35	17	2	2.4:1	25–64	0.16
16. Dancing	9 (5–20)	6	12	2.5	1:2.7	25–64	0.07
17. General exercise	5	12	26	2.1	2:8.1	25–64	0.12
18. Waterskiing/tubing/surfing	5	5	11	4	3.4:1	25–64	0.14

19. Golf	3	8	11	5	2.7:1	25–64	0.14
20. Bowling	1	21	7	2.6	1	25–64	0.08
21. Beach/picnic/camping	1	15	7	4.4	1.1:1	15–24	0.13
22. Horsebackriding	1	80	20	6	1.2:1	25–64	0.12
23. Playground	N/A	N/A	83	3.1	1.3:1	5–14	0.10
24. ATVs/mopeds/minibikes	N/A	N/A	57	8.6	3.4:1	15–24	0.16
25. Lacrosse/rugby/miscellaneous ballgames	N/A	N/A	41	2.1	2.5:1	15–24	0.14
26. Skateboards	7.5	81	38	3.7	5.7:1	5–14	0.11
27. Fishing	N/A	N/A	27	3.6	4.1:1	25–64	0.11
28. Toboggans/sleds/snow discs/tubing	N/A	N/A	14	4.2	2:1	5–14	0.17
29. Trampolines	N/A	N/A	6	1.7	1.1:1	5–14	0.09
30. Snowmobiling	N/A	N/A	4	6.1	3.3:1	25–64	0.27
31. Boxing	11	N/A	27	2.7	103:1	15–24	0.13
32. Running	9	50	34	1.9	2.3:1	25–64	0.12
33. Amusement park attractions	7	140	10	1.2	1	5–14	0.12
34. Softball	38	9	57	2.2	1.4:1	25–64	0.01
Average	110 (90–130)	24	86	2.4	3.7:1	15–24	0.006

[a] Estimate of injuries per 100,000 population (NEISS data[3]).

[b] NEISS Geometric severity ranking based on diagnosis, body part, and disposition (1 = least severe to 252 = lethal); database discontinued 1999.

[c] Coefficient of variation is a measure of estimated sampling error of the injury estimate expressed as a proportion of the injury estimate.

Note: N/A = not available.

and strains (40%), contusions (25%), fractures (10%), concussions (5%), and dislocations (15%).[20] Risk of injury during practice is higher than during a game. Preseason practice is 5.4 times more likely to result in injury than in-season practice, and injury rates are 2 to 3 times greater among those with prior football injuries than among those without. Non-contact and controlled practice activities have resulted in a 500% reduction in risk of injury over contact and non-controlled activities. Competition is associated with the highest severity of injury.[21] Preseason conditioning, resurfaced fields, and soccer-style shoes significantly reduce injury risk.

Basketball

While the risk of catastrophic or fatal injury is very low, the rate of injury exceeds that of all other sports;[22,23] 17.5% of all non-fatal, unintentional sports-related injuries to males occur in basketball; the rate for females is 9%. More injuries occur during practice, and those injured during games are more likely to be hurt in the second half. Injuries are evenly distributed between the upper and lower portions of the body. Females have a higher incidence of knee injuries, particularly to the anterior cruciate ligament. Sprains are the most common type of injury. The predominant injury type and location is sprain or strain of the ankle or foot. Few published data are available on injury risk factors.

Baseball

Of non-fatal, unintentional sports-related injuries, 4.6% occur in males during baseball; 3.5% in females.[23] Contusions and abrasions of the head and face are the most prevalent injury type and location. Mouth injuries (soft tissue, not teeth) occur with the greatest frequency in baseball. Fatalities in the sport are second only to football and result from being struck by a ball or bat. The cause of death is predominantly cardiac arrhythmia or arrest. Causative factors include failure to wear protective equipment during games and practice and the lack of required chest protection. Little League elbow increases with the number of years played, with the highest rates occurring among those pitching for 6 to 7 years. No trend data or epidemiologic studies designed to evaluate risk factors are available. Of injuries in fastpitch softball, 52% are major disabling ones; 75% occur during away games (5.3% injury rate) and 31% occur during training (5.3% injury rate).[24] Contusions and strains are the most common injury type; 19% involve the knee, and 23 to 47% the upper extremity.

Gymnastics

Of all non-fatal, unintentional sports injuries to females, 5.8% occur to females during gymnastics.[23] Injury rates rank second only to track and field. The risk of injury to females participating in gymnastics is twice that for females in any other sport and 3 times that of males in gymnastics.[25] Rates and severity are directly proportional to skill and performance level. The ankle (18%), knee (16%), low back (10%) and leg (10%) are most commonly injured in the female whereas the ankle (20%), wrist (18%), shoulder (9%) and low back (9%) are more common in the male.

Soccer

Of all non-fatal, unintentional sports injuries to males, 3.5% occur in soccer; 4.8% occur in females.[23] Injury incidence rates vary from 0.6 to 19.1 per 1000 player hours, with a male-to-female ratio of 1:2.[26] The rates are 0.5 to 32 per 1000 hours of play in youth soccer with game rates exceeding practice rates and females exceeding males 2:1.[27] The majority of injuries in soccer involve the lower extremities (61 to 81%), are mild to moderate, and are typically sprains or contusions. More injuries occur during games than during practice.

Track and Field

Although the rate of injury is significantly lower than in contact sports, the injury rate per 1000 participant hours is several fold higher. Most injuries involve the lower extremity (e.g., posterior tibial syndrome and ankle sprain) and occur during sprinting events. Higher levels of performance may be associated with higher injury rates.[28] Change of training program, surface, or shoewear is associated with higher rates of injury.

Wrestling

Heavy wrestling weights are associated with increased injury rates. "Rough-housing" and "take downs" have higher injury rates. Level of participation does not appear to correlate with injury severity or rate.

Racquet Sports

Incidence and prevalence rates increase with age, grip size, and amount of playing time per day. The rate of tennis elbow is about 30/100 (range 9 to 52 per 100). Risk factors (e.g., racquet and

grip size) and preventive strategies (e.g., equipment, exercise) have not been carefully studied. The injury rates for racquetball and squash are 2 times higher than those for tennis. While the lower extremity is most commonly injured, eye injuries and facial laceration are also frequent and appear to be increasing, most likely due to increasing numbers of participants. Lower back pain is the most common problem in young tennis players, followed by shoulder pain in the female, elbow problems in the male, and wrist injury.[29] Overall leg injuries occur approximately twice as frequently as upper extremity injuries.

Swimming

Lack of adult supervision in younger persons, alcohol use in adults, and preexisting illnesses are important in the etiology of drowning and associated aquatic morbidity. The knee is frequently injured (50% of injuries), and the incidence of shoulder pain may be as high as 65%.[30]

Skiing

More reports on skiing and skiing injuries are available in the scientific literature than on any other recreational activity. Over the last several decades, the injury rate has progressively declined (7.6/1000 in 1957 to <2.5/1000 in 1994). Collisions (15% of all injuries) account for the most serious injuries. Risk factors have been identified and include condition of equipment, gender (males, 56 to 75%; the highest rate in almost all studies), experience, ability, environmental conditions, fatigue, and age (inverse relationship). The knee is most commonly injured, especially the anterior cruciate ligament;[31] 3 to 15% of injuries involve the head, with an incidence of 0.25/1000 skier days.[32] Of all skiing injuries, 1 to 13% involve the spine, and 38% are not reported.

Hockey

Little information is available on the incidence and risk factors for this sports category. The head, face, and ankle are the most frequently injured sites. The use of protective gear, particularly helmets, reduces head injury rates by 50%. Spinal injuries from collisions with boards and strikes from behind are often serious. Rule enforcement against spearing is mandatory. Eye injuries are steadily declining. Fatalities usually result from being struck by a puck or stick.

Bicycling

Of all non-fatal, unintentional sports-related injuries, 14.6% occur to males in bicycling; 12.8% to females.[23] Most injuries are minor, but 20% are fractures and 5% concussions. Head injuries account for almost 70% of hospitalizations, but only 10 to 15% of recreational cyclists wear helmets.[33] Males are injured more frequently than females. Of victims over 14 years of age who suffer brain injury, 65% are intoxicated. Mountain biking is associated with rates of less than 1%.[34] Females are more commonly injured than males. Most injuries are superficial abrasions, contusions, and lacerations followed by fractures.

Inline Skating

The inline skating injury rate is 0.3 to 0.4%, with 51% of the injuries being considered severe. The most frequent injury site is the wrist (24%) and lower arm (14%). The most frequent injury type is fracture (41%) and sprain/strain (22%). Injuries involve the novice and intermediate levels most commonly (71%) and result from loss of balance (41%) or striking a stationery hazard (40%) on a street, sidewalk, or driveway (48%).

Dancing

The lower leg (38%), knee (37%), and foot and ankle (17%) are most commonly injured in aerobic dancing and ballet. Ballet dancers have a 100% injury rate at the professional level over the course of their careers, with a 17% risk at any one time. 80% of ballet injuries involve the lower extremity or spine.[35]

Skateboarding

Of all non-fatal, unintentional sports-related injuries to males, 2.8% occur in skateboarding.[23] Injury rates are 3 to 4 times higher in males ages 10 to 14 years than females in the same age group. Of these injuries, 33% occur during the first week of use, and 30 to 35% involve sprains, lacerations or abrasions, concussions, and fractures (50% of all musculoskeletal injuries) of the arms, legs, head (3.5 to 9% of all injuries), and face. Renal injuries are a common cause of hospitalization. Most injuries occur when the skateboard strikes an irregularity in the riding surface. The use of a skateboard park or adequate protective clothing (helmet, knee and elbow pads, and wrist guards) has not reduced injury severity. The American Academy of Pediatrics (AAP) does not recommend skateboarding for children under 5 years of age.[36]

Running

Women under the age of 30 have the greatest risk of overuse injuries, but 60% of all overuse injuries

involve males. Forty-two percent of injuries involve the knee, most commonly patellofemoral function. Training techniques (training errors are the cause of 50% of injuries), running shoes, biomechanical factors, training surfaces, and flexibility or strength are etiologic factors.

Snowboarding

The incidence of injury varies between 3.5 and 40 per 1000 snowboard days. Males are predominantly injured (72 to 90%). Mortality rates are <0.2/1,000,000 snowboarding days. The upper extremity is more commonly injured than the lower extremity The wrist is involved in 20% of all injuries and 50% of all fractures;[37] 33.5% of injuries involve the head; and 1 to 13% of injuries involve the spine.[32] Fifteen percent of all ankle injuries involve fracture of the lateral process of the talus.

Diving

The incidence of injury ranges from 92 to 100%, and the rate increases with increasing years of training and competition. No gender preference has been observed. The incidence rates are higher for pool training than dry land, platform diving than springboard diving, and practice than competition. The shoulder is injured 80% of the time, followed by wrist, hand, elbow, lower extremity, and rarely the spine, which are the most serious injuries. Non-orthopedic injuries predominantly involve the ear (perforations) and eye (corneal microdefects, ocular contusions, and retinal detachment).[38]

Playground Equipment

Of all non-fatal, unintentional sports related injuries to males, 4.3% occur during the use of playground equipment; 8.5% occur to females.[23] Falls, impacts, strangulation, electrocution, and crushing are the major causes of morbidity and mortality. Information on equipment type and risk factors is not available.

Equestrian Activities

Of all non-fatal, unintentional sports injuries to females, 3.6% occur during horsebackriding;[23] 20% of the injuries are severe enough to require hospitalization, and 10 to 20% involve serious head trauma. Annually, 70 to 90 deaths occur.[39]

SUMMARY

Only recently have reliable epidemiologic data on injuries related to sport and recreational activities become available, and they have consisted primarily of descriptive information. More comprehensive data, including incidence, prevalence, and severity of injuries, have been collected for several years in relation to traditional American sports such as football, basketball, and baseball. Unfortunately, only crude overall mortality data are available, and morbidity is difficult to quantify, as only one third of injuries are cared for by physicians. Nevertheless, it is evident that sports activities represent a significant public health problem.

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27 SPORTS INJURIES: A GENERAL GUIDE FOR THE TREATMENT AND REHABILITATION OF SPORTS INJURIES

Francis G. O'Connor and Richard B. Birrer

INTRODUCTION

The rehabilitative process begins at the time of injury. The first 30 to 60 minutes following trauma are referred to as the "golden period" during which injury pathology awaits the full expression of the biochemical mediators of inflammation. Thus, symptoms and signs are minimal while the clinical opportunities for diagnosis, treatment, and, ultimately, recovery are maximal. Unfortunately, many athletes do not present for help until hours or days following their injury. The delay is not without cost; anatomical landmarks are no longer distinct, the mechanism of injury has been forgotten, and perhaps the area has been reinjured. The dictum *primum non nocere* achieves its maximum relevance when applied to the therapy of sports trauma. The physician must understand the basic science of injury and the clinical importance of an early intervention strategy. The athlete must appreciate that drugs are not "magic bullets" and that there is wisdom in sound training practices. Both must understand the quintessential ingredient of time; there are no quick fixes in the healing process.

THE INJURY PROCESS

Macrotrauma

Inflammation is the response of tissue to injury, no matter what the cause or type. The process of inflammation is extremely complex and not well understood, involving a tangled web of many different cell types, metabolic substrates, and pathways. The biochemical phase begins during the first few minutes following an insult. A number of vasoactive substances (amines, kinins, leukotrienes, prostaglandins, anaphylatoxins) and

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chemotactic factors (complement, lipoxygenase products, fibrin, and collagen peptides) are released from traumatized tissue.[1] The vasoactive agents produce initial vasoconstriction and hemostasis followed by increased vascular permeability, transudation, and vasodilatation, while the chemotactic factors facilitate increased mobility and directed migration of inflammatory cells (neutrophils, eosinophils, monocytes).[2] Their release of degradative enzymes (e.g., cathepsins, elastase, collagenase) from lysosomes catalyzes the breakdown of tissue products. An inflammatory "soup" then brews in the tissues, with ingredients being added from damaged and exuding cells. Cellular debris is phagocytized by neutrophils initially. After a few days, monocytes differentiate into macrophages, which continue to phagocytize cells, and connective-tissue fragments.

Pathologically, the various biochemical mediators (prostaglandins, endogenous pyrogen) produce local reactions (the four cardinal signs of inflammation) and systemic reactions (malaise and fever). Pain and edema are major enemies of healing due to the impairment of muscular and joint motion. Bleeding in the form of hematoma and hemarthrosis produces further necrosis, particularly of cartilage, due to ischemic compression. Within 1 to 3 days, the reaction continues with the production of collagen (fibroblasts), new capillary bud formation (endothelial cells), and scar tissue consolidation (macrophages).[3] During the second week, fibroblasts continue to lay collagen on the loose mesh of connective tissue along normal stress lines if proper therapeutic exercise has begun; otherwise, excess disorganized collagen is laid down. By the end of the first month, proliferation slows, vascularity and edema decrease, and tensile strength increases. Scar maturation may require 1 to 2 years — an important consideration in assessing ultimate function.

Microtrauma

Perhaps the best-described model of overuse injury is the distinct histopathology of overuse tendinopathy. The pathology of overuse tendon injury begins as repetitive microtrauma causes a partial thickness tear of tendon that does not involve the outer sheath. In the absence of the rich blood supply of the outer sheath, the tendon attempts to heal via a local response. Microscopic analysis reveals that this local healing response is markedly different from the typical inflammatory cascade.[4] Instead of inflammatory neutrophils, macrophages, and monocytes, regions of tendinopathy contain activated fibroblasts, vascular endothelial cells, fat cells, and cellular debris. Nirschl and Pettrone[5] have termed this collection of fibroblasts and primitive vascular structures *angiofibrioplastic hyperplasia*. The end result of this failed healing response is a weak, gelatinous scar, appearing macroscopically as a dull, gray segment in the normally bright white tendon. Electromicroscopy reveals tendinopathic tendon to consist of randomly oriented collagen fibers and other tissue fragments without inflammatory histopathology.[6] Interestingly, investigators have recently isolated glutamate — a potent mediator of pain sensation in the central nervous system — from tendinopathic Achilles tendons using microdialysis.[7]

MANAGEMENT OF SPORTS INJURIES

The diagnosis and management of sports injuries require a multidisciplinary approach. The sports medicine physician's responsibilities are to establish a correct pathoanatomic diagnosis and direct rehabilitation that enlists the expertise of the directing physician, physical therapists, orthotists, athletic trainers, and coaches. The five-step management pyramid (Figure 27.1) has been designed to return the athlete to sports participation.[8]

MAKE A CORRECT PATHOANATOMIC DIAGNOSIS

Accurate diagnosis and management of most sports injuries require no more than a good history, physical examination, and selected radiographs. A thorough history is the key to successful diagnosis because it allows for correct injury identification that can be confirmed through physical examination and radiographs. Vague diagnoses, such as knee sprain, swimmer's shoulder, or shin splints do not clearly identify the anatomic dysfunction and should be avoided. A recent *Cochrane Review* on the evaluation of shoulder pain interventions noted that more research was needed to establish a uniform method of defining shoulder disorders (Level of Evidence B, systematic review).[9] Diagnoses such as patellar tendinopathy and chronic exertional compartment syndrome more clearly identify the painful, presenting structure; however, even these diagnoses are limited as they may not accurately describe the pathologic process of the injury and offer no insight into associated dysfunctions that occur along the rest of the kinetic chain.

The physician should begin the history by asking the athlete questions that identify the

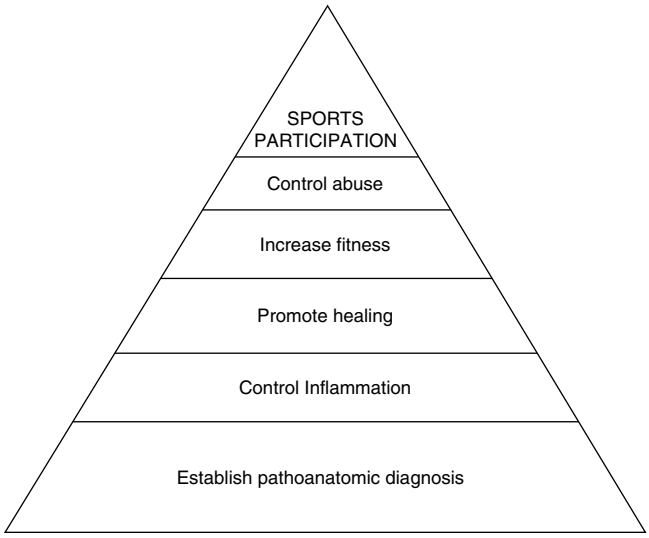

Figure 27.1 Sports injury management pyramid.

mechanism of injury for macrotrauma, or the "transition" that may have contributed to the repetitive overuse microtrauma: When did the injury first occur? Were you able to continue playing after the injury? Did you recently purchase new shoes or a new racquet? Have you changed training locations or your training regimen? Questions should also focus on the quality of the athlete's pain: Does the pain occur only with sport activity, or also with activities of daily living? Perceived pain phase scales, such as Puffer and Zachazewski's,[10] can often be helpful in classifying the injury, determining the prognosis, and gauging rehabilitative progress. Type 1 pain occurs after activity only, type 2 occurs during activity but does not impair or restrict performance, type 3 occurs during activity and is severe enough to interfere with performance, and type 4 is classified as chronic and unremitting.

General health questions should identify recurrent minor illness, sleep patterns, nutritional habits, and overall mood states that may provide clues to an overtrained state. Female athletes should be questioned about a history of stress fractures, menstrual abnormalities and eating habits. These findings are manifestations of the female athlete triad; early identification of the triad — eating disorders, amenorrhea, and osteoporosis — allows for interventions that may prevent considerable morbidity.[11] Determining an athlete's training intensity and goals helps the physician design a rehabilitation program that fits the patient's athletic objectives.

The physical examination seeks to identify the focal problem and uncover contributing intrinsic abnormalities. Using Macintyre and Lloyd-Smith's concept of "victims and culprits" can be helpful in detailed examination.[12] The victim represents the presenting problem, while the culprit is the dysfunction or anatomic abnormality that created the victim. An example would be gastroc-soleus inflexibility (culprit) contributing to plantar fasciitis (victim).

As previously outlined, the entire extremity and kinetic chain must be thoroughly examined for possible culprits when evaluating a specific injury. A runner who presents with running-related anterior knee pain requires a detailed examination of the knee as well as an examination of the entire lower extremity and pelvis. Leg length discrepancies, sacral rotations, hamstring inflexibility, forefoot pronation, and gluteal weakness are only a few of the many potential culprits in this case. The tennis player with elbow pain almost routinely demonstrates weakness in the rotator cuff. Indeed, the tennis player, baseball pitcher, or any overhead athlete presenting with upper extremity pain needs an examination that includes the lower extremity as well as the trunk, as all are involved in upper extremity motion in sports. Assessment of core strength should be included in both upper and lower extremity athletes (e.g., pelvic bridge assessment, single-leg squat test, and single-leg Trendelenberg exam for gluteal weakness). Failure to identify muscle imbalance patterns and structural malalignment

often sabotages an otherwise well-planned rehabilitation program.

As described, the physical examination is primarily biomechanical. On occasion, dynamic assessment such as slow-motion video analysis may be required. Finally, the examination should include an evaluation of athletic equipment, including braces, running shoes, and/or tennis racquets.

Radiographs aid diagnosis and can rule-out related injuries such as fractures, tumors, intraarticular abnormalities, or heterotopic calcification. Only a minority of sports injuries — including combined pathoanatomic presentations or those with clinical evidence of major soft tissue disruption — require more advanced imaging techniques. Electromyographic studies and intracompartmental testing can assist when clinically warranted.

CONTROL INFLAMMATION

Although inflammation may be required for proper healing of sports injuries, an excessive or prolonged inflammatory response can become self-perpetuating and self-destructive; therefore, controlling or suppressing inflammation is one of the primary goals of sports injury treatment. Control of inflammation has received considerable attention in the medical literature. The classic approach is RICE (rest, ice, compression, and elevation); however, we have modified the mnemonic by adding prevention/protection, modalities, and medications to create PRICEMM. Nearly all protocols for managing sports injuries begin with the athlete abstaining from or modifying exposure to abusive activity. Rest, however, does not mean halting all activity. Relative rest protects the injured area, while avoiding the consequences of deconditioning and disuse atrophy.

Functional management, which allows activity while protecting injured structures, is strongly advocated in acute macrotraumatic injuries. Functional management has been shown to be the favorable strategy for treating acute ankle sprains when compared to immobilization (Level of Evidence B, systematic review).[13] To prevent reinjury and ensure better compliance with rehabilitation programs, it is beneficial to emphasize what recuperating athletes *can* do to enhance healing and maintain fitness rather than what they cannot do. Athletes with lower extremity injuries, for example, can frequently duplicate land workouts in a swimming pool.

Modalities and medications may assist in controlling inflammation and are frequently incorporated in the treatment of sports injuries. Their role, however, has yet to be clearly defined.[14,15] No general consensus exists on the use of medications in the treatment of injuries in the sports medicine community. Inflammation is required for healing, which has raised the question as to whether these agents may be counterproductive. In overuse injuries, the histopathology has clearly demonstrated that these injuries are degenerative and noninflammatory. Almekinders,[16] in a detailed review of the present literature on tendinitis, concluded that current treatment methods may not significantly alter the natural history of disease (Level of Evidence B, systematic review). The clear lack of evidence at this juncture makes it difficult to either significantly support or refute the use of pharmacotherpy. The authors, however, as do the great majority of sports practitioners, use both approaches to assist in pain control and inflammation modification to assist the patient in making a quick transition from relative rest to rehabilitative exercise (Level of Evidence C, consensus expert opinion).

Pharmacologic modalities are a mainstay in the management of many sports injuries, with nonsteroidal anti-inflammatory drugs (NSAIDs) being among the most commonly prescribed agents. The two basic classes of NSAIDs (i.e., the carboxylic and enolic acid groups) contain a number of derivatives (e.g., propionic, oxicam, indole, pyrazolone). The NSAIDs block cyclo-oxygenase and hence inhibit the formation of prostaglandins, prostacyclins, and thromboxanes.

Differences exist in NSAID toxicities but little evidence is available clinically to distinguish the anti-inflammatory effects of the various compounds when full therapeutic dosages are employed.[17,18] Gastrointestinal distress due to loss of cytoprotection, headache, mental status change, renal impairment, impaired blood coagulation, and bronchoconstriction are recognized side effects of NSAIDs. Idiosyncratic and allergic reactions are rare. Indomethacin has more frequent side effects (central nervous system and gastrointestinal), and the butazones have more serious toxicities (blood dyscrasias, skin reactions). To date, no well-controlled sports-injury study has identified a superior NSAID. Because of the marked variability in response among individuals, switching NSAID class may produce an improved therapeutic result.

Recent agents on the market include the COX-2 selective agents. While COX-2 safety is the subject of much debate, these drugs have been demonstrated to limit gastrointestinal and nephrotoxic

adverse effects, theoretically by blocking only the COX-2 enzyme.[19] While these agents would clearly offer a significant advantage in the sports community, no data have yet been extrapolated to this population. Empirically, however, these agents would appear to be safer in athletes who have a known history of gastrointestinal toxicity with standard NSAIDs.

Corticosteroids are potent anti-inflammatories commonly prescribed for managing athletic injuries. Their employment in treating sports injuries, however, is controversial, and their role is not clearly supported by objective evidence-based data. A recent *Cochrane Review*, however, did demonstrate that corticosteroid injection therapy does provide superior short-term results in the management of tennis elbow when compared with oral and topical NSAIDs (Level of Evidence B, systematic review).[20] Despite an absence of support from the clinical literature, corticosteroids are widely utilized in sports medicine practice.

INJECTION/ASPIRATION

Aspiration and injection therapy are clinical and pharmacologic adjuncts that can be extremely valuable to the team physician. The procedures are relatively safe and well tolerated and require no special equipment. Sites of aspiration and injection include joints (synovitis), various bursae (bursitis), tendons (tendinitis), and linings of the tendons (tenosynovitis). The drugs most commonly injected are listed in Table 27.1. Their dosage equivalents and half-lives have little relevance in predicting clinical dosage and duration of relief (Table 27.2).[21] The systemic toxicity from a local injection, particularly suppression of the adrenal axis, is minimal. Mechanisms of action include phospholipase inhibition, reduction of prostaglandin formation, stabilization of lysosomal membranes, and fibroblast reduction. A combination of short- and long-acting steroids is often chosen in order to minimize side effects, provide quick relief, and secure a longer lasting anti-inflammatory effect. An anesthetic agent is usually added to the steroid in order to provide immediate analgesia, increase mobility, verify diagnostic accuracy, and serve as a carrier and distribution vehicle. Lidocaine (Xylocaine®) is short acting, whereas bupivacaine (Marcaine®) is long acting. Utmost care must be taken to avoid masking the symptoms of an obscure diagnosis through injection therapy.

Indications

- *Therapeusis:* Relieve pressure, pain, and inflammation and promote healing (range of motion).
- *Diagnosis:* Differentiate the causes of effusion (infectious, inflammatory, traumatic) by aspirating fluid

Contraindications (Relative)

- *Previous site injections:* Either one within the past month or a total of three to four within a year
- *Certain medical conditions:* Sepsis, localized cellulitis, coagulopathy, diabetes, pregnancy, septic joint/effusion
- Acute injuries, unstable joint, fracture, marked juxta-articular osteoporosis, total joint arthroplasty
- Allergy (rare)
- Uncooperative patient

Method/Technique

The site for injection must be carefully chosen, marked (fingernail impression, methylene blue, pen, etc.), and prepped using universal blood and body-fluid precautions. It is not routine to give anesthesia to the skin, particularly with a quick injection; however, ethyl chloride spray or a small wheal of an anesthetic agent decreases the pain of the injection. If an aspiration is planned, the needle should be rotated, advanced, and retracted, while sterile saline is instilled with external pressure applied to the area in order to maximize the removal of clotted blood or mucin and rule out extrasynovial swelling or hypertrophied synovium. Aspirated material should be analyzed (cell count, chemistries, culture) if it is nontraumatic. It is acceptable to use the aspiration needle for injection. A hemostat can be used to steady the needle at the skin surface when the syringe is changed. No resistance should be felt as the plunger is depressed. Amplification of pain or a gritty sensation (calcium deposits) are valuable signs indicating that the needle is "on target." Apply brief pressure to the site and then cover with a sterile adhesive bandage, although the area may be washed without restriction. Take the area gently through a complete range of motion to see if the injection was effective and to distribute the medication. Routine use of the area after an injection is not recommended as it may cause deterioration and further injury due to the masking effect. Rest is relative and should be continued

TABLE 27.1
Drugs Most Commonly Used in Injection Therapy

Steroid	Onset	Peak	Duration	Route	Dose	Potency
Betamethasone (Celestone®, Soluspan®)	1–3 hr	1–2 hr	1–2 wk	IA/IL/ST	IL: 0.2 cc/cm^2 IA: 1–2 cc	25
Dexamethasone acetate (Decadron®)	Rapid	8 hr	6 days–3wk	IA/ST/IL	0.8–16 mg	25–30
Dexamethasone sodium phosphate	Rapid	—	3 days–3 wk	IA/IS/IB/IL/ST	0.4–6 mg	25–30
Hydrocortisone acetate (Hydrocortone®)	—	24–48 hr	3 days–4 wk	IA/IS/IB/IL/ST	5–75 mg	1
Methylprednisolone acetate (Depo-Medrol®)	Very slow	7 days	1–5 wk	IA/IL/ST	4–80 mg	5
Prednisolone acetate	—	—	—	IA/IL/ST	4–100mg	4
Prednisolone sodium phosphate (Hydeltrasol®)	—	—	3 days–3 wk	IA/IL/ST	2–30 mg	4
Prednisolone acetate/sodium phosphate	—	—	3 days–4 wk	IB, IS, IA, ST	IA, IS: 20–80 mg prednisolone acetate + 5–20 mg prednisolone phosphate	4
Prednisolone tebutate (Hydeltra-TBA®)	Slow (1–2 days)	—	1–3 wk	IA/IL/ST	4–40 mg	4
Triamcinolone acetonide (Kenalog®)	—	—	Several weeks	IA/IL/IB/IS/ST	2.5–40 mg (up to 80 mg)	5
Triamcinolone diacetate (Aristocort®)	—	—	1–8 wk	IA/IL/IS/ST	3–48 mg	5
Triamcinolone hexacetonide (Aristospan®)	—	—	3–4 wk	IL/IA	IL: 0.5 mg/in^2 IA: 2–20 mg	5

Note: IA = intra-articular, IB = intrabursal, IL = intralesional, IS = intrasynovial, ST = soft tissue.

TABLE 27.2
Injection Therapy: Locations and Indications

Steroid (mg)[a]	Anesthetic (cc)	Region	Diagnosis	Site of Injection	Needle Size
10–20	1–2	Shoulder	Impingement syndrome	Subacromial bursa	22 gauge, 1–1.5 in.
20–40	1–2	—	Acromioclavicular arthrosis	Acromioclavicular joint	22 gauge, 1–1.5 in.
5–10	1–2	—	Bicipital tendinitis	Biceps tendon	22 gauge, 1–1.5 in.
20–40	2–4	—	Rotator cuffitis	Rotator cuff tendons	22 gauge, 1–1.5 in.
15–30	2–4	Elbow	Epicondylitis	Extensor muscle origin	25 gauge, 1–1.5 in.
40–60	4–10	—	Joint	—	25 gauge, 1–1.5 in.
5–10	1–2	Wrist	DeQuervain's syndrome	First extensor compartment	25–30 gauge, 0.5–1 in.
5–10	1–2	—	Flexor carpi ulnaris tendinitis	Flexor carpi ulnaris tendon	25–30 gauge, 0.5–1 in.
20–40	1–2	—	Joint	—	—
5–10	1–2	Hand	Flexor tenosynovitis	Tendon sheath	25–30 gauge, 0.5–1 in.
5–15	1–2	—	Ganglia (trigger finger)	—	25–30 gauge, 0.5–1 in.
4–8	0.5–1	—	Joint	—	25–30 gauge, 0.5 in.
10–20	2–4	Knee	Patella tendinitis[b]	Infrapatellar fat pad	22 gauge, 1–1.5 in.
10–20	2–4	—	Iliotibial band tendinitis	Iliotibial band Insertion fibula head	22 gauge, 1–1.5 in.
40–60	4–10	—	Joint	—	20–22 gauge, 1–2 in.
10–20	1–2	Ankle	Achilles tendinitis[b]	Retrocalcaneal bursa	22 gauge, 1–1.5 in.
15–30	2–4	Foot	Plantar fasciitis	Calcaneal origin of plantar fascia	25–30 gauge, 1–1.5 in.
5–15	1–2	—	Ganglia	—	25–30 gauge, 0.5–1 in.
4–8	0.5–1	—	Joints	—	25–30 gauge, 0.5 in.
10–20	1–2	Hip	Trochanteric bursitis	Trochanteric bursa	20–22 gauge, 1–1.5 in.

[a] Methylprednisolone acetate (Depo-Medrol®) 40 mg/mL.

[b] Rare to inject these sites as the potential for tendon rupture is significant.

Note: Use a 20- to 30-cc syringe for aspiration with an 18- to 20-gauge 1- to 2-inch needle.

overnight for small joints, several days for medium-size joints, and several weeks for large, weight-bearing joints.

For cases of tendinitis and tenosynovitis, the injection is made obliquely into the tendon sheath or surrounding tissue rather than vertically into the tendon substance. Movement of the needle with tendon movement indicates an intratendon position, indicating a need to withdraw the needle! Aspirate first to ensure that the needle is not in a vessel. Ganglia should be directly injected. Often the cyst disappears due to a single puncture; multiple cyst punctures with aspiration followed by steroid instillation may be necessary, however.

Complications

- Postinjection steroid flare (2 to 10% of injections), which occurs more commonly with suspensions and can be reduced with concomitant use of lidocaine and RICE
- Crystal steroid athropathy (0.8%), which occurs with suspensions
- Infection, sterile abscess (<0.1%)
- Skin changes (<1%), such as hypopigmentation, hyperpigmentation, or atrophy
- Necrosis of cartilage (weight-bearing joints), nerve, bone, and tendon (<1%),

including rupture secondary to injection pressure or catabolic effect of steroid
- Steroid flush (triamcinolone acetonide; <1%)
- Asymptomatic pericapsular calcification (40%)
- Uterine bleeding, posterior subcapsular cataracts

Athletes with injections adjacent to a weight-bearing tendon (e.g., Achilles, quadriceps) should be restricted from their sport for 2 to 3 weeks. Treatment failures are most frequently the result of failure to enter the appropriate space or trigger area. The best results are achieved when injection therapy is used adjunctively with other pharmacologic agents and a well-designed physical therapy program.

Miscellaneous Agents

Dimethyl sulfoxide (DMSO) is both a drug carrier and a drug. It can penetrate the keratin layer of the skin within seconds, carrying anesthetics, steroids, and other drugs. As a drug, it is analgesic, anti-inflammatory, and bacteriostatic. While the drug has U.S. Food and Drug Administration (FDA) approval only for the topical treatment of traumatic swelling in horses, DMSO enjoys a wide range of uses in sport subcultures. Common side effects include dysgeusia (garlic taste), halitosis (fish odor), and transient erythema and paresthesias at the site of application. DMSO may be teratogenic. Oral proteolytic enzymes such as trypsin and chymotrypsin are not recommended, as gastric denaturation makes them ineffective except perhaps in very large doses early in the inflammatory process. The injection of 1 to 2 cc of hyaluronidase directly into the injured area hydrolyzes hyaluronic acid, thus decreasing the ground substance viscosity. Minimal efficacy and the potential for hypersensitivity make the agents of limited use. Counterirritant lotions and creams (e.g., camphor and methylsalicylate) produce warmth and comfort through an axonally mediated increase in local circulation. Side effects include pruritus, hypersensitivity, and, when used excessively, epidermal burns.

PROMOTE HEALING

All too often after efforts to control inflammation and relieve a patient's pain, the athlete returns prematurely to participation and is reinjured. In fact, Ekstrand and Gillquist[22] demonstrated that failure to properly rehabilitate an athlete after an initial injury can cause recurrent injury. Athletes and healthcare professionals often fail to appreciate that rest and anti-inflammatory medications do not heal. Clinicians can ensure a successful return to sport only when inflammation control overlaps aggressive efforts to promote healing.

Healing involves the deposition, proliferation, and maturation of collagen-creating, vascular elements and fibroblasts in injured tissue. This healing cascade is best facilitated through a combination of site-specific rehabilitative exercise and general cardiovascular conditioning, the goal of which is to restore injured tissue to normal or near-normal anatomic function. Early exercise enhances tissue oxygenation and nutrition, minimizes unnecessary atrophy, and aligns collagen fibers to meet eventual sports-induced stresses. Progress through rehabilitative programs is best accomplished under the direction of a physical therapist or a certified athletic trainer, as each individual regimen is based on the particular injury and the athlete's specific needs.

Successful rehabilitative exercise programs incorporate full-motion strengthening of the injured tissue as well as addressing the strength and stability of the entire kinetic chain. For example, the athlete with patellofemoral syndrome typically requires attention to quadriceps and hamstring strength and flexibility; however, complete rehabilitation may require assessment and rehabilitation of the athlete's core stability. Weakness in the hip adductors can cause patellar tracking problems by increasing internal rotation of the femur and dynamically increasing the athlete's Q-angle. Strength deficits in the abdominal musculature or paraspinous muscles may also predispose the athlete to lower extremity injury. Because closed-chain, functional exercises recruit muscles in more physiologic motor patterns, they are preferred in both core strengthening and in rehabilitation of the primary injured tissue.

Good general body conditioning is also important in the promotion of injury healing because it:

- Increases regional perfusion through central and peripheral aerobics
- Provides neurologic stimulus to injured tissue through neurophysiologic synergy and overflow
- Minimizes weakness of adjacent uninjured tissue (decreases or eliminates destructive domino effect)
- Minimizes negative psychological effects
- Minimizes unwanted fat and helps control weight

A good general body conditioning program incorporates strength training of uninjured tissues with appropriate forms of aerobic exercise. Exercises commonly used for such conditioning include stationary bicycling, stair climbing, upper body ergometry, and water workouts.

Occasionally, an athlete with a sports injury will not respond to the typical treatment regimen. These individuals are at high risk of developing a chronic injury, most typically a chronic tendonopathy, which classically responds very poorly to all forms of conservative treatment. However, new evidence indicates that a high percentage of these injuries will respond to intense eccentric rehabilitation. The most compelling evidence involves patients with chronic Achilles tendinosis. Alfredson et al.[24] enrolled patients awaiting surgical debridement of their Achilles tendons into a 12-week program of twice daily eccentric calf strengthening. Over 90% of these end-stage tendinopathy patients returned to tendon-loading activities and removed their names from the surgical waiting list after completing this program. While the exact healing mechanism of eccentric exercise is not known, preliminary data suggest that it may alter the neovascular proliferation observed in these chronic tendinopathies.

Rehabilitative exercise generally restores an athlete's previous level of function. In some situations, however, mechanical derangements will require early consideration of operative intervention e.g., anterior cruciate ligament disruption. These scenarios are described throughout the body of this work, with clear indications for orthopedic consultation. While most overuse injuries will respond to a well-planned rehabilitative exercise program, a few patients may fail to respond and require surgery. Patients who fail to improve with conservative therapy, however, should seek a second opinion to be sure that possible "culprits" have been identified and treated before surgery is considered. If surgery is necessary, it should seek to provide a better physiologic environment for renewed rehabilitative effort through resection of pathologic soft tissue and correction of underlying anatomic risk factors. Surgery should be considered for a patient when:

- Rehabilitative program has failed (after 3 to 6 months).
- Quality of life is unacceptable.
- Weakness, atrophy, and dysfunction persist.

Increase Fitness

Once healing and rehabilitative exercise have restored damaged tissues to normal strength, a patient's tissues require further strengthening to achieve the supernormal endurance and power required for the demands of sport. Here, fitness exercises enter the management pyramid. These exercises involve sport-specific rehabilitation and general body conditioning.

A patient can begin these fitness exercises when he or she has achieved pain-free range of motion and strength and endurance tests indicate a return to the pre-injury state. Sport-specific activities work the athlete's target tissues, providing neurophysiologic stimulus and redeveloping propioceptive skills. Core stability programs should be expanded to incorporate complex movements combining the sagital, coronal, and transverse planes of motion. Sport-specific agility, speed, and skill drills such as plyometrics, interactive eccentric/concentric muscle loading, anaerobic sprints, and interval training all coordinate interaction of the athlete's antagonistic and supporting muscles.

CONTROL ABUSE

The final step of sports injury management is to control force loads to the rehabilitated tissue. Controlling tissue overload means modifying both intrinsic and extrinsic risk factors identified in the patient's history and physical examination. Effective control of tissue overload includes:

- Improving the athlete's sport technique
- Bracing or taping the injured part
- Controlling the intensity and duration of the activity
- Appropriately modifying equipment

Improving the athlete's sport technique is critical because abnormal and improper biomechanics quickly promote reinjury. Bracing and taping control abuse during rehabilitation and when the athlete first resumes sports activity. Handoll et

al.[25] have demonstrated through a systematic review of randomized trials that ankle supports can prevent ankle sprains, in particular in athletes with a history of ankle sprain (Level of Evidence B, systematic review).[25] Counterforce bracing helps control an athlete's muscle balance. Groppel and Nirschl[26] have shown that elbow counterforce braces decrease elbow angular acceleration and electromyogram muscle activity and thus are of value in treating tennis elbow. We have successfully used counterforce bracing to treat patients with tennis elbow, plantar fasciitis, Achilles tendinosis, and patellar tendinopathy.

Training errors — excessive frequency, intensity, and duration — are the principal risk factors for overuse injuries. The clinician must emphasize that more is not always better and explain that overtraining precipitates injury and causes fatigue and decreased performance. Athletes should be encouraged to follow basic training principles of progression and periodization, which imply gradual increases in workload and training cycles that emphasize programmed rest.

Modifying equipment requires paying attention to shoes, sport-specific equipment, and playing or training surfaces. Subtle abnormalities in an athlete's foot biomechanics can contribute to numerous lower-extremity overuse injuries. Physicians should attempt to correct these abnormalities through rehabilitation, proper footwear, and, if necessary, custom orthotics. Wide-bodied rackets or rackets with improper grip size often predispose tennis players to overuse injuries of the upper extremity. Lower-extremity injuries, such as plantar fasciitis and stress fractures, often result from poor or hard playing surfaces. An ideal playing surface provides adequate traction, cushion, and evenness so athletes can avoid excessive forces from repetitive pounding, twisting, and turning.

BACK IN ACTION

Traditionally, athletes have been allowed to return to activity when they demonstrate full range of motion and when the injured extremity shows 80 to 90% of the strength of the uninjured extremity (objectively measured with functional testing). These two criteria, however, are only minimums. Before an athlete returns to activity, the physician, coach, and trainer should consider asking two other questions:

- Does the athlete demonstrate sports-specific function?
- Is the athlete psychologically ready?

When all involved are satisfied with the answers to these questions, the athlete can safely return to full activity.

SUMMARY

Management of all sports and recreational trauma requires a comprehensive plan based on a solid understanding of the pathophysiology of injuries and applying the mnemonic PRICEMM. Appropriate application as soon as possible following injury will significantly mitigate the inflammatory process, particularly if applied during the "golden period." Rehabilitation and exercise therapy must be initiated after the acute phase; return to play may be allowed when the strength and range of motion of the injured anatomy are the same as the normal side. Preventive measures (taping, strapping, orthotics, etc.) should be employed during rehabilitation and return to play. Surgery may be required in 1 to 2% of cases, for certain acute or debilitating chronic injuries. As captain of the sports medicine team, the physician must design a progressive, integrated program for a safe return to play.

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28 PHYSICAL THERAPY IN TREATMENT OF SPORTS INJURIES

Robert M. Barney Poole

INTRODUCTION

Physical therapy is an essential element in the treatment of sports injuries and in the safe return of an athlete to his or her sport. A sports physical therapist is specially trained and may be board certified by the American Board of Physical Therapy Specialties of the American Physical Therapy Association. The sports physical therapist has special knowledge of sports skills, training methods, injury treatment and prevention, exercise physiology, sports psychology, and sports medicine. The physical therapist must work closely with the athlete and other members of the sports medicine team (see Chapter 2). The physical therapist performs an initial examination of the athlete and determines a diagnosis. A baseline is established from which progress can be measured. Goals are set with the consensus of the athlete, coach, parents, and physician. The physical therapist then designs and implements a rehabilitation program specifically for that athlete designed to meet those goals. Constant re-evaluation and modification of the program are necessary to ensure safety and continued progress.

REHABILITATION PRINCIPLES

The general goal of any treatment and rehabilitation plan is the safe return of the athlete to their prior level of competition, as soon as possible. Figure 28.1 depicts an idealized healing curve and rehabilitative program using the various therapeutic modalities and exercises. Functional progression is used to move the athlete through the recovery process, maximizing function and minimizing reinjury. Phase I (injury period) consists of reduction of the inflammatory process; Phase II (reparative period) promotes optimal healing through therapeutic exercises that are functionally progressive, specific, and balanced. Functional exercises must be constantly monitored and modified during this period to prevent reinjury and/or an increase in inflammation. Phase III (recovery period) is a period of strength and conditioning during which the athlete prepares for entry into regular training and competition. A monitored, functional progression of sport-specific and general fitness activities advance the athlete through levels of training. The result is a gradual return to individual or team competition.

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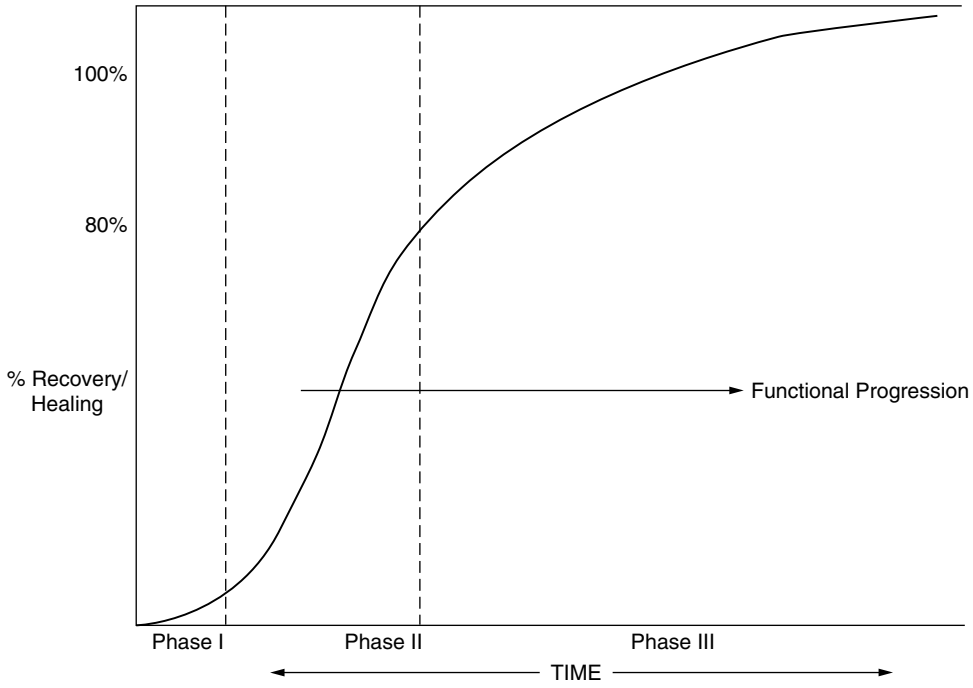

Figure 28.1 Under ideal circumstances for healing and rehabilitation, most musculoskeletal injuries progressively improve from the acute phase (I), through the intermediate, reparative phase (II), to the consolidation phase (III). The duration of each phase depends on the severity and type of the trauma, the rehabilitation program, and risk factors for further injury.

REFERRAL TO PHYSICAL THERAPY

Basic first-aid of sports injuries occurs on the field or in the training room. A physical therapist or athletic trainer examines the athlete and a diagnosis is determined. The injured athlete may be referred to the team physician for further examination and confirmation of the diagnosis by examination and imagining techniques. It is important that the team physician be advised of injuries as soon as possible after they happen. After physician examination, the athlete may be referred to physical therapy for follow-up care. In some states, a written referral to physical therapy is required, but in most states this is not necessary. A written referral, when required, should be signed by the physician, should include the athlete's name and diagnosis, and may include general recommendations for treatment. For examples of how and when to refer to physical therapy, see Table 28.1 and Treatment Presentations 1 through 4. A rehabilitation plan of care is developed with input from all members of the sports medicine team. The physical therapist or an athletic trainer may become the coordinator of the athlete's rehabilitation program (Table 28.2). Acute injuries may require modality use to decrease swelling and promote healing. An exercise program is important to regain range of motion and strength. Constant re-evaluation of the athlete and modification of the rehabilitation plan is needed to produce the desired result: the safe return of the athlete to the field. Communication among the physical therapist, physician, athlete, athletic trainer, and coach optimizes compliance and the program's success.[1]

MODALITY USE IN SPORTS INJURY MANAGEMENT

Hippocrates (400 B.C.) was one of the first proponents of cold as a therapeutic modality to reduce swelling and pain caused by acute trauma.[2] Ice packs, reusable cold gel packs, ice massage, and ice immersion are techniques frequently used to treat acute sports injuries. Cold application (>15°C) reduces the acute inflammatory response and permits earlier initiation of therapeutic exercise. Temperatures below 15°C should be avoided because the effect can be harmful. Duration of treatment using various

TABLE 28.1
Common Sports Injuries Referred to Physical Therapy

	Treatment	
Problem	Acute (0–72 hr post-injury)	Chronic (72+ hr post-injury)
Muscle strains	Ice, gentle range of motion (ROM)	Heat, active exercise, ultrasound
Ankle sprain	Ice, support,[a] gentle ROM	Contrast treatments,[b] support, active exercise
Knee injuries	Ice, support,[a] gentle ROM	Contrast treatments, muscle re-education, active exercise, support
Hip pointer	Ice, support	Special padding, ultrasound (phonophoresis)
Low back pain	Ice, modalities	Heat modalities, active exercise
Cervical pain	Ice, modalities, gentle ROM	Heat modalities, active exercise
Shoulder injuries	Ice, modalities, gentle ROM, support[a]	Heat modalities, active ROM and exercise
Elbow injuries	Ice, modalities, gentle ROM, support[a]	Heat modalities, iontophoresis, active strengthening
Wrist and finger injuries	Ice, ROM, support	Heat modalities, active strengthening

[a] Support: elastic wrap, athletic taping, splinting, casting.

[b] Contrast treatments: heat followed by cold.

TABLE 28.2
General Rehabilitative Program Guidelines

1. Supervised sessions once or twice daily
2. Specific rehabilitative prescription:
   - Early mobilization
   - Modalities
   - Exercise
3. Sports-specific skill + proprioceptive sense recovery
4. Use of substitute activity to maintain total fitness, flexibility, and strength
5. Correction of biomechanical, equipment, and training errors
6. Return to play

forms of therapeutic cold in contact with the skin should not exceed 20 to 30 minutes and may be repeated every 60 to 90 minutes until the inflammatory response resolves. An athlete should be discouraged from returning to participation after an application of ice because the protective pain mechanism may be compromised, precipitating further injury.

Therapeutic heat is used to relieve pain, increase circulation, facilitate tissue healing, and prepare stiff joints and tight muscles for exercise. Heat may be applied using moist heat packs or a warm whirlpool. Application time is usually 20 minutes, allowing at least 60 to 90 minutes between applications. With athletic injuries, heat application can be started after the initial acute phase of injury, or approximately 72 hours after injury. When applying heat, swelling should be monitored. An appropriate form of treatment during the intermediate phase of rehabilitation might begin with moist heat, progress through gentle exercise, and end with an application of cold and compression with a protective wrap to support the injured area. Ultrasound is a form of deep heat that uses high-frequency sound energy to produce changes in tissue temperature as deep as 5 cm. The benefits of therapeutic ultrasound include reduction of muscle spasm and pain relief.[3,4]

Phonophoresis is a technique that uses ultrasound to deliver medication through the skin into the tissues. An anti-inflammatory agent is driven into the inflamed area using high-frequency sound waves. This is commonly used in the treatment of conditions such as bursitis and tendinitis. Most commonly, a 10% hydrocortisone cream is rubbed into the skin, then a layer of ultrasound coupling gel is applied and ultrasound delivered.[4–6]

A variety of pulsed waveforms are available to strengthen and re-educate muscle, reduce pain, and diminish muscle spasm. High-volt pulsed current (HVPC) can be used to recruit muscles that are inhibited by post-traumatic or post-surgical pain and/or edema. The electrical stimulation provides tactile and proprioceptive input while creating active muscle contraction. HVPC promotes motor recruitment and helps diminish muscle atrophy.[7,8]

Transcutaneous electrical nerve stimulation (TENS) is a form of electrical stimulation used to diminish pain. A biphasic waveform is delivered by a small hand-held unit powered by a 9-volt battery. TENS may be helpful in allowing gentle range of motion to begin at an earlier stage with less pain.[9,10] Melzack and Wall[11] suggested that TENS stimulates sensory nerves, thereby closing the gate to pain sensation at the level of the spinal cord and preventing that pain from reaching the brain. Other theories suggest that TENS causes a release of endogenous opiates from within the central nervous system.[11,12]

Iontophoresis uses direct current to propel ions of medication into tissue.[13,14] A number of drugs may be used with iontophoresis for specific situations. Dexamethasone sodium phosphate (4 mg/mL) is most often used for treatment of inflamed tissue. A negative electrode containing the medication is placed directly over the area to be treated, and the positive electrode is placed approximately 2 inches away. Treatment time, with the current adjusted to a comfortable setting (up to 4.0 mA), is 16 to 20 minutes.[14]

Therapeutic modalities vary from the simple ice pack to complex and expensive electrical devices. They can be useful in reducing pain, swelling, inflammation, and other effects of sports injuries. They are most effective when applied and monitored by appropriate practitioners and combined with therapeutic exercise into a comprehensive rehabilitation program.

THERAPEUTIC EXERCISE

(See Appendix 7.) Restoring flexibility is an essential part of any comprehensive rehabilitation program. Without adequate flexibility and joint range of motion, normal muscle relationships cannot be re-established following injury. The first phase of the rehabilitation process is to restore joint range of motion without increasing the symptoms of the injury or causing further damage. Gentle active, active-assisted, or passive range of motion (ROM) exercise begins as soon as swelling is under control and comfort allows. During the early stages of the healing process, frequent ROM in a pain-free range minimizes inflammation. Careful attention to the stages of the healing process by continual re-examination is needed in order to appropriately progress the athlete's rehabilitation program.[14] As ROM increases, gentle stretching exercises begin.. The athlete is instructed in proper stretching techniques in order to gain muscle flexibility and maintain joint ROM while building strength.[15,16]

After ROM is restored, the strengthening phase can begin. The three types of strengthening exercises are isometric, isotonic, and isokinetic. Isometric exercises produce muscle force without joint movement. Resistance is adjusted by increasing or decreasing muscle contraction. Isometrics are useful during the acute phase in preventing loss of strength without causing further injury. Also during the acute phase, isometrics can be used to reduce swelling around the joint through the "pumping" action of the muscle. Isometrics promote healing by increasing circulation.

Isotonic exercises strengthen muscles through contraction of muscle fibers against constant resistance. Resistance may be supplied by using strap weights elastic bands or rubber tubing, free weights, or weight machines. In order to strengthen the muscle isotonically, the load (i.e., resistance) must be increased. When dealing with an injury, increasing the load too soon can cause further injury, thus prolonging the rehabilitation process. A high-repetition, low-weight program is recommended when initiating an isotonic strengthening program. Progressive resistance exercise (PRE) is one form of isotonic exercise.[17] Progressive resistance exercise is a system of dynamic resistance training in which a constant external load is applied to the contracting muscle and incrementally increased. Many different programs exist but most incorporate 2 to 3 sets of 6 to 12 repetitions, with a maximum of 6 to 12 repetitions. Techniques in which resistance in the early sets is less than in the later sets provide a warm-up for muscles. Techniques in which resistance in later sets is lower than in early sets adapt to muscle fatigue. All techniques call for increasing resistance over time.[18]

In any type of resistance training, especially post-injury training, care is taken to prevent further injury during the healing process. Proper technique and exercise through complete ROM are essential in order to prevent injury to muscles, the joint capsule, or connective tissue. Exercise that simulates actual athletic activity should be used when possible. Proper warm-up should be emphasized prior to resistive training, and flexibility exercises should be included to maintain functional ROM throughout exercise.

Isokinetic exercise involves muscle contractions through a range of motion at a fixed speed. The resistance varies and accommodates according to the ability of the muscles to contract. Isokinetic devices load the musculoskeletal unit maximally through the range while taking into account pain, fatigue, and other factors that affect

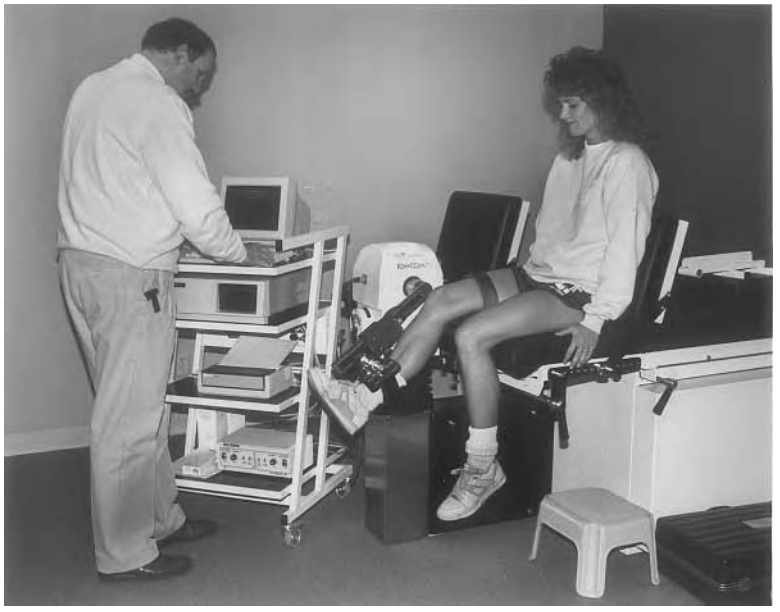

Figure 28.2 Isokinetic testing using the KIN-COM®(Chattanooga Corp., Chattanooga, TN).

force of muscle contraction. For this reason, it is relatively safe to use isokinetics in post-injury rehabilitation programs. The controlled speed allows muscle training at closer to functional limb speeds (Figure 28.2). Equipment for isokinetic exercise and testing is expensive and requires a good deal of space in the clinic.

In the final phase of rehabilitation, the athlete progresses to sport-specific activities; these are specifically related to activities the athlete will need to perform on the field during practice or competition.[18] Sport-specific activities should include balance or proprioception, coordination, and agility training. Proprioception is the basis for transferring flexibility and strength into agility and skill in competition and is an integral part of preparing the athlete for return to competition. Use of all or parts of actual agility drills used in practice for the athlete's sport may be started at half speed, emphasizing performance skill and confidence, with no increase in symptoms. Also useful in promoting proprioception are activities such as the balance board and one-leg standing for the lower extremity and the use of weighted bats, balls, or other equipment for the upper extremity.

Endurance and conditioning activities for general fitness should be a part of the rehabilitation process and should begin as early as possible. Maintaining the general fitness of the athlete as a whole, while protecting and strengthening the injured area, is essential to a good result and to sustaining motivation. The athlete can continue to work out in the weight room, incorporating aspects of his or her weight routine that do not involve the injured area. Some athletes must also incorporate short bursts of high intensity into their sports. Football players, for instance, should train for short, intense bursts of exercise such as those inherent in their sport; therefore, use of an exercise bike to maintain general fitness should also include short sprint work to help maintain anaerobic capacity.

All activities should begin with proper warm-up and stretching activities. After exercise, a cool-down session should also include stretching, after which ice may be applied as needed to minimize swelling and pain.

RETURN TO PARTICIPATION

The criteria for the return of an athlete to the playing field include return to normal levels of flexibility, strength, muscle bulk, endurance, agility, skill in sport-specific activities, and psychological factors. Flexibility is determined by assessing muscle length and goniometric joint measurement and is compared with the uninvolved side and with preparticipation physical exam data, if available. Flexibility and strength of agonists and antagonists must be balanced for optimal function. Strength is best evaluated by manual muscle testing. When possible, strength is compared to the uninvolved side. The rehabilitation

process is not finished until muscle bulk returns. Although strength may be adequate bilaterally with testing, a difference in muscle bulk may exist. This can be compared bilaterally by a simple circumferential tape measurement above and below the joint line. The athlete is not able to protect adequately against further injury due to decreased endurance and strength until adequate muscle bulk has returned. Coaches and athletic trainers compare the athlete's skill, agility, endurance, and coordination with pre-injury levels. The physical therapist provides objective data from both strength and range-of-motion evaluations. The information is used by the sports medicine team in deciding when to return the athlete to competition. The athlete is a source of some of the most valuable input into the decision to return to participation. Each athlete is different, having his or her own psychological make-up and reaction to injury and rehabilitation; consequently, the return to play is also different for each.[17,18]

SUMMARY

The sports medicine team consists of the physician, physical therapist, and athletic trainer. It is the job of these specialists to evaluate, diagnose, and treat injured athletes. The team goal is to return each athlete swiftly and safely to the playing field. Through the use of therapeutic modalities and constant re-evaluation and modification of individualized exercise programs, the athlete is taught how to regain the skills lost to injury and how to prevent further injury.

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Treatment Presentation 1

Chronic Lateral Epicondylitis

A 25-year-old recreational tennis player is referred to physical therapy with chronic lateral epicondylitis. The patient is treated daily for 10 days with frequent reassessment to determine progress.

Procedure

A. Localize the area of the lesion by palpation and thoroughly clean the skin.
B. Preheat for 10 minutes with a moist heat pack.
C. Follow heat treatment with transverse friction massage to the lateral epicondyle area for 5 minutes.
D. Instruct the athlete in proper technique for eccentric exercise and stretching (refer to Appendix 7).
E. Ice massage with ice pop for 15 minutes following exercise.

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Treatment Presentation 2

Hamstring Pull

The star defensive back suddenly cuts to his left, decelerating quickly. He feels a tearing followed by a sharp pain in his left upper hamstring. Grabbing his thigh, he limps off the field to the team doctor and therapist standing on the sideline. He is examined and found to have a hamstring pull. Treatment begins immediately.

Acute Sideline Management, Days 1 to 3

A. Ice packs, 20 to 30 minutes, several times a day
B. 1/2-inch foam or felt over injury with compression bandage
C. Weight bearing as tolerated with crutches
D. Electric stimulation as indicated for pain management

Subacute Phase, Days 3 to 5
(Active Bleeding Stopped)

A. Progressive walking to tolerance; discontinue crutches
B. Contrast treatments (15 minutes heat/15 minutes cold) and electric stimulation for pain management
C. Exercise program:
   - Active hamstring stretch, supine
   - Gravity assisted strengthening
   - Biking
D. Light massage and compression dressing

One Week Post-Injury

A. Progressive walk to run as tolerated
B. Moist heat
C. Ultrasound to increase circulation and promote tissue healing
D. Exercise program (refer to Appendix 7)
   - Hamstring curls
   - Hamstring stretch (passive)
   - Step-ups
E. Massage and compression dressing
F. Progress to sport-specific activities as tolerated

Two to Three Weeks Post-Injury

Criteria for return to activity:

- Range of motion within normal limits for involved joints
- Hamstring stretch without pain
- Run/cut, accelerate to normal
- Progressive strengthening and flexibility without pain
- Compression support

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Treatment Presentation 3

Conservative Treatment for Medial Meniscus Tear

A wrestler in the 125-pound weight class slips and twists his left knee. He feels a pop but continues to wrestle. The next morning his knee is swollen and painful. After examination he is diagnosed as having a medial meniscus tear and begins physical therapy.

Day 1 and 2

A. Compression wrap
B. Crutches with partial weight-bearing to tolerance
C. Cryotherapy — ice packs or cold compression unit for 20 minutes several times a day with elevation
D. Exercises:
   - Quad sets
   - Hamstring stretches
   - Straight leg raises
   - Active/active-assisted range of motion

Days 3 to 5

A. Compression wrap
B. Progress toward full weight-bearing; discontinue crutches
C. Cryotherapy — ice packs or cold compression unit for 20 minutes several times a day
D. Electric stimulation for quad muscle re-education
E. Continue the above exercises and add the following (refer to Appendix 7):
   - Hip flexion
   - Hamstring curls
   - Hip abduction
   - Biking, as range of motion permits

Days 6 to 10

A. Light compression dressing as needed
B. Walking program up to 2 miles
C. Cryotherapy as necessary after exercise, 20 minutes
D. Continue exercise program and add the following:
   - Step-ups
   - One quarter squats
   - Swimming

Days 10 to 14

A. Progressive weight training with machines and free weights
B. Progressive running; sport-specific activity (e.g., figure eights, cutting, carioca); return to sport
C. Cryotherapy when necessary after activity, 20 minutes

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Treatment Presentation 4

Acute Ankle Sprain

A 17-year-old baseball player is diagnosed as having a grade II lateral ankle sprain with moderate swelling and some lateral instability and begins physical therapy treatment.

Acute, Days 1 to 3

A. Tubular compression stocking applied to the extremity from the toes to just above the knee.
B. Cold and compression applied 20 minutes with elevation of the limb during treatment; place the ankle in an open basketweave. Reapply tubular compression stocking to the extremity from the toes to just below the knee. The athlete may be weight bearing with crutches as tolerated. Continue ice

packs and elevation at home for 20 minutes every other hour.

C. Exercise program:
   - Ankle range of motion
   - Isometrics for anterior tibialis, posterior tibialis, and peroneals, 20 repetitions frequently during the day

Subacute Phase, Days 4 to 6

A. Warm whirlpool (97°F) 15 minutes with active range of motion
B. Continue exercise program above and add the following:
   - Heel cord stretching
   - Heel raises
   - Toe raises
   - Strengthening (progressive resistive exercises) for anterior tibialis, posterior tibialis, and peroneals (refer to Appendix 7)
   - Proprioceptive exercises using balance board
C. Cold and compression and tape after exercise (as above)
D. Progress to gait without crutches, weight-bearing as tolerated
   - Progress to straightline jogging
   - Progress to specific sports activities (e.g., agility drills such as figure eights, cutting, carioca)
E. Ice after exercise for 20 minutes, as needed for swelling control

29 SPORTS MEDICINE RADIOLOGY FOR THE PRIMARY CARE PROVIDER

Donald J. Flemming and Stephanie A. Bernard

INTRODUCTION

A bewildering array of imaging modalities can be employed in the evaluation of sports injuries in modern practice. The challenge for the primary care provider is to order the appropriate examination that will most accurately evaluate an injury or complaint in the face of competing pressures of high patient expectation and insurance companies trying to reduce costs. Ideally, before ordering an examination, the provider should have an idea of the question that needs to be answered and have an idea of how the information that is obtained will alter the management of the patient.[1,2] An accurate history and physical exam delineate the clinical question, and this information, combined with medical or surgical management, dictates what imaging should be utilized. The most commonly employed imaging technology (Table 29.1) in the sports medicine setting includes conventional radiography, fluoroscopy, arthrography, computed tomography (CT), magnetic resonance imaging (MRI), ultrasound (US), and nuclear medicine.

CONVENTIONAL RADIOGRAPHY/FLUOROSCOPY

High-quality conventional radiography, whether film or digital, continues to be the single most

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TABLE 29.1
Diagnostic Utility of Imaging Modalities in Sports Medicine

Site/Condition	Plain Film	Computed Tomography	Magnetic Resonance	Ultrasound	Bone Scan
Neuroaxial					
Acute head injury	–	+++	++	–	–
Post concussion syndrome	–	+	+++	–	–
Facial fractures	+	+++	–	–	–
Acute spine fracture	+++	+++	++	–	–
Chronic back pain	–	++	+++	–	±
Extremity					
Knee, internal derangement	–	–	+++	–	–
Shoulder, rotator cuff	±	+ (arthrogram)	+++	++	–
Shoulder, labrum	–	++ (arthrogram)	+++ (arthrogram)	–	–
Elbow, tendon/ligament	–	–	+++	++	–
Elbow, ostechondral	++	++ (arthrogram)	+++	–	–
Wrist, fracture	++	++	+++	–	+
Wrist, ligament	–	–	+++ (arthrogram)	–	–
Hip, fracture	++	±	+++	–	+
Hip, labrum	–	–	+++ (arthrogram)	–	–
Foot/ankle, tendon	–	–	+++	+++	–
Foot/ankle, osseous/chondral	++	±	+++	–	+

Note: +++, most useful exam; ++, very useful exam; +, useful exam; ±, may be useful depending on the situation; —, not recommended.

important examination in the evaluation of many sports injuries. Plain x-ray is inexpensive and offers high specificity for osseous pathology, particularly in the extremities. Attention to radiographic technique, positioning, and film development are required to optimize evaluation. Real-time fluoroscopic examination of an extremity may aid the diagnosis and evaluation of injuries such as wrist instabilities. Fluoroscopy is also utilized for image-guided injections.

ARTHROGRAPHY

Arthrography, whether conventional or in conjunction with MRI or CT, improves the evaluation of soft tissue structures. Conventional direct arthrography requires the use of fluoroscopy to direct the injection and carries minimal risk of infection and allergic reaction to the injected materials. Patients tend to accept the minimal pain and anxiety associated with the injection, particularly in the setting of motivated athletes. Indirect arthrography is a newer technique that may be used successfully in some clinical settings. Indirect arthrography involves MRI of the affected joint following the intravenous injection of contrast. Contrast diffuses into the joint and has been shown to increase the accuracy of shoulder and wrist pathology in comparison to non-contrast examinations.

COMPUTED TOMOGRAPHY

Computed tomography continues to evolve as a technique and is an outstanding modality for assessing common sports injuries, particularly in areas of complex anatomy. CT has become the standard of care for evaluation of abdominal blunt trauma, spinal and acetabular fractures, and initial evaluation of closed head trauma. CT is moderately expensive but utilizes ionizing radiation. Data are obtained perpendicular to the axis of the gantry so generally images are obtained in

the axial plane. Creative patient positioning and angling of the gantry can result in oblique images being obtained, but reconstructions are often required to assess complex intraarticular fractures of the knee, elbow, and distal radius.

MAGNETIC RESONANCE IMAGING

The development of magnetic resonance imaging has revolutionized the evaluation of musculoskeletal disease, neurological disorders and sport injuries. The multiplanar capabilities and inherent contrast resolution of MRI offer the opportunity to evaluate muscles, tendons, ligaments, and cartilage, as well as osseous structures about a joint and the neurological axis. MRI relies on contrast between different tissues for the detection of pathology. T_1-weighted images are generated using short time to repetition (TR) and short time to excitation (TE). Fat is bright in signal on T_1-weighted images, and anatomy is well demonstrated on this sequence. T_2-weighted images show fluid to advantage. Pathology such as fracture, infection, and tumor frequently are associated with edema; therefore, pathology is frequently bright in signal on T_2-weighted images. Fat-saturation sequences, whether T_2 or inversion recovery, increase the conspicuity of fluid, so these sequences accentuate the detection of pathology associated with edema or fluid. Fat saturation can also be used with T_1-weighted sequences in high field magnets to accentuate conspicuity of enhancement following the administration of contrast.

Each sequence in each plane of imaging requires a finite amount of imaging time, usually varying from 2 to 5 minutes depending on the sequence. Most routine extremity examinations take approximately 30 minutes to complete but up to 1.5 hours of imaging time may be required in special circumstances. The radiologist customizes the examination based on the history provided by the referring clinician to make sure that the clinical question can be answered with the least amount of imaging time. Some clinical questions, such as possible hip or scaphoid fracture, can be answered in as little 5 to 10 minutes. These brief examinations will limit the amount of information that can be interpreted but allow a clinical question to be answered at a smaller cost.

The accurate evaluation of an injury by MRI starts with an accurate physical exam and history by the referring clinician. This critical information allows the clinician to direct the patient to the appropriate imaging center and permits the radiologist to tailor and optimize the examination. One common question that must be considered is whether to refer the patient to an open (low- or midfield) magnet or closed (high-field) magnet. With all other parameters being equal, high-field-strength magnets (>1.0 Tesla) produce higher quality images but only limited data suggest that this results in improved diagnostic yield. In general, a small structure such as the wrist is better imaged at higher field strength. Patients who are large or claustrophobic are generally referred to open magnets.

Superior clinical MRI results are not simply dependent on field strength. The generation of high-quality examinations is dependent on patient positioning/comfort, coil design, software/hardware parameters, and sequence optimization in addition to field strength. The accurate interpretation of an examination is highly dependent on the expertise of the radiologist. The literature has shown that radiologists who have specific interest and/or training in musculoskeletal radiology are more likely to produce accurate interpretations (Level of Evidence B, systematic review).[3] Optimum results are achieved when free interchange of information occurs between referring clinicians and interpreting radiologists both before and after the examination is performed. Feedback, whether negative or positive, is essential to improving performance for both the radiologist and the sports medicine physician.

Magnetic resonance imaging cannot be or should not be utilized in specific patients. Patients with certain implantable devices such as pacemakers, cochlear implants, stainless steel intracranial aneurysm clips, and intraocular metal and drug pumps cannot be safely imaged in MRI units because of the effect of the magnetic field. Patients with metallic implants in the region of clinical question should not be imaged in most cases because artifact will distort and obscure the anatomy. Titanium implants decrease metallic artifact and therefore minimize image distortion.

ULTRASOUND

Ultrasound is a relatively inexpensive technique that is widely utilized in Europe and Australia in the evaluation of musculoskeletal disease; its use in the United States is expanding. The development of portable US units has lead to the on-field use of this technique for the assessment of muscle injury, for example. Ultrasound is an outstanding technique that permits rapid assessment of a specific structure such as the Achilles tendon.[4,5] Global assessment of a joint that can be achieved by

MRI is not typically possible, however. Ultrasound is extremely operator dependent and outcome is highly reliant on the experience of the ultrasonographer.

NUCLEAR MEDICINE

The use of nuclear medicine in sports medicine is limited. Bone scintigraphy is extremely sensitive for the detection of metabolically active bone and therefore detects stress fractures earlier than conventional radiography; however, bone scans are relatively expensive and utilize ionizing radiation. Bone scans may demonstrate uptake in a pattern that establishes a specific diagnosis but, frequently, positive examinations do not offer sufficient specificity to prevent performance of another examination. If a bone scan leads to the use of a MRI or CT scan anyway, it is less expensive and more time efficient to start with these modalities in most sports injury settings.

NEUROAXIAL INJURY

Head Injuries

Over the past 25 years, the evaluation of injury patterns and frequencies in sports has led to changes in rules and protective equipment that have significantly decreased the incidence of many serious injuries. Nonetheless, head injuries, and specifically concussion injuries, remain a relatively frequent occurrence in contact sports. Somewhat unique to injuries of the neuroaxis is that the initial clinical examination may not reflect the severity of the underlying injury. In response, standardized clinical assessment and management guidelines have been put forward by the American Academy of Neurology,[6,7] as have recommendations for criteria in determining return to competition.[8,9] In general, given the complexity of accurate clinical assessment and the potential implications of undiagnosed injuries, a low threshold for obtaining head imaging should be maintained. An additional management consideration is that patients with head injuries in trauma series have demonstrated a 6 to 9% incidence of concomitant cervical spine injury. All patients should have appropriate spine immobilization until injury to the spinal column is excluded.

Conventional radiographs of the skull have very low utility in the setting of head trauma and most often serve only to delay further definitive imaging.[10] CT, because of its rapid acquisition time, ability to obtain diagnostic-quality images even in the uncooperative/semiconscious patient, and its simultaneous evaluation of brain, soft tissue, and bone structures, remains the primary imaging modality for evaluation of all acute cranial injuries (Level of Evidence C, consensus/expert opinion).[11] A follow-up CT examination at 24 hours post-injury will often demonstrate parenchymal hemorrhages that were not acutely evident. MRI is pursued as an adjunct to CT in the evaluation of patients in which neurological symptoms persist despite a normal initial and follow-up CT or in those for whom the severity of neurological symptoms is not explained by visualized CT abnormalities. While MRI identifies a greater number and degree of brain parenchymal injuries in the acute setting in comparison with CT, this increased sensitivity has not been shown to alter the management of these patients but primarily provides for better long-term treatment planning and outcome counseling.[12] Magnetic resonance angiography does, however, provide a noninvasive alternative to conventional angiography for vascular evaluation when specific concerns exist.

Facial Injuries

The majority of facial injuries in athletic competition comprise lacerations or soft tissue contusions. The decision to image facial trauma should be directed by physical examination and concerns for underlying skeletal injury or vital soft tissue structure damage. It should be remembered that approximately 2% of trauma patients requiring imaging for facial trauma have a coexisting cervical spine injury, and spine immobilization should be implemented as appropriate.[13,14] Specialized conventional radiographs may be helpful in specific settings, such as panorex radiographs of the mandible when the concern is isolated to this region. For the most part, however, the practice of obtaining conventional radiographs has been supplanted by CT imaging of the facial bones. Given the complexity of the facial bones, CT provides a more accurate identification of both the presence and extent of fractures as well as soft tissue injuries, thus providing critical information necessary for surgical planning. In conjunction with an ear, nose, and throat (ENT) or ophthalmologic specialist, occasional directed imaging of orbital or soft tissue structures by MRI or ultrasound may be performed. Nasal fractures can be documented on conventional radiography, but this information usually does not change the clinical management of the patient; therefore, the practice of routinely imaging nasal fractures is not cost effective.

Spine

Considerable variability exists between institutional practices in imaging evaluation of spinal injuries. Much of the variation is based on experience levels, availability of imaging modalities, and the institutional philosophy. Conventional radiographic examination remains the primary screening modality for spine injuries. The initial radiographs in the setting of suspected trauma should be obtained with spine immobilization in place and under the supervision of a physician. A minimum of two views of the spine are obtained. Additional views directed toward identified regions of concern may be added. While an algorithm approach simplifies initial imaging decisions, these algorithms should be used as a template by which imaging for the individual patient is developed. In general terms, conventional radiography provides a rapid, inexpensive, and effective initial evaluation for bone abnormalities. CT serves as a complimentary imaging tool to verify skeletal injuries suspected by radiography and to more fully demonstrate the complexity of injuries and their impact on the spinal canal in patients with known fractures. CT allows limited evaluation of the intervertebral disks and lacks sufficient contrast resolution to permit evaluation of the ligamentous support structures of the spine. Faster helical technology has permitted intravenous contrast-enhanced CT arteriography to produce diagnostic evaluation of adjacent vascular structures. The addition of MRI imaging allows for detailed evaluation of soft tissue structures, including the ligaments, disks, vasculature, and neural elements. MRI should be pursued in all patients with neurologic deficits. MRI may also be helpful in identification of a source of persistent pain following spine trauma when initial evaluation is unrevealing.

Cervical Spine

Much has been written about plain radiography of the cervical spine and the advantages of the myriad of views available.[15] While an obvious region of medicolegal concern, several studies indicate that, despite sustained trauma in the appropriate setting with a normal clinical examination, not all patients require radiographic evaluation of the cervical spine.[16,17] When conventional radiographs are obtained, the number of views may include a standard three-view series of anterior–posterior (AP), lateral, and odontoid views or a five-view series that includes the addition of oblique images based on institutional routine. Retrospective analyses have shown that 85% of all cervical spine fractures are demonstrable on the cross table lateral view alone and that 99% of fractures are visible with the addition of the AP and odontoid views.[18]

Whatever imaging protocol is decided upon, the conventional radiographs must provide adequate exposure of the spine and soft tissues from the level of the basiocciput to the cervicothoracic junction. Suspected fractures based on the plain films or non-visualized segments are further evaluated with CT imaging through the levels of concern. Sagittal and coronal reconstructed images provide improved recognition of fracture complexity and fragment displacement. MRI is performed in all patients with neurological deficits to evaluate cord or nerve root injuries. Additionally, MRI evaluates ligamentous, joint, and disk structures in patients with findings of instability or radiculopathy. MRI may be tailored to optimize the diagnostic accuracy of the examination if the radiologist is provided appropriate clinical information in advance.

Special Considerations: Craniocervical Spinal Abnormalities That Predispose to Cervical Cord Injuries

Due to a lack of evidence correlating isolated radiographic instability or the lack thereof and risk of cord injury in children with Down syndrome, radiographic evaluation is no longer a standard practice, although it is a requirement for the Special Olympics (Level of Evidence C, expert opinion).[19] Current recommendations are for radiographic evaluation of such children prior to sports participation to investigate cervical spine complaints. Certain developmental anomalies of the craniocervical spine such as vertebral segmentation and fusion abnormalities, os odontoidium and ligamentous instability, or development spinal canal stenosis in the setting of neuropraxias have been demonstrated to place the cord at an increased risk of injury and may serve as an absolute contraindication to participation in contact sports.[20,21] Evaluation by conventional radiographs with addition of flexion and extension views in the lateral projection is often sufficient. Persistent neurological symptoms and intermittent cervical symptoms without a correlative conventional radiographic abnormality should be further evaluated with MRI of the cervical spine. Occasionally, CT of the craniocervical junction is necessary to fully evaluate skeletal fusion abnormalities in this region.

Thoracic Spine

Traumatic injury of the thoracic spine is uncommon in sports not involving high velocities. When thoracic spine injuries do occur in collision sports, the majority consist of uncomplicated, compression-type fractures of the thoracic vertebra. While these traumatic injuries are infrequent, a presenting symptom of back pain in athletes, similar to that of the general population, is not uncommon. As a result, a variety of non-traumatic conditions of the spine may present initially in the adolescent or young adult athlete and pose a diagnostic dilemma. While this topic is beyond the scope of the discussion here, the sports medicine physician must have a familiarity with idiopathic conditions such as Scheuermann's disease, the variety of spondyloarthropathies, and the infrequently seen spine tumor, all of which may present to a sports medicine clinic in association with activity-related back pain.

Initial evaluation by AP and lateral plain radiographs is generally sufficient. In athletes with persistent pain but no demonstrable source by plain radiographs, a nuclear medicine bone scan performed with tomographic images can be helpful in localization of an abnormality to a specific region of the spine. In addition, nuclear medicine scan can provide reassurance of a lack of skeletal source of pain in those with normal single-photon emission computed tomography (SPECT) examinations. As described in the cervical spine section, CT is helpful in further evaluation of traumatic injuries, when present, for delineating displacement of fragments and encroachment on adjacent structures. Patients with localizable neurological abnormalities should have the suspected level of involvement imaged by MRI.

Lumbar Spine

Low back pain is not uncommon in athletes, much as it is in the general population. Unlike the general population, however, when low back pain persists in young athletes, a cause is able to be identified with imaging in approximately 80 to 90% of the cases.[22] This being said, a majority of acute low back pain immediately following injury is soft tissue or muscular in origin and will respond to conservative management. In the acute traumatic setting, plain radiographs should be performed of the spine when there are examination findings of focal tenderness that suggest fracture or if radicular symptoms are present. Overall, acute fractures of the lumbar vertebral bodies represent less than 10% of injuries to the lumbar spine in collision sports. When they do occur, the majority represent compression-type endplate fractures or fractures of the transverse processes and rarely spinous process fractures. CT is helpful in evaluating the extent of encroachment of fragments of the posterior aspect of the vertebral body on the spinal canal when a burst fracture is present.

A more common injury to the lumbar spine in athletes is spondylolysis, a fracture of the pars interarticularis found in the lower lumbar spine. This injury is thought to be multifactorial, with contributions from developmental weakness of the pars interarticularis and repetitive stress. Spondylolysis is especially associated with activities that involve hyperextension of the spine, resulting in stress injury and, over time, fatigue fracture of the pars interarticularis. This injury occurs most commonly at the L5 level, and the incidence decreases significantly at higher levels in the lumbar spine. For conventional radiographic imaging, the pars interarticularis is best visualized on oblique radiographs of the lumbar spine. The majority of pars fractures occur prior to the age of 15 years old. As a diagnostic dilemma, spondylolysis of L5 is present in approximately 5 to 7% of the general population with a considerably higher incidence in athletes that participate in sports requiring repetitive hyperextension of the spine.[22–24] Up to 25% of individuals with a radiographically demonstrable pars interarticularis fracture are asymptomatic. Bone scintigraphy may be helpful in distinguishing a quiescent pars fracture from a source of pain because this examination is exquisitely sensitive for areas of active bone remodeling.[25–27] CT may also provide added evaluation of complicating features of pars fractures that affect treatment planning and may be pursued in conjunction with an orthopedic specialist.

In adult athletes, degenerative disk and facet disease accounts for increasing proportions of low back pain with increasing age comparable to the general population. Conventional radiographs are commonly obtained for the initial evaluation of degenerative disk and facet disease but have little yield in the setting of chronic back pain. In the setting of radicular symptoms, MRI is helpful in evaluating for disk herniation and nerve root compression. In patients unable to undergo MRI, conventional myelography or CT following intrathecal contrast administration provides for similar diagnosis.

Fluoroscopically guided procedures (discography, facet and nerve root injections) may be employed in the evaluation and treatment of

chronic back pain disorders but their utility is controversial. Discography is used when a patient has multiple levels of degenerative disease to determine what disk is responsible for the patient's symptoms. Facet and nerve root injections may provide varying degrees and duration of pain relief and may aid in preoperative assessment.

Brachial Plexus

A temporary brachial plexopathy, more commonly identified as a "stinger" or "burner" by athletes, occurs when brachial plexus nerves are compressed or stretched; the condition is most often associated with contact sports. The symptoms are variable in severity but usually resolve completely within seconds to minutes and do not require imaging. Radiographic evaluation should be undertaken, however, in any athlete with repetitive occurrences despite appropriate protective gear or in whom neurological symptoms persist. Initial evaluation is by conventional radiography of the cervical spine and shoulder aimed at the identification of anatomic variants, fractures, or degenerative changes that may be a source of nerve compression. CT, while supplemental to evaluation of identified skeletal abnormalities, does not offer the degree of soft tissue contrast resolution for evaluation of the brachial plexus nerves. In patients with persistent neurological deficits or plexopathy, MRI is the imaging modality of choice for the brachial plexus, allowing direct evaluation of the integrity of the nerves. In patients unable to undergo MRI, CT following intrathecal injection of myelographic-grade contrast can be used to evaluate central nerve root avulsions and pseudomyelomeningocele formation when suspected. Imaging may be the diagnostic modality of choice in acute injuries because electromyography and nerve conduction studies may not reliably detect pathology for up to 2 to 3 weeks following a nerve injury.

EXTREMITY INJURY

Knee

The knee is one of the most commonly injured joints in sports injuries, and the clinical question dictates what imaging modality is utilized. Conventional radiography offers the opportunity to characterize fractures and arthritis but the evaluation of ligamentous or cartilage injury is limited to secondary findings. CT examination is limited to evaluation of intraarticular fractures in most cases. Reasonable ultrasound evaluation is restricted to tendon, muscle, and collateral ligament injury. MRI of the knee is the single best examination for global assessment of knee injury and disease. Cruciate ligament tear, meniscal pathology, collateral ligament tear, and tendon disease are all well characterized in most cases.[28,29] Both midfield and high-field magnets offer similar accuracy for evaluation of most common knee pathology.[30] Subtle hyaline cartilage defects, however, may be better imaged on high-field magnets.[31] Mild forms of chondromalacia may not be detectable regardless of field strength on routine imaging. MR arthrography is not used routinely but is sometimes performed when a question of meniscal tear arises in a patient with a history of prior meniscal surgery.[32]

Shoulder

Shoulder pain is common in athletes regardless of whether the athlete has a history of acute or chronic injury. The physical examination can be nonspecific,[33] and imaging, particularly MRI, is commonly used to aid in the delineation of pathology and to help determine the management of these often complex and confusing patients. Conventional techniques such as radiography and arthrography are commonly used in the evaluation of shoulder disease. Conventional radiography is the examination of choice for the initial evaluation of shoulder trauma and pain. Conditions such as fracture, dislocation, and arthropathy are quickly and inexpensively evaluated. The radiographic evaluation of the shoulder should be tailored to the clinical question. An evaluation of impingement frequently includes a suprapinatus outlet view, whereas trauma patients frequently are evaluated with an axillary view. Soft tissues, such as the rotator cuff and labrum, are not adequately evaluated by conventional radiography. Conventional arthography will reliably diagnose the presence of full-thickness rotator cuff tear but will not characterize the size of a defect, the status of the rotator cuff musculature, the status of the labrum or glenoid cartilage, or the presence of partial rotator cuff tears in most cases.

The use of CT in the assessment of shoulder pathology has decreased since the development of MRI. CT, however, is the examination of choice for characterization of a complex fracture of the humerus or glenoid. CT arthography (CTA) remains an outstanding method of diagnosing labral and rotator cuff pathology, particularly when multidetector CT is used with multiplanar reconstruction. CTA may be useful in claustrophobic patients and in patients who are too large to fit into a MRI unit. CTA may be the examination

of choice in a postoperative setting, such as evaluation of the labrum where metallic anchors have been employed that produce artifact on MRI that obscures the glenoid rim.[34]

Magnetic resonance imaging is the examination of choice in the evaluation of rotator cuff pathology[35] and shoulder instability[34] (Level of Evidence C, consensus/expert opinion). Non-contrast MRI, whether performed on a midfield or high-field magnet, provides an accurate method of diagnosing and characterizing a full-thickness tear of the rotator cuff. The size of the defect, retraction of tendon, and muscle belly atrophy are well characterized. Partial tears, however, may be missed and/or poorly delineated by routine non-contrast examination. Adhesive capsulitis may not be visible or may be difficult to appreciate on routine examination.

Magnetic resonance imaging offers the potential to demonstrate articular pathology such as labral tear and chondral defect. Detection of labral pathology is optimally performed in a high-field-strength magnet, and MR arthrography (MRA) is essential for the accurate detection and characterization of articular pathology.[36] In fact, some centers recommend the use of MRA in all patients under the age of 25 to 30 years because of the higher incidence of capsulolabral disease in this population. Creative positioning of the patient may increase the detection of both labral and rotator cuff pathology. Rotator cuff tear, if present, in younger patients is statistically partial thickness in nature and these tears are better delineated on MRA. MRA is typically performed by direct injection of dilute gadolinium into the joint. Indirect arthrography performed after the intravenous injection of gadolinium may offer acceptable accuracy in comparison to direct injection of contrast.[37]

Ultrasound can accurately detect a full-thickness rotator cuff tear in the hands of an experienced ultrasonographer.[38] The accuracy may approach that of MRI, but the examination cannot detect or delineate other articular pathology with the accuracy and comprehensiveness of MRI.

Elbow

Elbow injuries in athletes tend to be cleanly separated between acute injury and chronic overuse syndromes. The vast majority of acute injuries are adequately assessed with conventional radiography. Advanced imaging, typically CT, in the setting of acute trauma is used to better characterize a fracture for preoperative planning purposes. Most chronic repetitive injuries of the elbow tend to involve the soft tissues or cartilage and are best imaged with MRI.[39] Non-contrast MRI can usually accurately diagnose and characterize tendon disease (tendinosis, partial tear, and rupture), ligamentous injury (ulnar collateral ligament), and osteochondral defects and intraarticular fragments. The complex of injuries (osteochondral defect and ulnar collateral ligament injury) seen in the elbow of throwing athletes (Little League elbow) is usually adequately evaluated by conventional MR, but MR arthrography may provide a more accurate depiction of the extent of injury.[40] MR arthography may also be useful to more accurately characterize partial tears of the ulnar collateral ligament.[41] Ultrasound of the elbow can be used to assess epicondylitis and other tendinopathies.[42] The ulnar collateral ligament may also be accurately evaluated by experienced sonographers.[43]

Wrist

The wrist is a complex articulation that may be difficult to evaluate by conventional radiography alone, depending on the clinical presentation. Acute fracture and dislocation are usually but not always readily evaluated by radiography alone. Suspected fracture of the hamate may not be seen on plain film and is most cost effectively imaged on CT when radiographs are normal. An athlete with snuff box tenderness and normal radiographs is best imaged by a limited MRI examination.[44,45] A small number of fractures of the scaphoid will be missed if the clinician relies on casting with repeat radiography in 7 to 10 days. Complications of fracture such as avascular necrosis are best imaged by MRI.[46]

Ligamentous and triangular fibrocartilage (TFC) injuries can be evaluated by conventional arthrography or MRI. Imaging of these injuries should be discussed with the treating hand surgeon to determine how the exam changes surgical planning. Many surgeons rely on clinical examination and arthroscopy and do not rely on MRI findings. While fractures of the wrist may be accurately assessed in low- or midfield strength magnets, evaluation of the ligaments and TFC are best performed in high-field magnets with dedicated wrist coils following the injection of intraarticular contrast. The patient's wrist must be in the center of the bore of the magnet, necessitating creative positioning for optimal imaging in most cases. MRI arthrography has been shown to increase accuracy of ligament injury assessment[47] but may not change surgical planning.

Hip

Hip pain in the athlete includes articular, osseous, and soft tissue diagnoses. Careful physical examination is required to ensure hip pain is not secondary to spine, sacroiliac joint, or knee disease. The radiologic evaluation of hip pain is initially performed with conventional radiography to assess for arthropathy, fracture, or osteonecrosis. Radiography offers outstanding specificity but may not be sensitive in the recognition of early disease. Bone scan is useful for detection of stress fracture, osteonecrosis, and arthropathy, but positive results frequently lead to additional examinations such as MRI. CT scan has limited utility in the evaluation of hip disease of the athlete except for delineation of fracture and sacroiliac joint disease.

Magnetic resonance imaging is the examination of choice for assessment of hip pain because of the ability to accurately examine cartilage, bone, bursa, tendon and muscle with a single examination.[48] MRA in a high-field magnet is usually required for the accurate detection and delineation of subtle articular pathology such as labral tear[49] or chondral defect.[50] In our experience, MRI of an athlete with hip pain is more likely to yield a "normal" result than any other exam in the musculoskeletal system.

The snapping hip can be a difficult clinical conundrum. If the snapping is thought to be due to the iliopsoas tendon, this suspicion can be confirmed with either ultrasound or bursography. Ultrasound will confirm that snapping is due to the tendon without the use of ionizing radiation by direct visualization of the tendon.[51] Iliopsoas bursography is an easily performed fluoroscopic examination that not only confirms the diagnosis of snapping tendon but also offers the option of therapy if steroid is added to the injectate.[52]

Foot/Ankle

Foot and ankle injuries are extremely common in athletes and can be separated into two categories, osteocartilaginous and soft tissue, and these two categories can be further subdivided into acute and chronic. Most acute osseous pathology of the foot and ankle is adequately assessed using plain radiography. Chronic osseous injury such as stress fracture may be challenging to detect and document. Rest with repeat radiography in 10 to 14 days is the most inexpensive method to document most stress fractures, but when the athlete's competitive schedule does not permit rest or when plain film is normal, MRI or bone scan are recommended exams.

Chronic ankle pain following an ankle sprain is a common clinical dilemma and is best evaluated by MRI.[53,54] This modality offers the opportunity to evaluate and characterize osteochondral defects,[55] occult fracture, impingement syndromes,[56] and tendon disease,[54] which are common sources of pain in the chronic situation. MR arthrography is usually not required but can improve the assessment of ostechondral defects and impingement syndrome.

Soft tissue injuries of the foot and ankle are best evaluated with MRI or US. Both modalities provide excellent assessment of tendon pathology and US can delineate ligamentous pathology with experienced users.[57] MRI can determine that a ligament has been injured but has unproven utility in the evaluation of instability because apparently intact ligaments on MRI may demonstrate insufficiency on clinical exam.

Successful imaging of the foot and ankle requires accurate clinical history to ensure that the MRI examination is tailored to evaluate suspected pathology. Unlike the shoulder and knee, "one size fits all" protocols may not provide sufficient yield from imaging. For example, suspected forefoot pathology should be imaged differently from suspected midfoot or ankle disease.

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30 COMPLEMENTARY AND ALTERNATIVE MEDICINE FOR THE SPORTS MEDICINE PHYSICIAN

Anthony I. Beutler and Wayne B. Jonas

INTRODUCTION

Western biomedicine (i.e., the medicine practiced in American hospitals) represents just one of the many medical practices and philosophies in the world today. In fact, 80% of the world's population receives their medical care from a system outside of traditional Western biomedicine.[1] These other medical systems and practices are collectively referred to as *complementary and alternative medicine*, or CAM. Complementary and alternative medicine practices continue to increase in popularity and prevalence in traditionally Western populations; however, the safety and efficacy of many CAM practices remain undetermined and the question of how to integrate the evaluation and discussion of CAM therapies in everyday patient care continues to challenge physicians from many specialties. This chapter: (1) briefly defines CAM, (2) identifies which segments of the population are likely to use CAM, (3) outlines an ethical and evidence-supported approach for sports medicine physicians to use in evaluating CAM therapies or in caring for or counseling with athletes, and (4) summarizes the best efficacy and safety evidence for a few of the myriad CAM therapies used by athletes today.

A BRIEF DEFINITION OF COMPLEMENTARY AND ALTERNATIVE MEDICINE

Ayurveda, traditional oriental medicine, and Native American practices predate Western medicine, with spiritualism and traditional oriental medicine both boasting longer histories and larger enrollments than Western medicine.[2] The term *complementary and alternative medicine* is a decidedly Western term that seeks to describe the "broad domain of healing resources … [including] health systems, modalities, practices, and their accompanying theories and beliefs"[3] that fall outside the theories and treatments typically taught in Western medical schools and practiced in Western hospitals. Table 30.1 provides a CAM classification system based on the terminology used by the National Center for Complementary and Alternative Medicine (NCCAM) and the National Institutes of Health.

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TABLE 30.1
Classification of CAM Systems, Therapies, and Modalities

CAM Domains	Examples
Alternative heathcare systems	Traditional oriental medicine
	Acupuncture
	Ayurveda
	Homeopathy
	Naturopathy
	Chiropractic medicine
	Native American practices
	Tibetan medicine
Biological and diet therapies	Herbal therapies
	Orthomolecular medicine (megavitamin therapy)
	Special diets (macrobiotics, low-fat diets, high-carbohydrate diets)
	Nutritional biologic supplements (shark cartilage, bee pollen, glucosamine)
	Herbal medicine
	Antineoplastons
	Cell treatment
	Chelation therapy
	Neural therapy
Manual healing	Massage therapy
	Alexander method
	Acupressure
	Biofield therapy
	Chiropractic medicine
	Osteopathy
	Reflexology
	Feldenkrais
	Rolfing
Mind–body control	Meditation
	Yoga
	Hypnosis
	Biofeedback
	Guided imagery
	Prayer and mental healing
	Activity therapies (art therapy, dance therapy, music therapy, humor therapy)
Energy therapies	Therapeutic touch
	Qigong
	Reiki
Bioelectromagnetic applications	Magnetic field therapy

Sources: Adapted from Jonas, W. and Chez, R., *Curr. Family Med.*, 2003, in press; Najm, W., in *Textbook of Primary Care Medicine*, 3rd ed., Noble, J., Ed., Mosby, St. Louis, MO, 2001; the National Center for Complementary and Alternative Medicine (nccam.nih.gov).

The term CAM is exclusionary by definition in that it is Western biomedicine's name for everything that lies outside its bounds; however, the boundaries of CAM remain imprecise. For example, are glucosamine prescriptions for osteoarthritis and dry needling of chronic tendinosis CAM treatments or accepted Western treatments? Indeed, as our understanding of CAM techniques and treatment continues to evolve, so do the boundaries and definitions of what is considered CAM. Still, however unwieldy and imprecise the term, the words *complementary* and *alternative* do accurately describe the utilization of these practices by individuals in Western society. Survey data suggest that less than 5% of those who use CAM do so exclusively, or as an alternative to conventional biomedicine.[4] The remaining 95% utilize CAM to complement, or in addition to, conventional medical treatment.

WHO USES COMPLEMENTARY AND ALTERNATIVE MEDICINE?

Americans spend more than $27 billion out of pocket annually on complementary and alternative medicine.[5] Visits to CAM practitioners rose from 400 million per year in 1990 to 600 million per year in 1996. In the year 2000, visits to complementary and alternative medicine providers rose to 65 visits per 1000 population per month. Primary care clinicians saw 113 visits per 1000 population per month in that same time period.[6] CAM use rates hover around 50% of the population in the United States, United Kingdom, and Australia and reach 75% in France. With 100 million Americans supplementing their diets with vitamins, herbs minerals, and amino acids, perhaps the more appropriate question is "Who doesn't use CAM?"

Many large, well-designed surveys reveal that CAM users tend to be better educated, wealthier, and more likely to have a chronic disease or chronic pain than CAM nonusers.[4,7–10] Minorities, especially African-Americans, are less likely to use CAM, while women are more likely to use CAM. CAM users are more likely to hold a holistic philosophy toward their health. Interestingly, dissatisfaction with conventional care does not predict those who use CAM as a complement to conventional medicine; however, mistrust of and disaffection with conventional medicine are more common in those who use CAM exclusively (alternatively), as is a desire to retain control over one's health care.[5–7,9]

The population of many developing countries relies on CAM practices for most or all of their healthcare needs.[11] And, even in countries dominated by Western biomedicine, the public employs CAM practices in the treatment of a wide array of major and minor medical problems. Popular and multiple CAM remedies exist for minor ailments such as colds, acne, muscle pain, and arthritis. On the other side of the disease spectrum, 50% of patients with cancer and 50% of those with human immunodeficiency virus (HIV) will utilize CAM therapies over the course of their illness.[12] Yeh et al.,[8] however, note an important distinction in their recent study of CAM use in patients with diabetes mellitus. While 57% of those with diabetes used CAM therapies in a year, only 20% used these therapies to treat their diabetes. Yeh notes that CAM use is likely to be less in maladies where the population believes that traditional medicine has a good understanding of the disease process and effective treatments for the disease itself.

USE OF CAM AMONG ATHLETES

Athletes use CAM therapies to enhance performance and to speed return to play following an injury. Caffeine, creatine, guaraná, and *Gingko biloba* are just a few examples of substances with purported performance-enhancing effects. Microcurrents, electrical stimulation, iontophoresis, spinal manipulation, and acupuncture represent a smattering of the myriad of treatments used to control pain and speed the athlete's return to the playing field. The high pressure and high stakes of athletic competition demand heightened vigilance on the part of the sports medicine physicians to ensure the safety of those entrusted to their care.[13]

THE ROLE OF THE SPORTS MEDICINE PHYSICIAN IN CAM

Sports medicine physicians should help patients make informed choices about CAM treatments and therapies, just as they would assist their patients in decisions involving conventional medical treatments. While many effective strategies may be employed in advising CAM patients, the strategy proposed by Jonas[14] suggests that the physician's counsel should vary depending on the specific patient and specific treatment being discussed. Specifically, the sports medicine physician should *protect*, *permit*, *promote*, and *partner* with their patients regarding CAM practices.[15]

Protecting Patients from Risk

Not surprisingly, conventional physicians are frequently faced with questions about the safety of

CAM. Many CAM practices present minimal risk to the patient when performed by competent providers; however, two of the most popular CAM practices in the United States — herbal and high-dose vitamin supplementation — have potential for serious, even fatal toxicities.[16] Many patients harbor the false assumption that "natural" equals "safe." A very public example of the risk of some CAM supplements and the naiveté of many athletes occurred with the recent death of Steve Bechler. A professional pitcher for the Baltimore Orioles, Bechler's collapse and death during spring training of 2003 was at least partially due to the diet herb ephedra.[17] Hence, the sports medicine physician should remind his patients that many known pharmacologically active herbs have real effects, real side effects, and relatively narrow therapeutic indices. Examples of these herbs include foxglove (digoxin), belladonna, ergot (ergotamine), and colchicum (colchicine). Even in the absence of direct toxicity, polyconventional and poly-CAM pharmacy creates the potential for herb–herb, drug–drug, and drug–herb interactions that can be serious.[18] Also problematic is the lack of quality control in herbal and nutritional supplements. Contamination, varying potencies, differing absorption rates, and other quality issues with these unregulated supplements have been well documented.[16]

Many CAM treatments involve minimal risk for direct toxicity, including biofeedback, meditation, prayer, and acupuncture (see Table 30.2). But, even these innocuous practices may indirectly cause harm if used in place of more effective treatments. While the patient must be allowed to make the final decision regarding which therapies to pursue, the physician should detail the direct and indirect risks (as well as benefits) associated with all therapeutic options.

Ephedra (ma huang) acts as a stimulant to raise heart rate, blood pressure, and central nervous system (CNS) awareness.[19–21] Studies of ephedra and other drugs in its class show no performance-enhancing benefit at therapeutic dosages used in colds or asthma or at even twice therapeutic dosage.[20–25] Performance enhancement may occur at dosages greater than three times therapeutic,[26] or when ephedra is combined with caffeine;[27–29] however, at these higher dosages, ephedra has effects similar to those seen in methamphetamine use, with side effects of stroke, mania, cardiac arrhythmias, and death.[30] Caffeine is known to potentiate the effects and side effects of ephedra,[31] and caffeine and ephedra combinations have been banned by the U.S. Food and Drug Administration (FDA) since 1983;[32] however, many dietary supplements circumvent this ban by combining "natural" ephedra sources with guaraná, an herbal form of caffeine. Natural ephedra product purity is predictably poor. A recent review of 20 ephedra-containing herbal supplements showed that the majority contained many different types and quantities of various ephedra alkaloids.[33] Five supplements contained norpseudoephedrine, a Schedule IV controlled substance. The total ephedra content of each capsule ranged from 0 to 154% of the stated label content.[34] The systemic use of ephedra containing products, including cold remedies, is banned by the National Collegiate Athletic Association (NCAA) and International Olympic Committee (IOC). Clearly, then, because performance enhancement occurs only in dosages or combinations with serious side effects and given the possibility of violating antidoping regulations, sports medicine physicians should protect their patients against ephedra supplementation (Level of Evidence A, randomized controlled trials).

Permitting the Use of Unproven Therapies

Many physicians feel uncomfortable allowing their patients to engage in unproven therapies; however — if the therapy has little potential toxicity and is not used as an alternative to proven, more efficacious treatment — the wise physician should consider the effects of spontaneous healing and placebo. Even if this relief comes through nonquantifiable spiritual means or a placebo effect, it will still be welcomed by the patient and should be accepted by the physician as well. Ideally, the physician should blend and utilize the specific and nonspecific factors in all remedies to maximize patient benefit and comfort.[35] Homeopathic arnica (a CAM treatment frequently used for muscle bruising and soreness[36]) represents a therapy that can often be safely permitted by the sports medicine physician (Level of Evidence B, non-randomized clinical trial). Assuming that homeopathy is not used as an alternative to a proven therapy, its potential for toxicity is low and the risk of side effects virtually zero. Though toxicity from the compounds used in homeopathy may be theoretically possible, the dilute solutions and tiny doses employed make this unlikely.[37] The risk from biologic contamination is also quite low as many homeopathic remedies are diluted with high concentrations of alcohol. Homeopathy involves a system of individual prescription that emphasizes holism and seeks to

TABLE 30.2
Summary of Evidence and Recommendations for Common CAM Therapies

CAM Therapy	Purported Benefit	Evidence	Cost[a]	Toxicity (Side Effects)	Cautions (Interactions)[b]	Regulated Substance	Recommended Action
Glucosamine	Relief of osteoarthritis (OA) pain and stiffness, temporomandibular joint (TMJ) dysfunction	Multiple trials and meta-analysis consistently show that glucosamine sulfate is more effective than placebo for OA.[58–61] Recent data suggest that pain relief is similar to or better than NSAIDs,[62] especially long term.[63] Limited evidence suggests that glucosamine may slow progression of OA.[64] Most studies involve knee OA, but more limited data suggest that glucosamine may be effective in TMJ syndromes[65] and OA of the spine.[66]	Moderate ($1–$2 per day)	Mild gastrointestinal distress, comparable to placebo	Use with caution in diabetics or impaired glucose tolerance (IGT); may increase insulin resistance. Concern for shellfish allergy, but no reactions reported.	No	*Promote* — effective treatment for OA. If no clinical response after 6 to 8 weeks of supplementation, discontinue due to moderate cost.
Ephedra (ma huang, herbal ecstasy, teamster's tea, zhong ma huang)	Weight loss, enhanced athletic performance, respiratory conditions, asthma	Small, short-term weight loss (2.7–5.3 kg) over 6 wk–6 mo in patients with BMI < 30 when used with other stimulants (caffeine, guaraná, etc.)[67,68] Not thought to improve athletic performance[69–72] unless used in combination with caffeine[73,74] or in very high doses.[75,76]	Low ($0.30–$0.75 per day)	Dizziness, restlessness, anxiety, heart palpitations, tachycardia, hypertension, myocardial infarction, stroke	Adverse effects occur even in healthy patients; capsules often contain impurities, including banned substances.	Banned by International Olympic Committee (IOC) and likely to be restricted by FDA in near future.	*Protect* — As negative publicity builds, the number of ephedra-free products increases; however, these may not be any safer than original ephedra products.
Chondroitin	Relief of osteoarthritis, topical use for relief of dry eyes	Several trials suggest that chondroitin + NSAIDs is more effective than NSAIDs alone in OA.[77–80] Combination tablets contain glucosamine, manganese, and chondroitin, but it is unclear if combination therapy is better than treatment with any individual component.[81] Topical uses include FDA-approved product for cornea/cataract treatment (Viscoat®) and investigational treatment for dry eyes.[82]	Moderate ($1–$2 per day); often sold in "joint health" combos	Mild gastrointestinal distress comparable to placebo; combo tablets may exceed safe daily dosage of manganese and cause central nervous system irritability	Potential exists for contamination with diseased animal parts. Use with caution in patients taking anticoagulants (chondroitin has heparinoid structure and may have anticoagulant effects)	No	*Permit* — No adverse effects in 5-year studies; if no clinical effect is noted after 6 to 8 weeks of therapy, discontinue chondroitin (due to moderate cost).

TABLE 30.2 (CONTINUED)
Summary of Evidence and Recommendations for Common CAM Therapies

CAM Therapy	Purported Benefit	Evidence	Cost[a]	Toxicity (Side Effects)	Cautions (Interactions)[b]	Regulated Substance	Recommended Action
Homeopathy (arnica)	Relief of delayed onset muscle soreness (DOMS)	Homeopathy is a medical system involving multiple pharmacologic treatments. No single homeopathic treatment has conclusively demonstrated efficacy in DOMS.[83–85] A few, small trials have reported positive results in marathon runners;[86,87] similar trials exist that report no such benefit.[88] Trials are limited by their small treatment groups, the bewildering array of investigated homeopathic remedies, and the differing methods of inducing and defining DOMS.	Low ($0.10–$0.20 per day)	Headaches, fatigue, rash, dizziness, gastrointestinal upset, and increased symptoms reported but not above placebo levels[89]	Reports of severe allergic reactions appear anecdotal.	No	*Permit* — The homeopathic system is too complex to be adequately evaluated by the current evidence; however, costs are low and toxicities minimal in the hands of trained practitioners.
Magnetic field therapy (pulsed electromagnetic field therapy, PEMF)	Decreased pain and stiffness in osteoarthritis	PEMF therapy is already considered a proven remedy for delayed union fractures.[90] Similar fields have been found to stimulate proteoglycan synthesis in chondrocytes,[91] which has fueled interest in treatment. Anecdotal accounts abound on Internet sites; however, a recent *Cochrane Review* found only three published articles that met inclusion criteria for their meta-analysis.[92] The meta-analysis concludes that, while PEMF therapy produces statistically significant reductions in knee OA pain and disability, pain and disability from cervical OA is not significantly reduced. Moreover, though statistically significant, the reduction in knee OA symptoms is not likely to be clinically significant. Many questions remain regarding optimum dosage, frequency, and technical specifications of PEMF therapy.	High ($20–$200 per day)	Unknown; effects of PEMF treatment on body tissues are unknown	Caution is advised in this unregulated arena; high-cost interventions with no proven benefit may create potential for fraud and abuse.	No	*Protect* — High-cost treatment has unknown side effects and uncertain efficacy. Until completion of further study on optimum treatment regimens and long-term effects or toxicities, protect patients from "high-tech" regimens that provide none of the promised relief.

Acupuncture	Decreases in: 1. Lateral elbow pain 2. Low back pain	Like homeopathy, acupuncture can be considered an alternative health care system. The limited available evidence is insufficient to completely endorse or refute this system as a whole. For acupuncture in lateral elbow pain, a recent *Cochrane Review*[93] found four small RCTs, all unique enough to preclude combination into a single meta-analysis. The review concludes that no pain decrease has been demonstrated beyond 24 hours and that insufficient evidence exists to support or refute the use of acupuncture in lateral elbow pain. Acupuncture for low back pain is discussed in a separate *Cochrane Review*,[94] in which 11 RCTs are summarized, most of poor quality. The highest quality RCTs compare acupuncture with placebo or sham acupuncture; of these, one reported positive results[95] and one negative results.[96] The review concludes that acupuncture should not be used in treating low back pain because current evidence suggests it is ineffective and other proven, effective therapies exist.	Variable ($20–$100 per session)	Commonly reported side effects: pain, fatigue, bleeding, fainting, nausea, and dizziness	Pneumothorax, broken needles, and increased risks of infectious disease have been reported but are uncommon in the hands of licensed practitioners.	No	*Permit* — A preponderance of evidence suggests that acupuncture is effective for postoperative nausea and dental pain.[97] Unfortunately, acupuncture has no proven efficacy in lateral elbow or low back pain. It may have some effect in osteo-arthritis; however, other, lower cost treatments exist for these maladies. Patients should be permitted to use acupuncture for OA with a warning regarding lack of data and potential for high costs.

TABLE 30.2 (CONTINUED)
Summary of Evidence and Recommendations for Common CAM Therapies

CAM Therapy	Purported Benefit	Evidence	Cost[a]	Toxicity (Side Effects)	Cautions (Interactions)[b]	Regulated Substance	Recommended Action
St. John's wort	*Oral:* antidepressant, anxiolytic, insomnia, adjunct to weight loss *Topical:* wound healing, anti-inflammatory cream	Considerable, but not unanimous evidence for efficacy in mild–moderate depression.[98–101] Antidepressant efficacy is better than placebo, similar to low-dose tricyclic antidepressants (TCAs)[102,103] and slightly less effective than selective serotonin reuptake inhibitors (SSRIs).[104 105] St. John's wort should not be used in severe depression, as higher dosages increase the risk for potentially severe skin reactions. Preliminary evidence suggests that St. John's wort may be effective in obsessive–compulsive disorder (OCD).[106] St. John's wort has *in vitro* antibacterial, antiviral, and anti-inflammatory properties that would support its topical use;[107,108] however, no clinical data exist.	Low ($0.10–$0.50 per day)	Insomnia, restlessness, agitation, anxiety, mild GI distress, headache; daily dose over 2 g (5 mg hypericin) increases risk for potentially severe photodermatitis; fewer side effects than TCAs and SSRIs	Multiple, potentially severe herb–herb and herb–drug interactions (TCAs, triptans, oral contraceptives, anti-HIV drugs, warfarin, digoxin) are possible. Combined use with other antidepressants may increase risk for serotonin syndrome.	Not banned by athletic regulatory agencies; banned by government in France. United Kingdom, Japan, and other European governments are investigating.	*Permit* — Acceptable in monitored patients with mild–moderate depression with simple medical regimens if patient prefers it over conventional treatments.

Ginkgo leaf (*Ginkgo* seed is also used as a supplement, but has different purported efficacies and toxicities than *Ginkgo* leaf)	*Oral:* slows onset of dementia and memory loss, relieves claudication and premenstrual syndrome (PMS) *Topical:* used to treat chilblains (mild cold injury) and improve blood flow in wound healing	Majority of evidence indicates some effect in stabilizing or slowing dementia progression.[109–111] In other countries, *Ginkgo* is the treatment of choice for dementia,[112] with effects similar to cholinesterase inhibitors used in the United States.[113] *Ginkgo* appears effective in increasing cognitive function in middle-aged individuals without complaints of memory loss.[114,115] *Ginkgo* + *P. ginseng* may be even more effective for this indication (see below). *Ginkgo* also appears to increase walking distance in patients with vascular claudication.[116,117] Insufficient clinical evidence exists to evaluate the other purported benefits of *Ginkgo* therapy.	Moderate ($1–$2 per day)	Mild gastrointestinal upset, palpitations, constipation, headache, skin reactions; diarrhea, vomiting, and weakness in larger doses; serious spontaneous bleeding and seizures reported with *Ginkgo* use	Does not "reverse" dementia and is not a cure for Alzheimer's. Use with extreme caution in patients on anticoagulants or antiplatelet therapy. Avoid in patients with seizure disorder or with other drugs that lower seizure threshold.	No	*Permit* — Acceptable in monitored patients without medical contraindications if patient prefers it over conventional drug treatments; contraindicated in patients on anticoagulants.
Creatine	Increases lean body mass, improves strength, improves athletic performance	Considerable, but not unanimous, support exists for an ergogenic effect in repetitive strength tasks lasting <30 sec;[118,120,121] this effect may be most pronounced in individuals with low to normal levels of endogenous muscle creatine.[122] Increased body mass may impair performance in endurance events. Caffeine consumption may negate ergogenic effects of creatine supplementation.	Low ($0.25–$0.75 per day)	Common side effects: gastrointestinal upset, diarrhea, cramping; reported side effects: cardiomyopathy, renal failure, arrhythmias, rhabdomyolysis	Use with caution with other nephrotoxic drugs (i.e., NSAIDs, cyclosporine, aminoglycosides)	No; the National Collegiate Athletic Association (NCAA) prohibits universities from providing creatine for their athletes.	*Permit* — Can be used after discussion of risks and benefits in healthy patients in appropriate events. Not appropriate for use in athletes with kidney disease, pediatric athletes, or athletes prone to dehydration.

TABLE 30.2 (CONTINUED)
Summary of Evidence and Recommendations for Common CAM Therapies

CAM Therapy	Purported Benefit	Evidence	Cost[a]	Toxicity (Side Effects)	Cautions (Interactions)[b]	Regulated Substance	Recommended Action
Massage therapy	Relief of delayed onset muscle soreness, minimize strength loss after exercise	A few, small trials show mixed results for preventing DOMS with post-exertional massage therapy. Most studies suffer from small sample sizes. Three trials show a report a significant positive effect of massage on DOMS;[123–125] three others show no significant effect or have such severe methodological flaws that their results cannot be interpreted.[126–128] A recent review concluded that, while massage is a promising therapy for DOMS, larger, more rigorous trials are required.[129]	High; greatest effects found in repeated massage sessions for 48 to 92 hours post-exercise	No known toxicities	None	No	*Permit* — Despite high cost and questionable efficacy, no harmful effects have been reported from massage.
Spinal manipulation	Relief of low back pain and low back stiffness; allows more rapid return to play following low back injury	Bulk of evidence suggests that spinal manipulation is at least as effective as conventional treatment for low back pain.[130–133] A recent review noted that 9 of 10 RCTs with the highest quality scores concluded that spinal manipulation was better than the control treatments.[131] However, justifiable concern still exists for differences in manipulation techniques and case reports of severe complications.[133] Some authors distinguish between sciatica and low back pain (LBP). Most of these discourage manipulation for sciatica; however, insufficient evidence exists to support or refute this view.[134]	High; *New England Journal of Medicine* reviewed 8 visits in 12 wks (may be covered by insurance)	None	Rare reports of stroke, paralysis, or spinal chord damage in the literature, but almost exclusively from C-spine manipulation. No severe complications reported in over 15,000 patients enrolled in RCTs.[131]	No	*Permit* — In the hands of a competent practitioner, L-spine manipulation is safe and likely effective, although it is higher cost than conventional treatment.

Cromium picolinate	Increases muscle mass, decreases body fat, improves glycemic control in diabetes, lowers cholesterol, treats mild depression	Increased muscle and decreased fat were suggested by early, poorly designed studies.[135,136] Better, newer studies suggest no ergogenic or fat-burning effect.[137–138] Improved glycemic control and lipid profiles are produced in diabetic patients;[139,140] time to effect and size of effect depend on daily dose. Promising initial studies have been done on chromium in depression, but more data are needed.[141]	Low ($0.10–$0.30 per day)	Cognitive and motor dysfunction Sleep changes, mood changes, headaches	Not a cure for diabetes; some concern for DNA mutagenic effects with high-dose, long-term use[142]	No	*Protect* — Minimal toxicity and low cost with short-term use are outweighed by risk of DNA mutations and no proven limited benefits of long-term chromium use.
Panax ginseng (different species of *ginseng* are not equivalent and in many cases are not even biologically related; the data reported here are for *Panax ginseng*, the most commonly used *ginseng*)	Improve athletic performance, increase cognitive function, and treatment of many other ailments	Multiple studies suggest no enhanced athletic performance with oral *ginseng* use.[143–146] *P. ginseng* use has not been shown to improve memory,[147] but *P. ginseng* + *Ginkgo* may be effective in improving memory in healthy, middle-aged people.[148] Minimal clinical evidence suggests that *ginseng* might be effective in diabetes mellitus treatment,[149] in decreasing cancer risk,[150] in improving mood,[151] and in preventing influenza and the common cold.[152]	Low ($0.25–$0.75 per day)	Insomnia, tachycardia, palpitations, mastalgia, vaginal bleeding, amenorrhea	Intensifies effects of other stimulants (caffeine, guaraná, tea); *ginseng* has hormonal (estrogenic) effects; continuous, long-term (>3 mo) use is not well studied.	No	*Permit* — Studies suggest minimal toxicity in short-term use. Long-term use is questionable due to risk of estrogen-like effects and minimal reported benefits of *ginseng*

[a] Cost data from www.epocrates.com and www.drugstore.com, unless otherwise referenced.

[b] The authors are grateful to the owners of the Natural Medicines Comprehensive Database who made their database available. Herbal toxicities and interactions are from this database, unless otherwise referenced.

treat patients based on their temperament, personality, and emotional responses, as well as their medical diagnoses.[38] The sports physician should permit the integration of CAM therapies that "are neither harmful nor expensive, but that may enhance [these] non-specific healing factors."[39]

Promoting Proven CAM Therapies

Sports medicine physicians should promote safe, effective, and proven therapies to their patients, regardless of the medical system from which these treatments originate. As medical knowledge continues to expand, patients and physicians find themselves with more treatment options and more information about the efficacy of different treatment strategies. Sports medicine physicians should guide their patients to safe, effective, and proven treatments. Glucosamine treatment for knee osteoarthritis is an example of a CAM therapy that should be promoted. In animal and *in vitro* models of osteoarthritis, glucosamine improves cartilage metabolism, rebuilds damaged cartilage, and demonstrates some anti-inflammatory properties.[40] More recently, a meta-analysis of glucosamine treatment concluded that glucosamine supplementation demonstrated a moderate to large effect on osteoarthritis symptoms.[40] The cost of glucosamine supplementation is relatively high (approximately $1/day) when compared with generic ibuprofen ($.05/day) but is substantially lower than commonly prescribed, once-daily NSAIDS and the new COX-2 inhibitors, which typically cost in excess of $2 per day of therapy (drug prices from *The Medical Letter* and epocrates.com). Other than mild gastrointestinal upset, glucosamine has no known side effects or toxicities. Clearly then, sports medicine physicians should present glucosamine supplementation as a viable and favored alternative to their patients with osteoarthritis (Level of Evidence A, meta-analysis of randomized controlled trials).

Partnering with Patients Who Use CAM

Over 60% of patients who use CAM do not inform their physicians that they are using CAM treatments.[5] This CAM communication gap between physicians and their patients results in a wasteful, as well as potentially dangerous, environment. Effective partnering requires that physicians understand why patients choose alternative practices, that physicians have access to reliable data detailing the safety and efficacy of CAM practices, and that physicians actively question and communicate with patients about their CAM use.[42] Patients use CAM therapies for a variety of reasons. They may be enticed by marketing schemes or by anecdotal success stories from friends. Partnering with these patients may require an explanation of the role of science in medicine and encouragement that they incorporate all types of evidence into their health care decisions.[14] However, studies suggest that these patients represent the minority of CAM users. Most patients choose CAM because conventional therapies have significant side effects or risks, have not been effective in relieving their condition, or are out of harmony with their emotional or spiritual values.[4,5,43] These same studies also show that CAM users are more likely to be educated than ignorant, more likely affluent than poor, and more likely holistic and spiritual than merely neurotic. In short, many patients that use CAM practices exhibit character traits that incline them to actively participate in their medical care. The physician who chooses to repudiate all knowledge and to discourage discussion of CAM practices is most short sighted.

Effective partnering also requires that the physician have access to reliable information on the safety and efficacy of CAM practices. While a comprehensive database of all reviews of CAM treatments is not yet a reality, several commercial sources are available. The Cochrane Collection conducts and publishes systematic reviews of randomized controlled trials for all heath care practices, CAM or traditional. The abstracts of the *Cochrane Reviews* are available free of charge at www.cochrane.org. Full-text reviews can be ordered online or viewed online on a fee basis. An additional source of information is the Natural Medicines Comprehensive Database. Although dealing mostly with herbal and vitamin supplementation, the database has separate editions for medical professionals and consumers. Both versions may be viewed online as a subscription service at www.naturaldatabase.com. Additional resources for CAM information are listed in Table 30.3. Equally important to information access, however, is the physician's system for evaluating the evidence, applying the evidence responsibly to their patient population, and, finally, assisting each patient to make healthcare decisions based on the best available information for that individual.

TABLE 30.3
Sources of CAM Information for Healthcare Practitioners

Source of CAM Information	Description	Where to Go
The Cochrane Library	*Database of Systematic Reviews:* Systematic reviews of RCT of CAM and conventional therapies *Controlled Trials Register:* Extensive bibliographic listing of controlled trials and conference proceedings	www.cochrane.org gateway.ovid.com
Natural Medicines Comprehensive Database	Comprehensive listing and cross-listing of natural and herbal therapies; separate "all known uses" and "effectiveness" sections, safety ratings, mechanisms of action, side effects, herb–drug interactions, and review of available evidence	www.naturaldatabase.com
National Library of Medicine	Powerful search engine that allows searches of PubMed and all government guidelines combined; includes "synonym and related terms" option	*Search engine:* hstat.nlm.nih.gov *Individual guidelines:* www.guideline.gov; www.cdc.gov/publications
Focus on Alternative and Complimentary Therapies (FACT)	Quarterly review journal of CAM therapies. Contains evidence-based reviews, focus articles, short reports, news of recent developments, and book reviews on complimentary medicine.	www.exeter.ac.uk/FACT
PubMed Clinical Queries Search Engine	Includes clinical queries filter to limit search results (click Clinical Queries on the left blue banner to access the filter); for the most comprehensive search, use the key words "complementary medicine"	www.pubmed.org
National Center for Complementary and Alternative Medicine	*Clinical trials section:* Listing of clinical trials indexed by treatment or by condition; cross-linked to www.clinicaltrials.gov and PubMed	www.nccam.nih.gov

EVALUATING THE EVIDENCE

So how do we evaluate the at times overwhelming mountain and sometimes embarrassingly small molehill of medical evidence? The first important step is to realize that evidence comes in many different types and forms. For the sake of discussion, we will say that evidence is like food. We all want good food to eat, and we all want good evidence on which to base medical decisions. So what makes food and medical evidence good? We can easily agree on basic standards for purity and hygienic preparation for food. Contaminated food causes sickness instead of sustaining health. Similarly, tainted evidence may overstate findings, make false claims, or be misleading in other ways. Instead of enlightenment, impure evidence can lead to epidemics of error. (An overview of the important process of distinguishing evidence from propaganda and advertisement is provided in Box A.) However, outside of these basic standards of hygiene, what constitutes good food or good evidence depends largely upon our own objectives and personal biases. For example, many people believe that the ideal diet of good food can be described by the food pyramid. The food pyramid provides a hierarchy of what types of foods should be emphasized in an individual diet (e.g., grains and vegetables being preferred over meats; eating more fruit and less sugar and fat), but this hierarchy does not hold for all individual situations. Good food for individuals with diabetes mellitus, celiac sprue, or lactose intolerance may require substantial modifications to the food pyramid, while planning party refreshments for a 7-year-old's birthday may require abandoning the hierarchy all together!

Evidence-based medicine (EBM) attempts to establish a hierarchy for medical evidence (Figure 30.1). In the hierarchy of EBM, case series represent

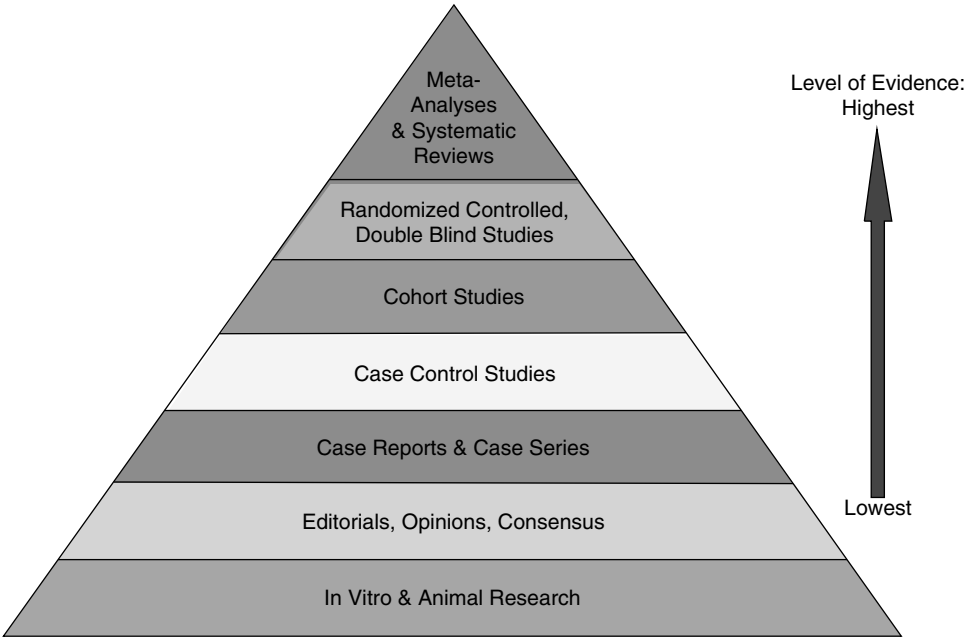

Figure 30.1 The evidence hierarchy established by evidence-based medicine (EBM) may be conceptualized as a pyramid.

a lower grade of evidence. Higher grades, in order of ascendance, are observational studies, nonrandomized trials, and then randomized controlled trials (RCTs), with systematic reviews or meta-analysis representing the pinnacle, or highest form of evidence. The hierarchy of EBM is based on the scientific method, which uses experimentation and observation to establish causal links between intervention and outcome. In many circles, evidence-based medicine has become nearly synonymous with good medicine; meta-analyses and randomized controlled trials now connote good evidence. However, the evidence hierarchy of EBM only provides good answers for EBM questions. EBM is unquestionably useful for establishing a specific treatment (CAM or conventional) as the cause for clinical improvement and for determining the relative efficacies of different therapies. The randomized controlled trials of EBM can provide clear quantification of the risks and benefits of CAM treatments compared to another CAM treatment, compared to conventional treatment, or compared to placebo treatment. However, because RCTs depend on the controlling or freezing of many variables, they are difficult to sustain over long periods of time and hence are less valuable for the study of chronic disease.[44]

Clinical outcomes research is a less recognized source of good evidence on which to base patient counseling and clinical decisions. Outcomes research is more like clinical practice, as it involves a wide range of patients quality of life data and longer periods of time.[45] Rather than looking for a specific, therapeutic cause and measurable effect, outcomes research provides the probability of a therapeutic effect and how large or small that effect is in routine clinical care.

The best type of evidence to use in evaluating a specific CAM treatment depends on the nature of the therapy, the type of information desired, and the individual patient (see Figure 30.2). Randomized controlled trials provide essential safety and risk–benefit data for therapies that are potentially costly, toxic, or high risk. For low-risk therapies or treatments used in chronic conditions, clinical outcome data can provide information on the probability and magnitude of long-term benefit or harm.[46] The patient's individual beliefs (e.g., prior plausibility) can also influence the evaluation of a CAM treatment.

Jonas and Chez[47] suggest building an evidence *house* instead of an evidence *hierarchy*. The evidence house would have a room for mechanism of action studies, a room for pharmacokinetics, and a room for RCTs and systematic reviews. The house also would also have a room for outcome studies of long-term data and a room for clinical relevance. Regulatory authorities and basic scientists may be

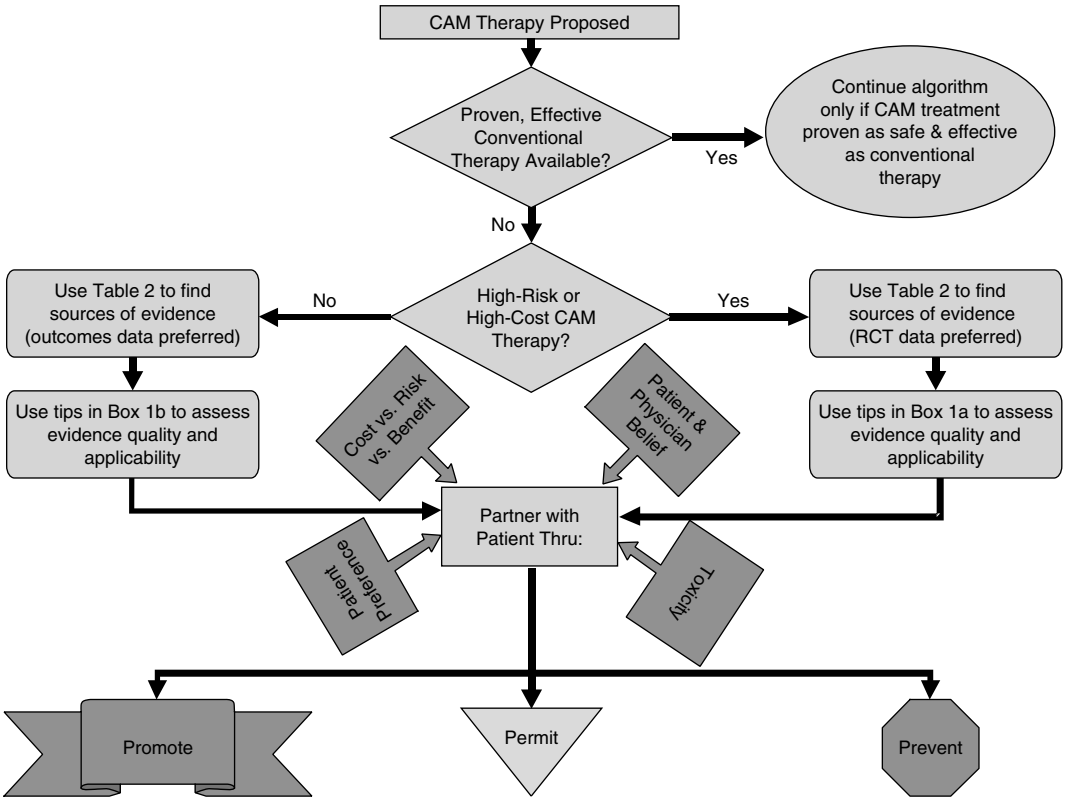

Figure 30.2 Flowchart of CAM decision-making process.

most interested in the RCT and pharmacokinetic rooms, while healthcare providers and patients may be more interested in the outcomes room or stories of cure room. Each room, like each type of evidence, may serve a different purpose, but every room should be constructed from materials of the highest quality. The house of evidence concept emphasizes that if we select one type of evidence at the exclusion of others we will make important clinical decisions based on incomplete information.[48]

CONCLUSIONS

Effective patient–physician partnering requires that the sports medicine physician carefully evaluate good evidence from many sources to protect, permit, or promote specific CAM therapies. Understanding who chooses CAM treatments and why they choose CAM can allow physicians from all disciplines to close the dangerous CAM communication gap. Providing evidence-supported answers to CAM questions will allow sports medicine physicians to continue to deliver a high standard of quality and compassionate care to their uniquely motivated patients.

BOX A

Evaluating Medical Evidence

Evaluating the medical evidence is an important skill for sports medicine physicians and physicians in all disciplines. A complete discussion of how to read a research paper is beyond the scope of this chapter, but several such comprehensive guides have been published and are available.[49–54] The following steps provide a brief overview of a literature assessment strategy designed to assist the sports medicine physician in evaluating the quality of published evidence. The specifics of evaluating a randomized controlled trial (RCT) differ slightly from the evaluation of qualitative outcomes research; however, the basic questions are the same for both processes and will be treated jointly in an effort to provide a concise and practical guide to evaluating the medical evidence. The method outlined here is adapted from Greenhalgh and Taylor's series, *How To Read a Paper*.[49] By using these five questions to evaluate published evidence, the sports medicine physician can estimate the quality of an individual

article or assess the body of evidence that exists for a specific CAM or conventional treatment.

Evaluating a Randomized Controlled Trial

I. Did the Study Ask a Valid Question?

The first step in evaluating a research publication is to ask whether the research question is an important one. Or, more specifically, does the study address an issue that matters to you and your patient? For example, if a 25-year-old female patient wants to know about using homeopathy to decrease muscle soreness after running a marathon, a RCT comparing homeopathy to conventional antiemetics for the treatment of hyperemesis gravidarum is not likely to be helpful in answering her question. The study should also measure valid clinical endpoints rather than presumed biochemical markers; for example, a trial demonstrating a decrease in muscle calmodulin levels in rats receiving a homeopathic solution is intellectually interesting but clinically unconvincing. A more useful trial would be a randomized controlled trial that measured perceived muscle soreness in marathon runners receiving either homeopathy or saline drops.

II. Whom Is the Study About?

No matter how appropriate the research question and how well designed a study may be, the results will only be applicable if the study population is similar to the patient. Ask yourself the following questions:[49]

- *Who was included in the study?* Does the study group's age, education, and gender match that found in your clinical practice?
- *Who was excluded from the study?* Were patients with co-morbid disease excluded from the study? Was the level of co-morbid disease higher in the study than in your sports medicine practice?
- *Did the subjects receive a realistic level of care?* Were the study patients given extensive patient education? Did they have increased access to healthcare providers and healthcare support as a result of their being in the study? Did the sponsoring company provide equipment that would not be available to your patients?

While none of these issues may be sufficient to completely invalidate the results or reproducibility of a study, they may raise issues about the applicability of the study data to patients in your practice.

III. Does the Study Design Match the Study Question?

Another valid question is if the research method is appropriate to the study question. Randomized controlled trials are structured experiments that compare one or more interventions with each other or placebo. RCTs are useful for asking quantitative research questions of "how many" or "how much" of this compared to that. An appropriate RCT question would be "Does 100 mg of saw palmetto or 2 mg of terazosin yield greater improvement in urinary stream in 50-year-old men?" If the studied outcome is more qualitative — "Why do 50-year-old men complain of more nocturia than 60 year-old men?" — then an RCT is less likely to be appropriate.

The length of follow-up in the study is another important variable. A study comparing the analgesic properties of capsaicin cream with codeine in post-operative patients may only require a 48- to 72-hour follow-up period; however, a study that examines the effect of *Ginkgo* supplementation on memory loss should involve years of follow-up. A classic example that illustrates this need for long-term follow-up in studies examining chronic therapies is Vaz's report on the efficacy of glucosamine compared to ibuprofen in osteoarthritis.[55] Vaz showed that after 2 weeks of therapy, patients with knee osteoarthritis who were treated with ibuprofen had less knee pain than a similar group treated with glucosamine. However, at 4 weeks of therapy, the pain scores of the two groups were relatively similar, and by 6 weeks of therapy the glucosamine group reported less pain than the ibuprofen group. Thus, studies with inappropriately short lengths of follow-up should be viewed with caution.

It is also important to ensure that the study accurately measures what it claims to examine. For example, a study may clam to measure how often sports physicians discuss hormone replacement therapy with oligomenorrheic athletes. But, how did the study measure this frequency? If the investigators reviewed patient records, then the study assumes that medical records are 100% accurate. If the investigators sat in on patient visits, the presence of an outsider may have influenced this potentially confidential discussion. The issue of how investigators choose to measure human experience leads into the topic of identifying and minimizing bias.

IV. Was Bias Addressed and Minimized?
Bias is almost unavoidable in developing research methods and the interpretation of the results. A comprehensive discussion of bias is too lengthy for inclusion here, but a few of the more obvious questions of bias include:

- Who is funding the research?
- Did the subject recruitment process preferentially select or discriminate against any particular group of patients?
- Are the study and placebo groups randomly assigned and statistically similar?
- Are outcomes assessed by blinded professionals?

Good research will include a discussion of potential biases or perspectives and what efforts were undertaken to minimize these.[56] Beware of the study that purports to be completely free of bias.

V. Does the Study Have a Statistical Chance?
A discussion of sample size, effect size, and statistical power is a mark of a well-done trial. The number of patients needed in a trial depends on how large an effect the investigators expect their intervention to cause. Interventions that cause small effects may require unrealistic numbers of patients to prove their theoretical utility. Worse yet, these small effects may not be clinically significant. For example, a study may claim and prove that meditation and prayer lowered systolic blood pressure by 3 points; however, this small biologic effect is likely to be clinically negligible. Further, if a measure of clinical importance were to be attempted, the number of subjects needed in such a trial would be nearly impossible to fathom. The statistician's advice is still sage, that a trial should be "big enough to have a chance of detecting … a worthwhile effect if it exists, and … be reasonably sure that no benefit exists if it is not found in the trial."[57] While the sports physician may (or may not) entirely understand the statistical theory behind the interplay of sample size, effect size, and statistical power, research that does not contain a discussion of these vital issues must be interpreted with caution.

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31 CASTING OF SPORTS INJURIES

Anthony J. Campagna

THE TREATMENT OF CHOICE

A fundamental change in thinking relating to the principles of immobilization has taken place over the last 150 years. Where initially motion was regarded as dangerous and to be avoided by all means, the patient is now encouraged to be a participant in early motion activities under the guidance of the treating physician and support team. The concept of functional fracture treatment has led to the development of modern materials that offer increased options to this end. Because the choice of treatment considers risk, patient function, outcome, and economics, the ultimate decision to operate or treat an injury in a closed way should be evidence based.

CHOOSING THE MATERIAL

Understanding the immobilization requirements dictated by the injury will determine the material of choice and the design of the cast. The clinician's familiarity and capabilities with the material chosen will ultimately determine the quality and functionality of the finished cast. For the last 20 years, water-activated synthetic casting materials have led the way in functional casting because they offer early, lightweight strength and long-term durability. Having a choice of rigid or semi-rigid synthetic casting tapes, or the option of combining the two materials, the clinician can truly design a cast that accomplishes functional stabilization.

APPLICATION CONSIDERATIONS

To achieve its function, the finished cast must have a glove-like fit and be of appropriate dimensions to adequately immobilize the injury. The cast should exactly replicate the arm or leg in such a way that the cast and the extremity function as one unit. The extent of the cast should not go beyond what is needed to protect the injury, allowing full mobility distal and proximal to the ends of the cast as the injury permits. Before beginning the application, the clinician should have an idea of what the finished cast should look like so that each step in the process has a specific purpose toward accomplishing the final result. In order for this to happen, the applier must understand and be able to control the materials being used. Questions should be directed to the casting tape manufacturer relating to the proper handling of the materials being used.

DANGERS AND COMPLICATIONS

The unyielding nature of rigid casting materials leads to two potential dangers: *pressure* and *constriction*. To avoid complications resulting from pressure, special attention must be paid to bony prominences and superficial peripheral nerves by applying extra padding to these areas. Care must be taken when molding the cast to prevent indention of the material and creation of a pressure point. Constriction can be caused from without

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Figure 31.1

Figure 31.2

Figure 31.3

as a result of excessive tension being applied to the casting material during application. Constriction from within can result from uncontrolled swelling of the extremity after the cast is applied. If a cast is applied during the acute phase of treatment, the RICE (rest, ice, compression, elevation) methodology should be implemented to help control swelling. While the cast provides rest and compression to the injury site, the patient must be assigned the responsibility of applying ice and maintaining elevation. Understanding and reacting to warning signs either communicated by the patient or observed personally are critical in the prevention of potential complications. The four main warning signs are: *pain* (excessive or pinpoint), *pallor* (pulseless), *poikilothermia* (cool), and *paresthesia/paralysis*. In the presence of any of these warning signs, the clinician should make an early determination if intervention (splitting or removing the cast) is necessary.

APPLICATION PROCEDURE FOR A SHORT ARM CAST

- Before beginning any casting procedure, all of the supplies that will be required should be collected and brought to the application site. The supplies should also include a pair of bandage scissors (Figure 31.1).
- Choose the appropriate width of stockinet to maintain a glove-like fit and then cut the length to achieve the ultimate dimensions. Allow for extra stockinet proximally so it can be folded back on itself to provide a double layer for added comfort (Figure 31.2).
- Apply a double layer of stockinet to the thumb to fold back over the fiberglass before the final wrap is completed (Figure 31.3).
- The cast padding should extend from the distal palmar crease to within two fingers of the crease of the elbow. Enough tension should be applied to maintain a glove-like fit. The cast padding should overlap by one half while proceeding up the arm to create a minimum of two layers of padding. Extra padding should be applied over the ulnar styloid (Figure 31.4).
- Open the roll of synthetic casting tape, leaving the empty foil pouch on the counter. Begin wrapping at the wrist *without* exposing the casting tape to water (Figure 31.5).
- Before passing through the web-space, cut the casting tape to within approximately one-half inch of the distal border of the tape. The cut edges can be folded under while proceeding through the web-space with the casting tape (Figure 31.6).

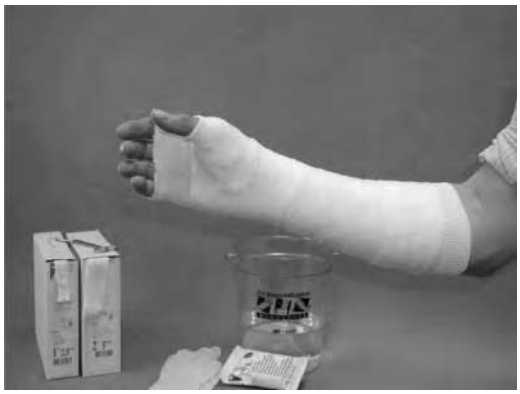


Figure 31.4

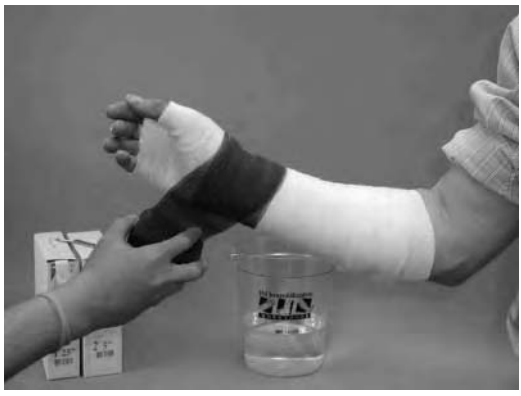


Figure 31.5

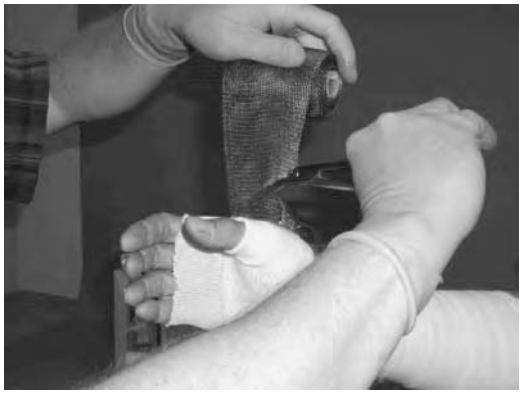


Figure 31.6

- Wrap a second time around the hand and through the web-space, cutting as before (Figure 31.7). Proceed wrapping up the arm, overlapping the casting tape by one half. The casting tape should be applied without tension because the cast padding has established the fit (Figure 31.8).
- When the proximal border of the cast padding is reached, cut off the remaining roll of casting tape and place it on the foil pouch (Figure 31.9).
- Roll back the stockinet at the distal and proximal ends and around the thumb (Figure 31.10).
- Dip the remaining casting tape in water and begin wrapping from the proximal end, covering the edge of the rolled back stockinet. Continue wrapping to the wrist and complete one final pass through the web-space using the same cut technique as before. Make sure to cover the edges of stockinet around the hand and thumb. Dip gloves in water and proceed to smooth and then mold the cast. Continue the molding process until the resin has

Figure 31.7

Figure 31.8

Figure 31.9

Figure 31.10

Figure 31.11

reached its set time (resin is sufficiently rigid to retain the desired shape when hands are removed, approximately 3 minutes) (Figure 30-11).

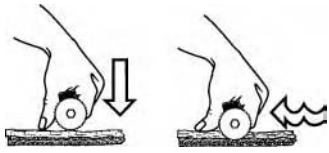


Figure 31.12

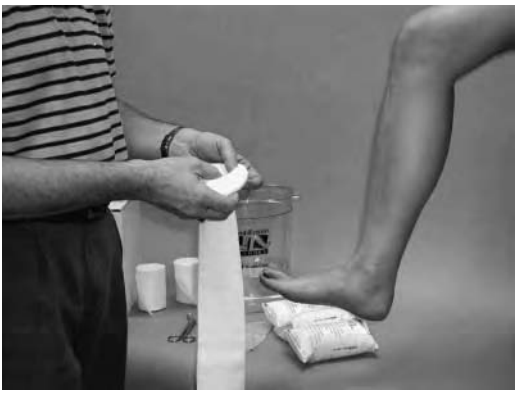
Figure 31.13

CAST REMOVAL

Proper cast-saw technique is essential for the safe removal of any cast. The saw should be grasped firmly with contact being made between the surface of the cast and thumb of the hand holding the cast saw. Contact between the thumb and the cast helps control the depth of penetration of the cast-saw blade. The blade should be kept perpendicular to the surface of the cast when cutting. The cast-saw blade should be pushed straight down into the cast until the complete thickness of the cast has been penetrated. Remember, the thumb controls the depth of penetration. This step is repeated, making a series of connecting cuts up or down the cast from one end to the other (Figure 31.12). After the cast has been bivalved, the stockinet should be cut both proximally and distally so the anterior shell may be lifted off.

APPLICATION PROCEDURE FOR A SHORT LEG CAST

- All of the supplies that will be required should be collected and brought to the application site (Figure 31.13).
- Cut the stockinet to length and roll it into a donut before applying. Remember to allow extra length when cutting the stockinet to provide a double layer at the proximal end for comfort (Figure 31.14).



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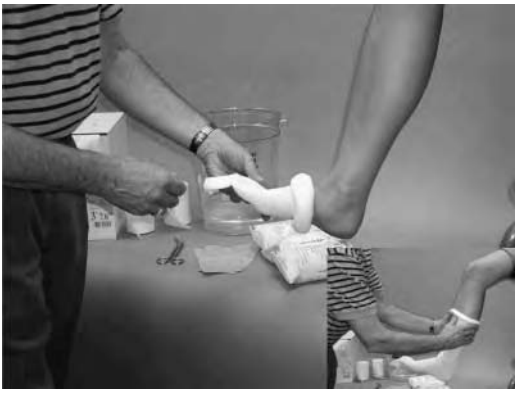
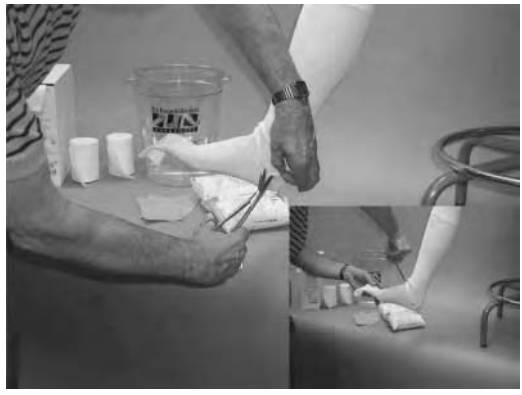


Figure 31.15

- Leaving approximately 2 inches of stockinet extending beyond the toes, begin unrolling the stockinet over the foot and up the leg (Figure 31.15).
- Roll back the extra stockinet so two layers can be folded back over the proximal end of the casting tape (Figure 31.16).
- To remove the wrinkles at the bend of the ankle, make an opening cut in the stockinet at the heel and continue the cut over the ankle to the heel on the other side (Figure 31.17).
- Lift the distal portion of the cut stockinet over the proximal portion to eliminate the wrinkles (Figure 31.18).
- Begin wrapping the cast padding distally, making sure that the distal border of the padding extends beyond the metatarsal heads on the plantar surface of the foot. Continue wrapping around the foot and ankle and proceed up the leg, overlapping the padding on itself by one half (Figure 31.19).
- The proximal border of the padding should extend to the tibial tuberosity (Figure 31.20).

Figure 31.16



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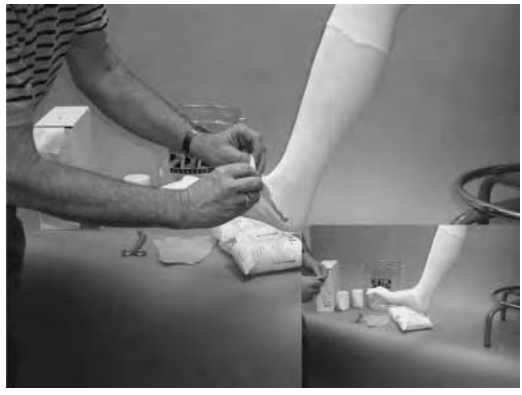

Figure 31.18

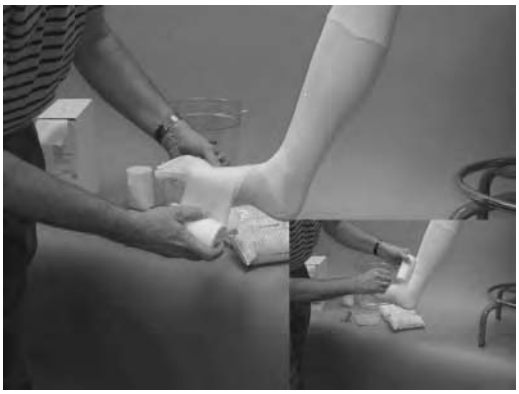

Figure 31.19

Figure 31.22

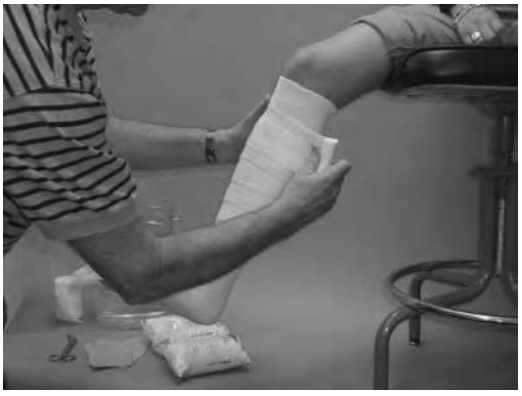

Figure 31.20

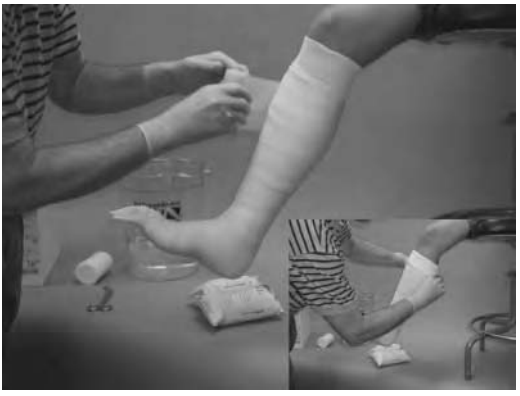

Figure 31.23

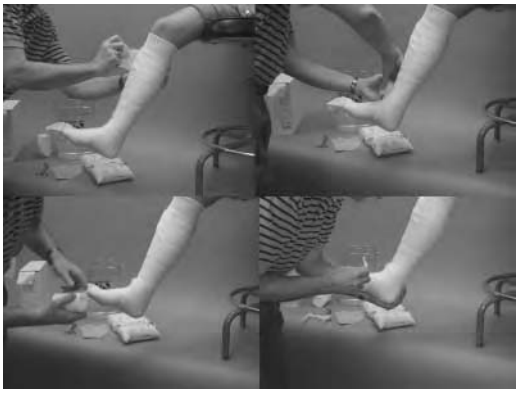

Figure 31.21

- Extra padding should be applied over bony prominences and superficial peripheral nerves and tendons, such as the perineal nerve (Figure 31.21, upper left) and medial and lateral malleolus (Figure 31.21, upper right). Additional padding should be added around the medial and lateral malleolus and the Achilles tendon (Figure 31.21, lower left) and the heel (Figure 31.21, lower right).
- Without exposing the casting tape to water, begin wrapping the first roll distally starting at the distal border of the cast padding. Continue wrapping around the foot and ankle and up the leg, overlapping the casting tape on itself by one half (Figure 31.22).
- When the proximal border of the casting padding is reached, start rolling the casting tape back down the leg to finish the roll. Fold the double thickness of stockinet over the proximal border of the casting tape (Figure 31.23).
- Begin the second roll without exposure to water, starting at the distal-most border

Figure 31.24

Figure 31.25

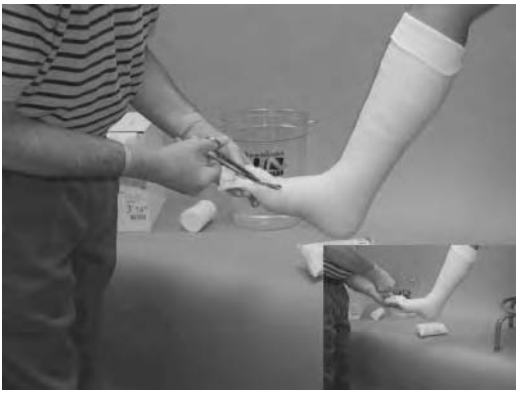

Figure 31.26

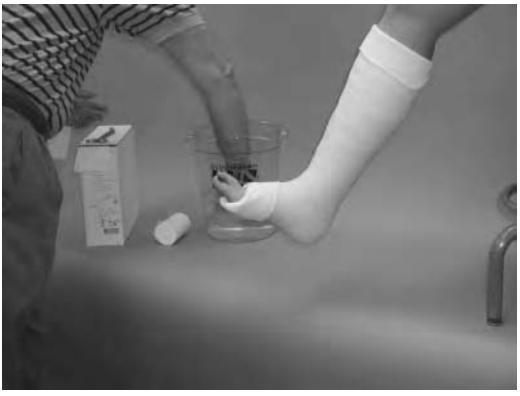

Figure 31.27

of the foot. After one wrap, fan-fold the casting tape to create four layers, and wrap the tape completely around the metatarsal heads, keeping the roll sideways to maintain the four layers. When the dorsum of the foot is reached, rotate the roll so that it returns to a single layer and continue to wrap around the foot and ankle (Figure 31.24).

- When wrapping around the foot and ankle, fold the material back on itself under the heel one time, capturing it with the casting tape while continuing to wrap. This provides reinforcement for heel strike (Figure 31.25).
- After the reinforcement roll is completed, make a cut on each side of the distal dorsal portion of the cast. These cuts should be through the cast padding and casting tape, leaving the stockinet in tact. The cuts should be long enough to allow the toes freedom to extend during the toe-off portion of the gait. Cut away the excess material between the two cuts (Figure 31.26).
- Fold back the stockinet over the distal end of the cast. Open the third and final roll of casting tape and immerse it in room-temperature water, giving three firm squeezes while the roll is submerged. Ring out the excess water (Figure 31.27).
- Begin wrapping proximally, covering the stockinet with the first wrap, and then proceed down the leg. After capturing the distal stockinet, finish the roll by wrapping around the foot and ankle and back up the leg (Figure 31.28).
- Wet your gloves and rub the entire cast to give it a smooth finish. Because the first two rolls were not exposed to water, minor positioning adjustments can be made at this time. Continue to mold the

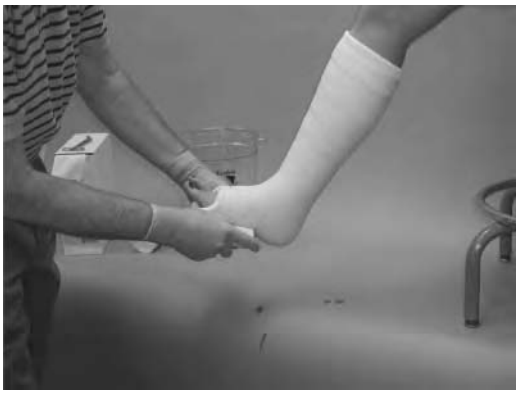

Figure 31.28

Figure 31.29

cast until the resin is set (approximately 3 minutes) (Figure 31.29).

- The cast is ready for weight bearing in 20 minutes. A cast shoe must be worn during *all* weight-bearing activities (Figure 31.30).

REMOVAL OF A SHORT LEG CAST

Use the same cast-saw technique as described in the cast removal section above. To simplify the removal of a short leg cast, or any cast, start by bivalving the cast on opposite sides of the extremity as would normally be done. Cut the stockinet proximally on each side where the casting tape has been bivalved. Free the anterior shell of the bivalved cast by cutting from bivalve to bivalve the stockinet that is folded over the proximal end of the cast. Now lift the anterior shell, separating

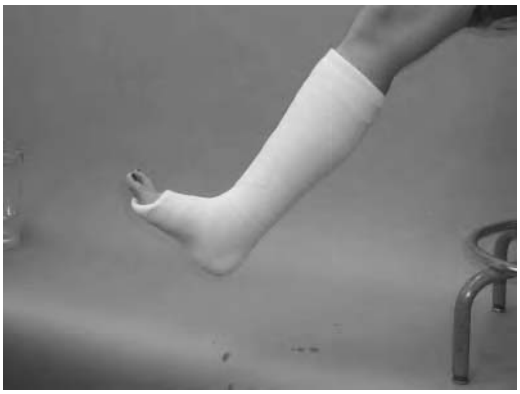

Figure 31.30

it from the cast padding. Cut the distal attachment of the stockinet to completely free the anterior shell from the rest of the cast. The remaining cast padding and stockinet can now be cut to remove the posterior shell.

SUMMARY

Understanding the immobilization requirements of the injury is critical to determining the ultimate design of the cast and the best materials to use. The design gives purpose to each step in the process of constructing the cast. The clinician's understanding of and capabilities with the materials being used will greatly impact the finished results. Casting is an art that can only be mastered by doing. Clinicians must be their own best critics, learning and improving with each cast applied.

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32 THE USE OF ORTHOSES FOR ATHLETES

Kurt Jepson

INTRODUCTION

The sports physician must have a solid understanding of normal joint arthrokinematics, injury pathoetiology, and basic orthotic principles prior to prescribing orthoses for patients. Inherent joint stability is the result of various anatomic inputs. The presence of adequate ligamentous systems and their functional integrity, intact proprioceptive pathways, and architectural congruency may be classified as *static* joint stabilizers. *Dynamic* stabilizing components include contractile forces, neuromuscular loops, and compressive loads. An ideal orthosis compensates for as many of these factors as possible in the presence of deficiencies. A competent diagnosis and comprehensive treatment plan including re-education and strengthening are of primary importance, rather than mere orthosis application. The characteristics of a good orthotic are presented in Table 32.1. The literature indicates that cost effectiveness and documentation of efficiency may be the most difficult to obtain.[1–3]

FUNCTIONAL KNEE ORTHOSES

Isolated and combined rotary knee instabilities have been at the forefront of sports medicine research for some time. Since their inception in the 1960s, derotation braces have undergone numerous design changes, keeping pace with biomechanical research and material technology. They are classified by the American Academy of Orthopedic Surgeons as functional knee braces and by definition are designed to provide stability for unstable knees.[4]

Custom brace manufacturers fabricate an orthosis using a positive model of the knee retrieved by means of a coordinate and/or tracing system done by the practitioner (Figure 32.1). Various off-the-shelf models also seem to function on a similar level and are more cost friendly given current reimbursement climates (Figure 32.2).[2,5–7] Custom functional braces may range from $500 to $600 wholesale, while off-the-shelf models are in the $150 to $375 price range.

Numerous structural and biomechanical factors should be considered prior to selecting a functional knee brace. Cage design orthoses do not have viscoelastic properties and therefore restrict all knee motions, not just pathomechanical extremes. Some designs may interfere with normal arthrokinematics more than others.[8,9] Some evidence indicates that bracing may compromise hamstring function, which may be particularly disadvantageous in anterior cruciate ligament compromised patients.[9–11]

Prior to prescribing a derotation brace, practitioners should be comfortable with their examination skills in order to identify and classify knee instability. A review of the classification of knee joint instabilities is suggested.[12,13]

The ability of a brace to control anteromedial rotary instability (AMRI) and anterolateral rotary instability (ALRI) may be more vital to stability than controlling frontal and sagittal plane motions.

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Figure 32.1

Figure 32.2

TABLE 32.1
Characteristics of a Good Orthosis

Increases stability of unstable joints
Limits pathomechanical input to adjacent joint structures that could pose a risk to neighboring joints
Does not interfere with normal joint function
Does not pose a risk to others
Can be adapted to various somatotypes
Is cost effective
Has documented efficacy

The ultimate goal of bracing is to negate translational and shear forces that contribute to instability or degeneration of secondary joint structures. This is best addressed by matching a patient's specific instability with a device that will help control unwanted motion. In addition, the intended use of an orthosis in sport will also direct selection. Parameters include relative size and strength of the athlete, environmental factors, contact or non-contact sport use, and forces to be encountered, extrinsically or produced intrinsically.

Hinges, the frame, uprights, and suspension straps comprise the major components of a functional knee brace. Braces typically have range of motion limiters and various contour accommodating pads. The hinge is essential to brace function. Various designs are currently used. Uniaxial (single axis) hinges allow motion in the sagittal plane only. Normal tibiofemoral rotation may be restricted in these braces. Also, the stationary axial position of the brace resists a posteriorly shifting joint axis during flexion. The result is often *pistoning* of the orthosis and slippage distally. Biaxial (polycentral) hinge designs attempt to accommodate the multiaxial components of knee motion, but few mimic simultaneous anteroposterior (AP) excursion and transverse plane rotation. A unilateral upright bar and hinge assembly may best address this multiplanar requirement but in the process sacrifices the mediolateral stability offered by cage designs.

Suspension systems serve two purposes. They hold the orthosis in its proper position and provide the means for force–counterforce input. Suspension systems and related straps should allow for soft-tissue volume changes due to muscular contraction, perfusion, edema/effusion, and eventually hypertrophy. Materials must have sufficient rigidity to oppose translatory tendencies of the lower leg. Cage, shell, and cuff contours should match those of the individual to maximize control, disperse externally applied forces away from the knee joint, and limit pistoning. Accomplishing this will be more challenging with an off-the-shelf brace.

The prescribing practitioner should scrutinize how a manufacturer attempts to control excessive rotation about the knee orthotically. Medial or

lateral tibial plateau anterior translation is the precursor to pathomechanical motion and is best countered by proximal tibial cage components that follow bony contours precisely. AMRI and ALRI forces can then be opposed via the rigidity of the cage. High-density foams provide a comfortable interface between the anterior tibia and the brace frame. Braces that rely on derotation straps should be avoided as they lack sufficient rigidity to counter AMRI and ALRI forces. Neoprene undersleeves provide an additional unwanted interface between the brace and osseous components and should be utilized only when cutaneous irritation becomes an issue. Similarly, braces should not be worn over clothing.

Most derotation devices incorporate variable extension stops in their hinges. Reducing extension to –10°, thereby limiting the "screw home" mechanism of the knee, is suggested as an adjunct to rotational control. Selection of the amount of extension restriction should be based on specific motion requirements of the sport (or position), as well as any special prophylactic needs (e.g., posterior corner compromise). Some form of posterior tibial restraint should be included to prevent backing out of the brace.

Brace components such as condylar pads must maximize brace–knee congruency as they have supportive, suspensory, and force dispersion responsibilities. High-density foam or pneumatic designs are prevalent. Materials should be comfortable and resilient to deformation. Options such as patella cups and ski boot attachments should be prudently prescribed based on biomechanical design and patient specificity.

Most patients who utilize these orthoses report added confidence during activity and fewer episodes of functional instability.[14,15] The reason for these comments is unclear, as few studies demonstrate translational control, especially at high load. Forces exceeding 120 N, common during dynamic activities, are minimally controlled.[16–18] These orthoses do exhibit some degree of mediolateral protection.[1] Braces with a hinge, post, and shell design appear to limit anterior tibial translation more efficiently than other types, but, again, as forces increase the ability of a brace to control unwanted motion decreases.

Organizational mandates may require covering the brace with a padded sleeve to protect opposing players in a contact sport situation. Sports covers are readily available.

Despite a lack of documented efficiency, functional derotation braces continue to be recommended and used.[20] Anecdotal success may be attributable to proprioceptive enhancement, altered muscle firing patterns, and/or psychological factors.[15,21] A study by Risberg et al.[22] did demonstrate enhanced closed kinetic chain testing abilities 6 months after ACL reconstruction in a braced vs. non-braced population. It is interesting to note that the same study also demonstrated detrimental effects from long-term brace use.

Figure 32.3

Braces may offer beneficial initial stabilization to the post-reconstructed knee when encountered forces are relatively low. Benefits of long-term use are unclear. Brace form and functionality are extremely important to patient compliance and potential prophylaxis.

PROPHYLACTIC KNEE GUARDS

Lateral knee orthoses (classified by the American Academy of Orthopedic Surgeons as "prophylactic knee braces") were developed to prevent or reduce the severity of medial knee injuries due to valgus overload.[21] In theory, lateral knee guards supplement medial knee stiffness and elevate failure parameters. Prophylactic knee braces consist of a single upright positioned on the lateral aspect of the knee (Figure 32.3). Suspension occurs via hook and loop strapping and/or taping. Adequate lateral condylar clearance is achieved via angulation away from the joint line superiorly and inferiorly, creating a "buffer zone" that eliminates bruising and localization of forces associated with lateral blows. Paulos et al.[24] emphasized the importance of non-contact between the brace and the lateral joint line via surrogate knee studies.

They also related upright rigidity to effectiveness of injury protection.[25] These unilateral upright braces commonly utilize a polycentric sagittal plane hinge. Transverse plane motions are not controlled. Exteriors are padded to lessen threat to opposing players. When applied properly, they do not interfere with athletic function.[26] A small metabolic cost may be incurred.[27] Lateral impact load distribution occurs through soft tissue compression proximal and distal to the joint line and flex of the upright. Sufficient tissue compression on those athletes with significant adipose tissue may not result prior to critical medial opening under valgus load. Such morphotypes may derive little benefit from lateral bracing.[28]

Unfortunately the literature fails to provide a uniform consensus on the efficiency of lateral knee guards.[29] Reduction of the rate and severity of injury to collateral ligaments and other structures has been documented in football players using lateral knee braces.[28,30] The requirement for surgical intervention due to extensive injury in those athletes also decreased. In a closely controlled study at West Point, Sitler et al.[31] noted lower injury rates, decreased severity, and no increased incidence of ankle injuries. Their data indicate defensive players may benefit more from brace use than those playing offensive positions.

Other research, however, has concluded that lateral knee bracing offers no significant protection away from the laboratory setting and may indeed predispose the athlete to injury.[32,33] The National Sports Injury Surveillance System (NSISS) and Athletic Injury Monitoring System (AIMS) have concluded that the incidence and severity of injury is higher for braced individuals. The concept of medial collateral and anterior cruciate ligament *preloading* has been introduced.[34]

In those individuals with significant medial or global laxity a cage orthosis may better address their stability requirements.[19] Until well-designed prospective data become available, team physicians, sports physical therapists, and athletic trainers will need to balance the pros and cons of prophylactic knee bracing. If utilized, braces should be well constructed, be affordable, fit properly, and never be considered a replacement for physical conditioning and proper technique.

REHABILITATION KNEE BRACES

Rehabilitation braces address protective goals during the healing process while allowing for early controlled motion and strength activities. The American Academy of Orthopedic Surgeons describes rehabilitation braces as "those designed to allow protected motion of injured knees treated operatively or nonoperatively." Most rehabilitation braces follow simple design principles (Figure 32.4). Bilateral uprights, generally composed of alloys or composites, parallel limb lengths to maximize medial–lateral and anterior–posterior stability. Due to the long lever arms involved, external forces are dissipated over large surface areas, thereby limiting focal loads. Hinge systems allow motion in the sagittal plane and incorporate flexion and extension stops. Transverse plane motion of the knee is difficult to control with these othoses.[35] The upright and hinge components are affixed to cloth or foam sleeves, which in turn are applied to the limb with hook and loop strapping. Some manufacturers utilize rigid polymer shell suspension systems. A surrogate knee study by Cawley et al.[36] demonstrated the importance of integrated strap and bar assemblies, as well as numerous straps, in regard to controlling unwanted knee motions. They also noted that upright contact with the limb was very important to the "stiffness" component and translation control. Sizing, therefore, is important despite off-the-shelf choices of small, medium, and large.

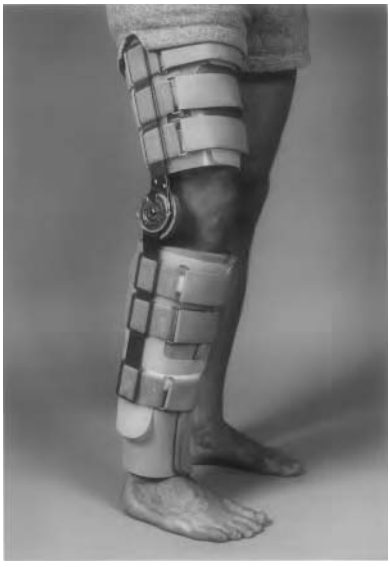


Figure 32.4

The practitioner may find occasions where a functional knee orthoses (see Figure 32.2) will provide adequate protection after injury. Initial utilization of this type of appliance negates the expense of a rehabilitation brace being replaced by a sports brace. Many functional knee orthoses also enable the practitioner to limit range of motion (ROM).

A rehabilitation brace use is indicated for conservative treatment of ligament sprains and postoperative management of reparative or reconstructive procedures. The selection of a rehabilitation brace over traditional casting and splinting is justified for numerous reasons. Patient compliance is high due to the comfort level and ease of application. Hygiene is enhanced. They are cost effective, especially when compared to serial casting. Wounds are easily accessible for treatment and monitoring. Their cumbersome dimensions limit excessive ambulation and send a message to others that the patient requires special attention and avoidance. Finally, the practitioner's ability to adjust range of motion based on the stage of physiologic healing helps reduce adverse changes within the affected limb. Constant passive motion machines and electrical muscle or bone stimulators are quite compatible with the braces. Brandsson et al.[37] noted fewer postoperative problems in a sample of braced patients vs. a non-braced group.

Rehabilitation braces allow the practitioner to keep pace with ever-changing concepts concerning the extent and duration of immobilization following an injury. Adaptability, protective capability, and compatibility with current rehabilitation protocols support their utilization.

PATELLOFEMORAL ORTHOSES

Due to the forces of compression and mediolateral translation created during functional patellofemoral joint movement, extrinsic control is extremely difficult. Various attempts at identifying the ability of bracing and taping to direct patellar movement have produced conflicting results.[38–40] Despite the absence of identifiable mechanisms for symptom alleviation in patellar dysfunction patients, practitioners continue to use bracing with clinical success. Reversal of aberrant firing patterns in patellofemoral dysfunction (PFD) patients identified by Voight and Weider,[41] proprioceptive enhancement and synovial redirection via bracing have been proposed.[42] Most braces are utilized to counter lateral tracking. Improved patellar tracking enhances kinematic efficiency as well as nutritional stimulus to the hyaline cartilage.[43,44]

Patellofemoral orthoses are manufactured in various designs (Figure 32.5). Synthetic sleeves provide the base for secondary features such as buttresses or tensioning straps, while simultaneously acting as a suspension system. Closed or open patella designs are available and should be chosen based on such factors as relative patellar

Figure 32.5

mobility, peripatellar edema, retropatellar pain, and crepitus.

Buttresses are usually composed of high-density foams or felt. Their placement selection is determined via the clinicians' evaluation of aberrant patellar posture and goals to redirect the patella to a more neutral position. Medial, lateral, superior, inferior, or any combination of designs may be required to best address an individual's control needs. Some buttress designs incorporate dynamic pulling mechanisms,[21] while others combine taping and directing straps.[46] When utilized in combination with thorough lesion-specific rehabilitation interventions, patellar bracing enhances the clinician's ability to successfully manage patellofemoral dysfunction.[47]

Simple patella alta may only require an infrapatellar strap or band to improve trochlear congruency and stability. These devices are also used successfully in patients with infrapatellar tendinitis ("jumper's knee") and Osgood–Schlatter disease to lessen pain and improve function. Force dispersion within the distal quadriceps mechanism is a likely explanation for their effectiveness.

COUNTERFORCE FOREARM ORTHOSES

A counterforce armband is an orthosis consisting of a high-density foam, felt, gel, or air-filled bladder affixed to nonelastic strapping with hook and

Figure 32.6

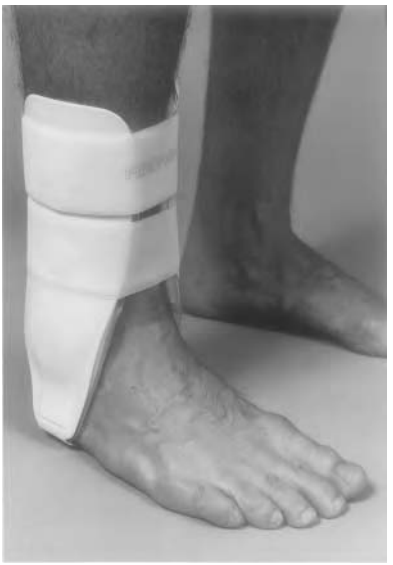


Figure 32.7

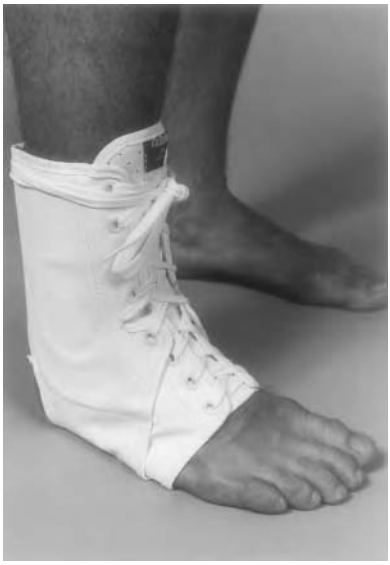
loop closure. For patients involved with lateral epicondylitis, the orthosis is positioned so the pad compresses the dorsal radial musculature 1 to 2 inches distal to the common extensor origin (Figure 32.6). In theory, the band reduces proximal extensor load by dispersing forces traveling through the dorsal musculature. Prior investigators hypothesized that extensor origin forces were lessened by means of muscle fiber compression and alteration of length tension relationships.[48,49] Wadsworth et al.[50] however, demonstrated that use of a counterforce armband in subjects with lateral epicondylitis actually increased wrist extensor and grip strength. Clinical effectiveness may be attributable to circumferential force dispersion prior to its concentration at the lateral epicondyle, rather than via extensor inhibition. A recent study by Walther and Kirschner[51] demonstrated a significant reduction in acceleration amplitudes and vibration within the extensor musculature using braces with pads placed at the proximal forearm. Counterforce bracing is a useful aid in the management of lateral epicondylitis. In the acute stage, they disperse pathomechanical forces and remind the patient that full functional use of the affected extremity is not advantageous. Long-term use of the brace may aid prophylaxis by reducing excessive tensile load at the lesion site.

FUNCTIONAL ANKLE ORTHOSES

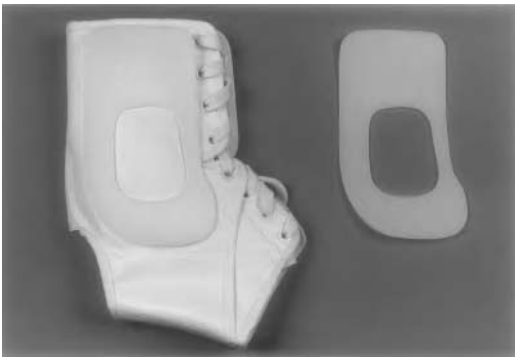
Functional orthoses are a valuable adjunct to the management of ankle injuries. They allow a protected return to rehabilitative activities and possibly an earlier return to sport. They provide superior support, are convenient, and cost less than tape. Ankle orthoses should meet standard orthotic goals (see Table 32.1). Braces follow two designs: rigid medial and lateral uprights or open-toed sock designs that incorporate front lacing or hook and loop straps. Some designs combine these concepts. Materials include thermoplastics, air bladders (Figure 32.7), nylon or canvas fabrics, and neoprene. Some designs include medial and lateral pockets for the insertion of semi-rigid uprights (Figure 32.8). Improved rigidity may help combat excessive inversion–eversion forces. Other resistance to potential pathomechanical forces may be provided by heel-lock and figure-eight straps (Figure 32.9).

In addition to stabilization, ankle orthoses have tissue-compression capabilities. Because these are not custom braces, adjustments for changes in joint contours are easily made. Effusion control is maximized as the orthosis is tightened during application. Pneumatic or gel-filled bladders that line some orthoses are said to milk away edema by means of internal pressure pulsation during ambulatory activities.[52] Kinesthetic activity may also be enhanced through tissue approximation and mechanoreceptor stimulation.[53,54]

Unlike prophylactic or post-injury taping, ankle braces can be repetitively applied by the patient, thus lowering cost.[55,56] Proper fitting of an ankle orthosis is vital to its performance. Not only should the brace fit snugly circumferentially about the ankle complex, but it also should attempt to approximate the talocrural and subtalar articula-



A



B

Figure 32.8

tions. Vertical loading of bony contours, in addition to mediolateral rigidity, enhances stabilizing forces.

Range of motion restriction varies based on the design of the brace. Braces that lace anteriorly tend to restrict plantar flexion and dorsiflexion more than stirrup designs. Most seem to limit inversion significantly.[57,58] Significant variance in muscular activity may also occur when different orthoses are worn, although the clinical relevance is currently unknown.[59–61]

Environmental factors (e.g., moisture, heat) and material fatigue tend to have an adverse effect on the supportive capabilities of tape. Ankle braces, tightened periodically, retain their supportive characteristics much better than tape.[62,63]

In conclusion, functional ankle orthoses appear to fill a prophylactic and protective niche.

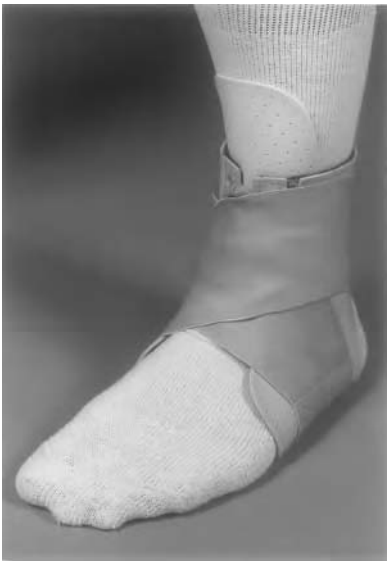


Figure 32.9

When compared to traditional taping, they offer superior supportive input to the ankle complex, particularly in the later stages of exercise. Advantages of these braces include low cost, ease of application, resilience to environmental and structural stresses, and the relatively small chance of skin breakdown with repetitive use. When incorporated into early rehabilitation protocols, they may offer beneficial kinesthetic and tissue-compressive inputs. As with any orthosis, ankle braces are intended to supplement joint stability and should not take the place of conditioning or proper neuromuscular rehabilitation.

FOOT ORTHOTICS

Athletic non-traumatic injuries to the foot and ankle complex are often the result of training error, anatomical variants, and biomechanical variables.[64,65] Foot orthotics help compensate for anatomic and biomechanical variants to allow for more efficient, less stressful locomotion. An ideal orthotic device allows the foot to move unrestricted through normal ranges of motion while limiting unwanted compensatory movements in response to intrinsic structural flaws or dynamic insufficiencies. Their efficacy is well documented.[66–69]

The subtalar joint is the primary site of compensatory motion in response to proximal or distal pathomechanical variances. The practitioner must select a device that will allow this triplanar joint to move through a required range of motion while controlling excessive movement. The concept of subtalar neutral positioning and its role in kinetic

A

B

Figure 32.10

chain function has been debated for years.[70] An orthotic need not place the subtalar joint in its precise anatomic neutral position to be successful. Many patients respond well to adapted generic footbeds and/or shoe changes. Some problems require more precise control, and a truly custom appliance fabricated from a cast model is necessary (Figure 32.10). This author believes casting subtalar neutral in an open-chain configuration is most beneficial when prescribing a fully custom appliance.

Appliances are typically classified as flexible, semi-rigid, or rigid.[71] Corresponding materials include aliplast, cork and thermoplastics, or carbon graphite. Selection is predicated by patient body weight, intended use, diagnosis, and foot type. As a general reference, flexible planus feet respond best to more rigid appliances, with the converse being true for rigid cavus feet. Variability exists, however, and the inexperienced practitioner should not hesitate to utilize other qualified clinicians to obtain the most favorable result possible. This is particularly true if a custom device is to be utilized.

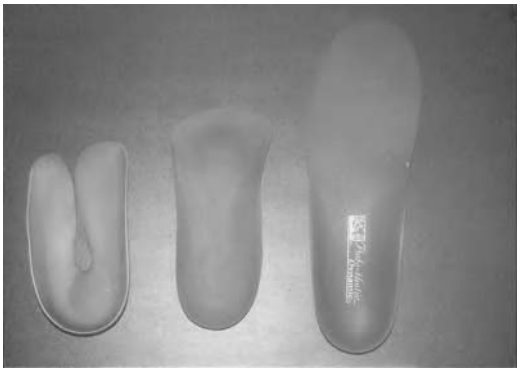


Figure 32.11

Several cost-effective devices are available for use. Some come with intrinsic posting (canting) typically for varus foot anomalies. Most off-the-shelf appliances require custom posting via extrinsic wedge application and feathering. Varus or medial posting is most common (Figure 32.11). Such devices limit pathomechanical pronation associated with plantar fasciitis, posterior tibial tendinitis, and patellofemoral dysfunction.

In some cases, efficient orthotic implementation can be as simple as placing a metatarsal pad in the forefoot of a Morton's neuroma or metarsalga patient. Patients with Seaver's disease or Achilles tendinitis often respond favorably to a varus heel wedge. All problems do not require a full appliance. Many simple devices are easily fabricated from felt, self-stick cellular foams, or cork. Prefabricated wedges and pads are cost effective, easily inventoried, and readily available (Figure 31.12).

Foot orthotics provide a useful adjunct for treatment of non-traumatic foot and ankle overuse injuries. They are not a panacea, however, and should be prescribed in conjunction with physical therapy, medications, activity modification, and shoe changes.

SUMMARY

Practitioners prescribing functional, rehabilitative, and prophylactic orthoses must have knowledge of normal arthrokinematics, injury pathomechanics, and orthotic principles. Patients should understand that the efficacy of orthotic appliances is

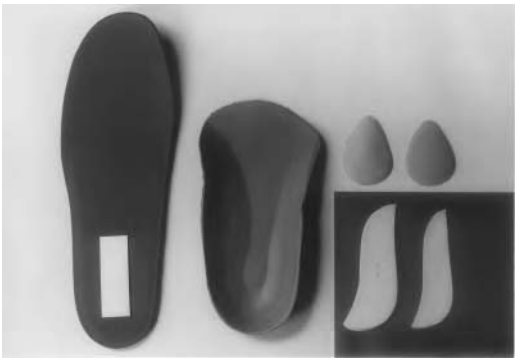


Figure 32.12

still under scientific scrutiny. Their use should never replace neuromuscular rehabilitation, conditioning, or proper playing techniques.

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33 TAPING PROCEDURES FOR SPORTS INJURY*

Robert M. Barney Poole and Jessica M. Poole

INTRODUCTION

Athletic taping and strapping is an art practiced daily by athletic trainers in the clinic, in the training room, and on the sidelines of athletic competition. Various types of athletic tapes and elastic wraps are skillfully applied to provide support for and prevention of sports injuries. This chapter provides the primary care physician with a procedural guide for taping of common sports injuries. Each method discussed is by no means the only way a particular injury may be taped or strapped, but the methods selected are those most commonly used by athletic trainers and are the easiest to implement. Illustrations with written instructions are provided to give even the most inexperienced practitioner a basic guideline for taping and strapping of sports injuries.

A few tips to consider when taping for sports:

1. Taping should be applied for short periods of time, such as during practice and games, or as support immediately following an injury. After 15 to 20 minutes of exercise, support declines by 20 to 50%.
2. Never tightly encircle soft tissue, as this could impair circulation.
3. When applying underwrap, cover all areas to be taped, leaving no gaps or bare spots.
4. Do not force the tape to go where it does not want to go; wrinkles that cause blisters may result.
5. Overlap tape by one-half width to minimize blistering.
6. Tape adherent and grease pads are helpful to improve comfort but are neither mandatory nor necessary.
7. Neatness counts.

TAPING PROCEDURE 1: FINGERS

Generally, fingers are taped secondary to trauma such as dislocation, fracture, or sprain (e.g., "jammed" finger). Taping should support the joint and allow the athlete to safely and comfortably participate in his or her sport.

* In the second edition, this chapter was authored by Robert M. Poole and Robin C. Gaines.

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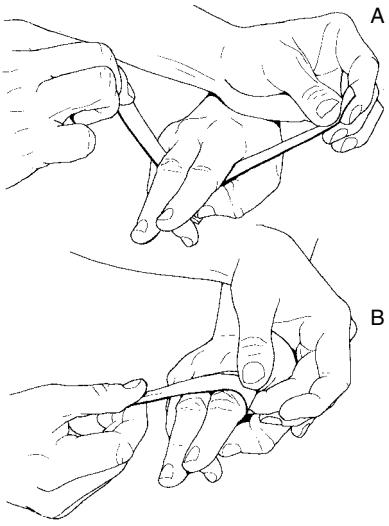

Figure 33.1A–B

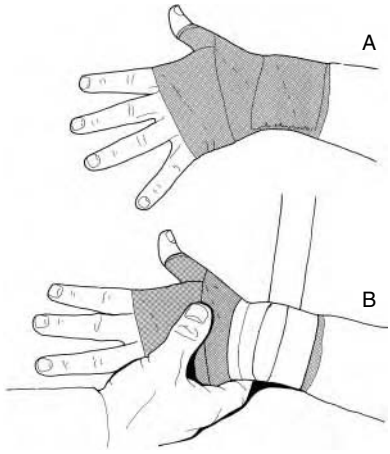

Figure 33.2A–B

Materials Needed

- White zinc oxide tape (1-inch) or elastic tape or a non-adhesive material such as Coban®
- Tape-adherent spray

Position

The athlete should sit comfortably on a table with arm extended and fingers spread, supporting the involved wrist with the other hand.

Procedure

1. Approximate the injured finger with an adjacent finger (Figure 33.1A). Fingers 1 and 2 or 3 and 4 should be paired as this provides the greatest support with the least amount of potential reinjury. Begin taping with a 1-inch strip of tape distal to the proximal interphalangeal joint (Figure 33.1B).
2. Finish by applying a 1-inch strip distal to the proximal interphalangeal joint. A piece of foam or felt may be placed between the fingers for comfort and to decrease mobility.

TAPING PROCEDURE 2: WRIST/THUMB

Because of the bony configuration of the wrist, it is important to tape the wrist in a neutral position, regardless of whether the injury is a flexion or extension injury. Taping the wrist in a neutral position provides support against hyperextension or hyperflexion and allows the athlete to continue to participate in sports activities without further injury to the wrist.

Materials Needed

- White zinc oxide athletic tape (1.5-inch)
- Underwrap

Position

The athlete should sit comfortably on a table with the arm extended, fingers spread, and wrist in neutral position, with the palm side of the hand down. The athlete should support the wrist and forearm with the uninvolved hand during taping.

Procedure

1. Apply a thin layer of tape-adherent spray to the surface to be taped. Prepare the wrist with underwrap as shown in Figure 33.2A, covering approximately a 4- to 6-inch area from the carpal bones, extending proximally toward the elbow.
2. As shown in Figure 33.2B, apply five to six strips of tape at an angle, beginning distally and working proximally, overlapping by half the width of the tape each time and tearing after each strip is applied. Tearing the tape after each strip is applied guards against wrapping the tape too tightly and cutting off circulation to the fingers.
3. A thumb spica may be added to the wrist taping to provide support for the thumb. A 1-inch strip of white athletic

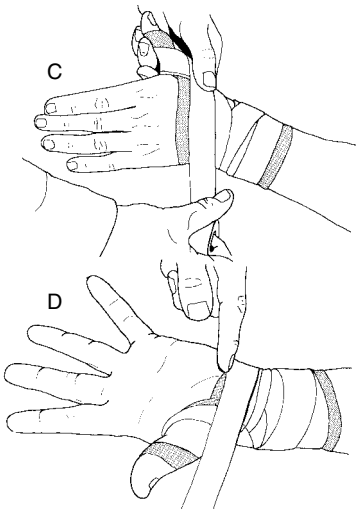

Figure 33.2C–D

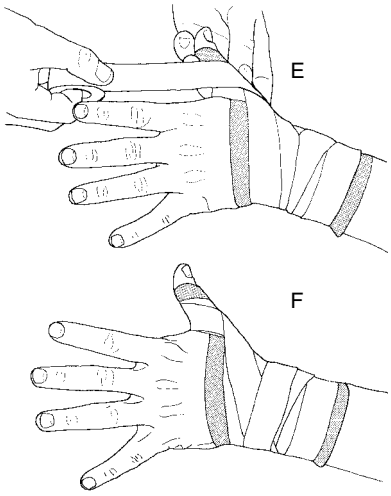

Figure 33.2E–F

tape is placed on the dorsal portion of the hand (Figure 33.2C) and wrapped over the first interphalangeal joint of the thumb, continuing in a figure-eight pattern, back around to the dorsal side of the thumb and hand (Figure 33.2D). Repeat this procedure two or three times, overlapping the tape by one-half width (Figure 33.2E. The independent thumb spica strips are then anchored on the wrist. The finished wrist and thumb spica taping is shown in Figure 33.2F.

TAPING PROCEDURE 3: ELBOW

The elbow is taped to prevent hyperextension in cases such as postdislocation or other postextension injury to the elbow and for myositis ossificans of the biceps secondary to repeated trauma.

Materials Needed

- Elastic tape (2-inch)
- White zinc oxide athletic tape (1.5-inch)
- Tape-adherent spray
- Underwrap

Position

The athlete should sit comfortably, facing the table with hand on the table and the affected elbow bent approximately 30°.

Procedure

1. Apply a thin layer of tape-adherent spray to the surface to be taped. Apply underwrap, distal to proximal, using oblique overlapping strips. Apply anchor strips 3 to 4 inches proximal and distal to the joint line, using 1.5-inch athletic tape (Figure 33.3A).
2. Prepare an extension-limiting splint by overlapping 6 pieces of 1.5-inch athletic tape in an X pattern (Figure 33.3B).
3. Apply the extension-limitation splint to the elbow with the strips crossing the antecubital fossa. The elbow should be limited in extension 20 to 30° (Figure 33.3C).
4. Tape the splint to the upper and lower arm with 2-inch elastic tape applied obliquely to prevent wrinkles; anchor it in place with 1.5-inch white athletic tape (Figure 33.3D).

TAPING PROCEDURE 4: SHOULDER WRAP

One of the easiest ways to provide support to an injured shoulder is to use a 6-inch elastic wrap. An athlete with a mild acromioclavicular (AC) separation or strain/contusion to the shoulder may be able to participate in sport more comfortably while wearing this type of wrap. In the case of a chronically subluxed shoulder, a strap that limits abduction may be applied using a modification of this method.

Materials Needed

Two 6-inch elastic wraps
White zinc oxide athletic tape (1.5-inch)

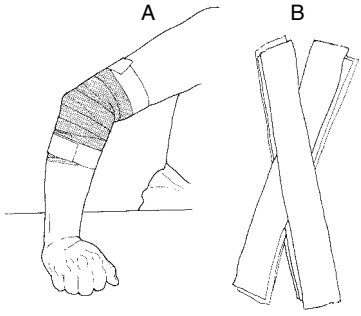

Figure 33.3A–B

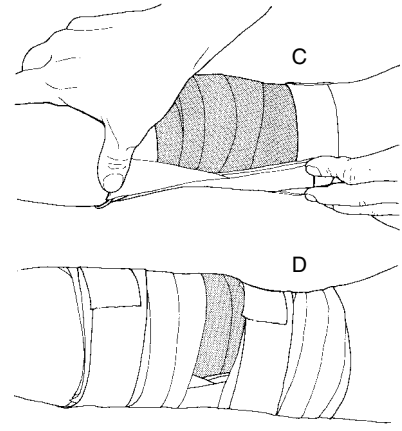

Figure 33.3C–D

Position:
The athlete should sit comfortably on a plinth or taping table or may stand.

Procedure

1. Begin applying the elastic bandage on top of the injured shoulder (Figure 33.4A).
2. Pull the bandage down in front of the shoulder, under the involved arm, and back over the shoulder (Figure 33.4B).
3. Extend the elastic wrap across the chest, under the opposite arm, and around the back in a simple figure-eight pattern (Figure 33.4C), taping down the end of the elastic wrap with white athletic tape.
4. In a modification of this procedure (Figure 33.4D), a second elastic bandage may be used to limit abduction of the injured arm. The bandage is wrapped around the arm, extended around the torso, and brought back around the arm (Figure 33.4E).
5. Adjust the amount of allowed abduction by applying white athletic tape to the bandage between the arm and the body (Figure 33.4F).

TAPING PROCEDURE 5: KNEE

A knee sprain may involve the medial or lateral collateral ligaments and/or other structures. If the injury is a mild sprain, a full medial and lateral taping helps to limit rotation and support the injured collateral ligaments. With the improvement of bracing in recent years, taping of the knee is rare and may only be used when bracing is not available.

Materials Needed

- Underwrap
- Tape-adherent spray
- White zinc oxide athletic tape (1.5-inch)
- Elastic tape (3-inch)

Position
Have the athlete stand on a table. Place a taping block or roll of tape under the heel of the leg to be taped. The knee should be flexed 15 to 20°.

Procedure

1. Prepare the knee by applying a thin layer of tape-adherent spray and underwrap. Apply anchor strips to the thigh and calf approximately 6 to 8 inches from the joint line (Figure 33.5A). A lubricated protective pad may be used over the popliteal fossa to protect this area against chafing.
2. Extend strips of 3-inch elastic tape from the anterolateral portion of the thigh posteromedially to the calf. Cross over the medial collateral ligament and finish on the anterolateral tibia (Figure 33.5B).
3. Apply additional 3-inch strips, beginning anteromedially on the tibia, moving up the inside of the knee joint, and finishing on the anteromedial thigh. Repeat this step 3 to 4 times. Overlap

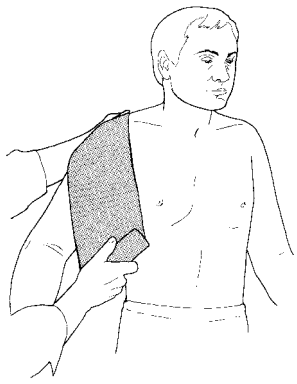

Figure 33.4A

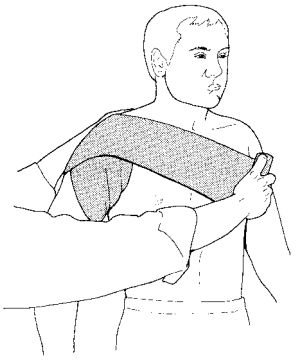

Figure 33.4B

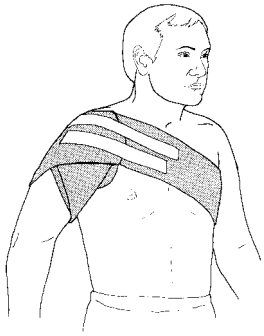

Figure 33.4C

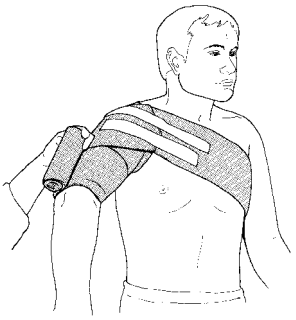

Figure 33.4D

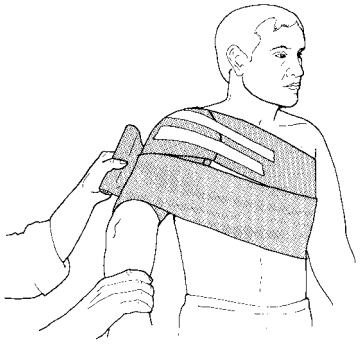

Figure 33.4E

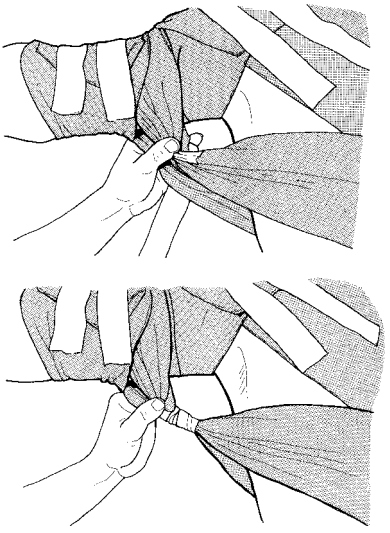

Figure 33.4F

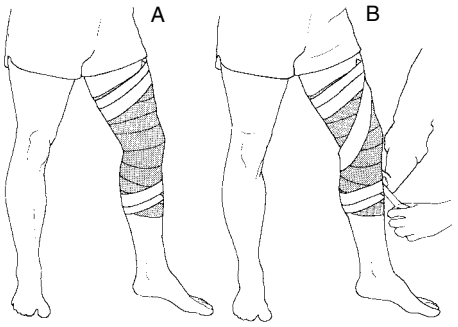

Figure 33.5A–B

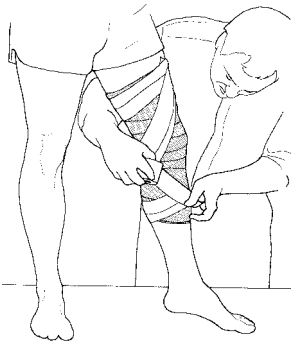

Figure 33.5C

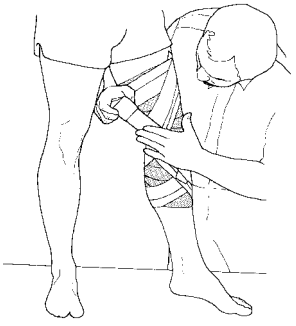

Figure 33.5D

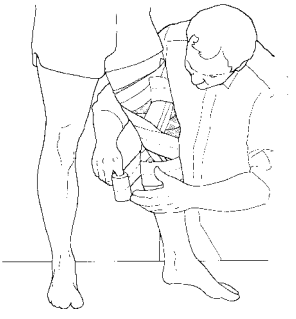

Figure 33.5E

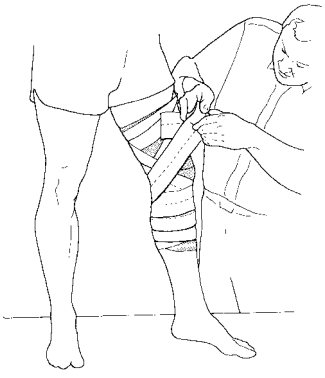

Figure 33.5F

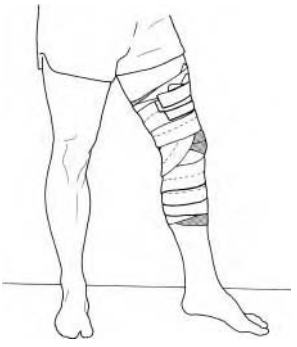

Figure 33.5G

each strip by one-half width to produce a basketweave pattern on the medial portion of the knee (Figures 33.5C and D).

4. This procedure may be repeated on the lateral portion of the knee for lateral collateral ligament support.
5. Complete taping, using 3-inch elastic tape to form a figure-eight pattern (Figures 33.5E and F).
6. Figure 33.5G, the finished procedure, shows the end of the elastic tape secured with 1.5-inch white athletic tape.

TAPING PROCEDURE 6: LATERAL ANKLE SPRAINS (OPEN BASKETWEAVE)

Most (85%) ankle injuries occur with inversion, resulting in damage to the lateral ankle structures. The athlete presents with swelling and pain. An open basketweave taping provides support and compression and decreases swelling while allowing room for expansion or contraction of the tissue. This procedure provides comfort and enough support for partial weight bearing to full weight bearing ambulation with crutches.

Materials Needed

- White zinc oxide athletic tape (1.5- or 2-inch)
- Underwrap
- Tape-adherent spray
- elastic tape (2-inch)
- Heel pads (optional)

Position

The athlete sits comfortably with foot extended approximately 12 inches over the edge of a table with dorsiflexion to neutral. When applying tape, stand facing the plantar surface of the foot.

Procedure

1. Spray the area to be taped with a thin layer of tape adherent. Place the heel pad appropriately (Figure 33.6A). Apply a thin layer of underwrap to the ankle and foot area. Apply underwrap distal to proximal. Be careful to apply the underwrap to the skin obliquely to prevent wrinkles (Figure 33.6A).
2. Apply two separate and overlapping anchor strips at the musculotendinous junction of the gastrocnemius (Figure 33.6A, A). Leave a 1-inch gap between the beginning and end of the anchor strips to allow for possible increased swelling. Apply a distal anchor strip (Figure 33.6A, B) that encircles the distal longitudinal arch. Be careful not to constrict the head of the fifth metatarsal.
3. Apply a strong basketweave of tape strips, creating a stirrup that begins on the medial proximal anchor strips, passes under the heel, and attaches to the lateral proximal anchor strips (Figure 33.6B, A). To prevent inversion, apply moderate tension by pulling laterally. Next, apply a horseshoe anchor to the foot (Figure 33.6B, B), beginning on the medial distal anchor and passing around the heel to the lateral distal anchor.
4. Figure 33.6C shows the basketweave pattern with three stirrups (A, B, C) and three horseshoe anchors (1, 2, 3) interwoven. Maintaining the 1-inch gap on the dorsum of the foot, apply

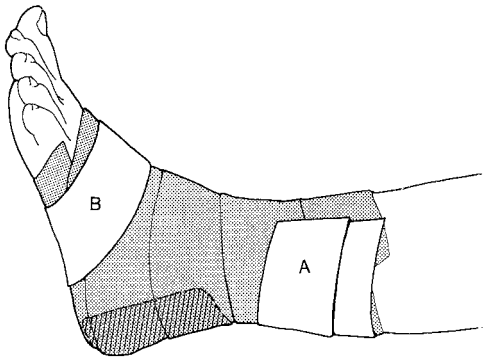

Figure 33.6A

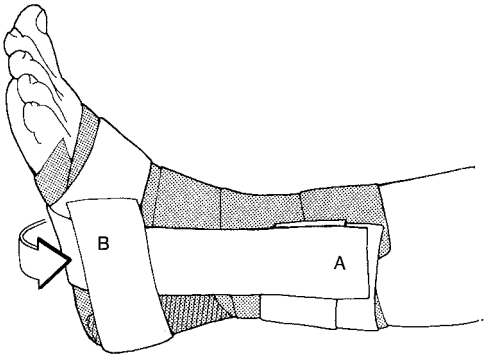

Figure 33.6B

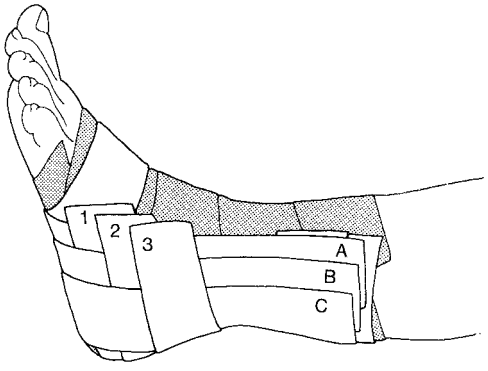

Figure 33.6C

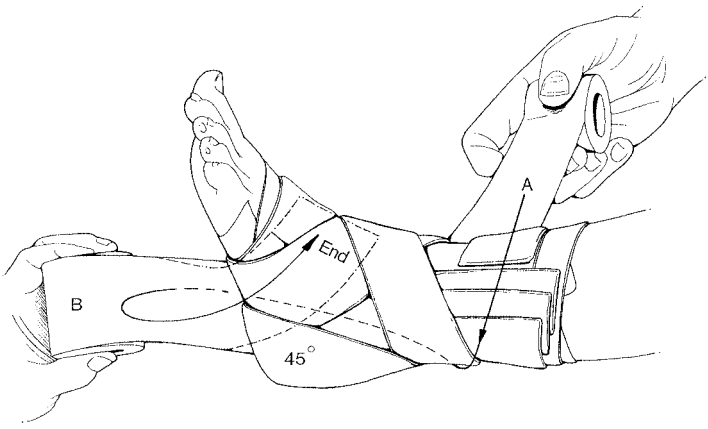

Figure 33.6D

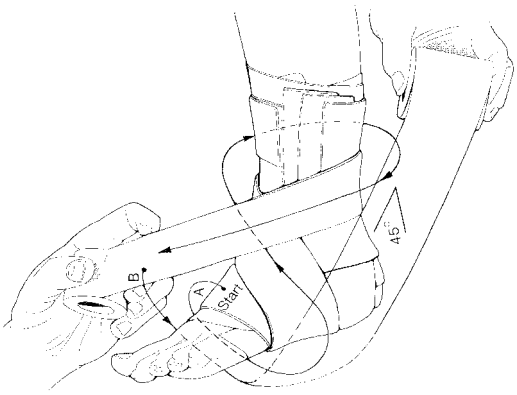

Figure 33.6E

anchors, distal to proximal, up the foot to the proximal anchors.

5. Using 2-inch elastic tape, apply a figure-eight bandage beginning on the dorsum of the foot (Figure 33.6D, A). Pull the tape medially, then pass it beneath the arch of the foot and up onto the lower leg. Continue the strip around the back of the lower leg and extend it back across the dorsum of the foot. Without tearing the tape, progress into a heel lock by angling the tape up and around the lateral heel at a 45° angle (Figure 33.6D, B).
6. Pass the tape around the lower leg and down across the medial heel at a 45° angle, finishing on the dorsum of the foot (Figure 33.6E). This step may be repeated one or two times. Close any gaps left in the tape by applying anchor strips as in step 2.

TAPING PROCEDURE 7: LATERAL ANKLE SPRAINS (CLOSED BASKETWEAVE)

After the athlete has undergone treatment and rehabilitation for a lateral ankle sprain, a closed basketweave taping may be applied to support the ankle and to prevent reinjury. The closed basketweave provides support to lateral ligaments and does not severely restrict movement.

Materials Needed

- Underwrap
- Tape-adherent spray
- Heel and lace pads (optional)
- White zinc oxide athletic tape (1.5-inch)

Position

The athlete should sit comfortably with the knee extended and the foot in neutral to slight dorsiflexion, 12 inches off the edge of the table. When applying the tape, stand facing the plantar surface of the foot.

Procedure

1. Spray the area to be taped with a thin layer of tape adherent. Place the heel and lace pads appropriately. A thin layer of underwrap may be applied. Begin applying underwrap distal and wrap it proximally. Be careful to apply the underwrap to the skin obliquely to prevent wrinkles (Figure 33.7A).
2. Apply two separate and overlapping anchor strips, one at the musculotendinous junction of the gastrocnemius (Figure 33.7B, A) and one over the distal longitudinal arch (Figure 33.7B,

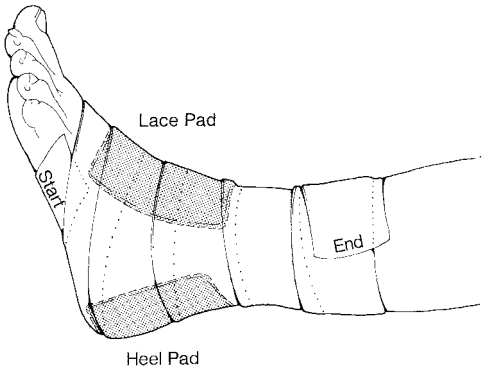

Figure 33.7A

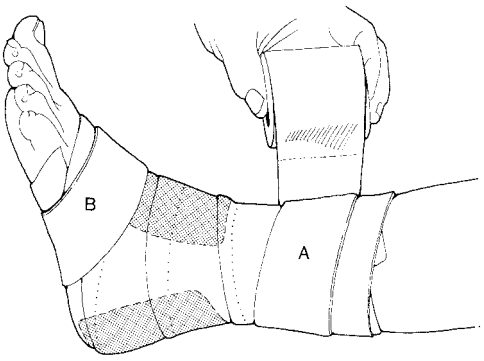

Figure 33.7B

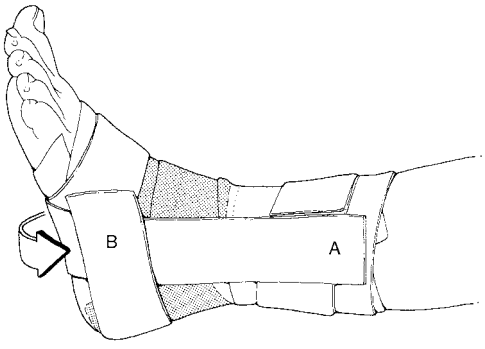

Figure 33.7C

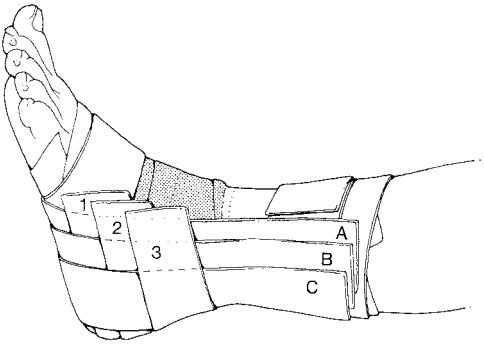

Figure 33.7D

B). Apply these strips obliquely so they do not wrinkle. Be careful not to constrict the head of the fifth metatarsal.

3. Apply a strong basketweave of tape strips, creating a stirrup that begins on the medial proximal anchor strips, passes under the heel, and attaches to the lateral proximal anchor strips. To prevent inversion, apply moderate tension by pulling laterally (Figure 33.7C, A). Next, apply a horseshoe anchor to the foot, beginning on the medial distal anchor and passing around the heel to the lateral distal anchor (Figure 33.7C, B). If extra support is needed to prevent inversion, moleskin tape may be substituted for zinc oxide tape stirrups.
4. Figure 33.7D shows the finished basketweave pattern with the three stirrups (A, B, C) and three horseshoe anchors (1, 2, 3) interwoven. Apply three sets of each, forming a basketweave pattern in the sequence shown. Overlap the tape by half each time.
5. A figure-eight is applied, beginning on the dorsum of the foot (Figure 33.7E, A). Pull the tape medially and pass it beneath the arch and up onto the lower leg. Continue the strip around the back of the lower leg and extend it back across the dorsum of the foot. Without tearing the tape, progress into a heel lock by angling the tape up and around the lateral heel at a 45° angle (Figure 33.7E, B).
6. Pass the tape around the lower leg and down across the medial heel at a 45° angle, finishing on the dorsum of the foot (Figure 33.7F). This step may be repeated one or two times. Close any gaps left in the tape by applying anchor strips as in step 2. If more support is needed, 2-inch elastic tape may be substituted for the heel locks and figure eights. If even more support is desired, a roll of 3-inch 3M Softcast™ material may be used over the finished ankle taping.

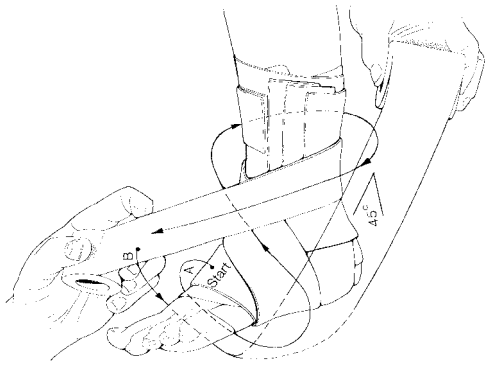

Figure 33.7E

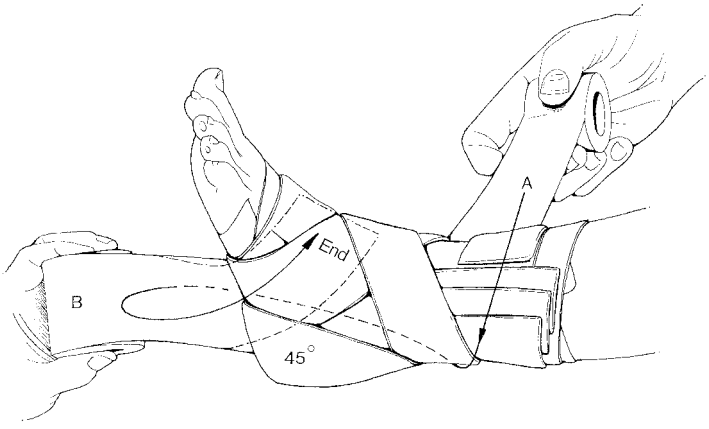

Figure 33.7F

TAPING PROCEDURE 8: LONGITUDINAL ARCH (PLANTAR DYE OR LOW DYE TAPING)

Sprains of the longitudinal arch of the foot can be painful and debilitating. Taping can help to support the arch to decrease pain and improve function. Support of the arch can also be helpful in the management of shin splints and plantar fasciitis.

Materials Needed

- White zinc oxide athletic tape (1.5-inch)
- Tape-adherent spray

Position

The athlete should sit comfortably on a table with the foot in neutral position and extended over the edge of the table.

Procedure

1. Apply a thin layer of tape-adherent spray to the plantar surface of the foot.
2. Apply a horizontal strip of 1.5-inch zinc oxide tape around the foot over the metatarsal heads as shown in Figure 33.8A.
3. Split the 1.5-inch roll of tape in half, making each strip approximately 3/4 inch. Apply a strip of tape beginning on the anchor strip medial to the great toe and passing posteriorly around the heel and back to the anchor strip at the great toe, as shown in Figure 33.8B.
4. Apply a 3/4-inch strip beginning laterally at the fifth metatarsal head, passing posteriorly behind the heel and diagonally across the foot, and ending at the anchor strip laterally, as shown in Figure 33.8C.

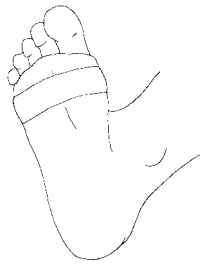

Figure 33.8A

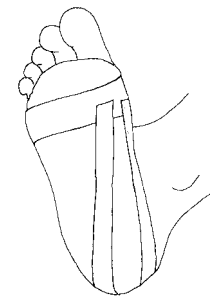

Figure 33.8B

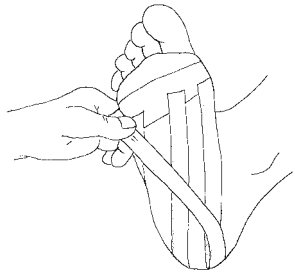

Figure 33.8C

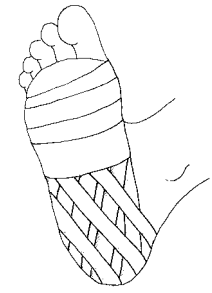

Figure 33.8D

5. Alternate steps 3 and 4, overlapping tape strips by one-half width until the entire plantar surface of the foot is covered.
6. Apply horizontal strips of 1.5-inch tape on the plantar surface of the foot, beginning at the metatarsal heads and moving proximally toward the heel, overlapping each strip by one-half width, as shown in Figure 33.8D.

TAPING PROCEDURE 9: HYPER-EXTENDED GREAT TOE

A hyper-extended great toe (also known as "turf toe" because it frequently occurs on artificial turf) can be a painful and debilitating injury. The athlete may be able to return to participation with less pain with tape support.

Materials Needed

- Underwrap
- White zinc oxide athletic tape (1-inch and 1.5-inch)
- Elastic tape (2-inch)

Position

The athlete should sit comfortably with the foot extended over the edge of a table and dorsiflexed to neutral.

Procedure

1. Prepare the forefoot by applying a thin layer of tape-adherent spray and underwrap (Figure 33.9A).
2. Prepare a 6-inch tape splint by overlapping strips of 1-inch white athletic tape. Apply the splint to the foot from the distal end of the great toe to midarch (Figure 33.9B).
3. Apply strips of 1-inch white athletic tape to anchor the splint in place, in a figure-eight pattern (Figure 33.9C). Apply these strips obliquely to prevent wrinkles and overlap them by one-half width to provide strength (Figure 33.9D).
4. Apply strips of 2-inch elastic tape to support the arch and reinforce the splinting of the great toe (Figure 33.9E). The ends of the elastic tape are secured with white athletic tape to prevent rolling.
5. The finished procedure is shown in Figure 33.9F.

SUMMARY

Athletic trainers practice the art of taping and strapping daily. Practice is required for skillful application of tape in the office or on the field. This chapter has outlined specific tape applications for some of the most common sports injuries. These are by

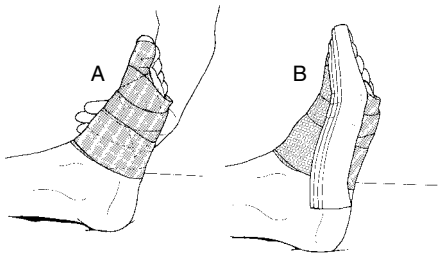

Figure 33.9A–B

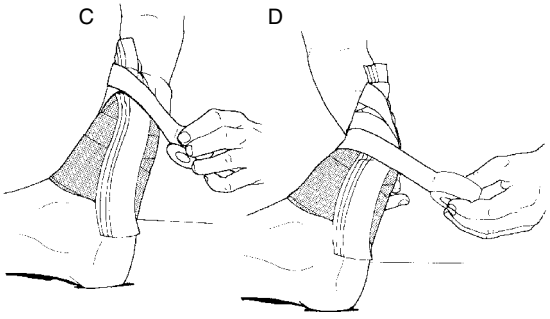

Figure 33.9C–D

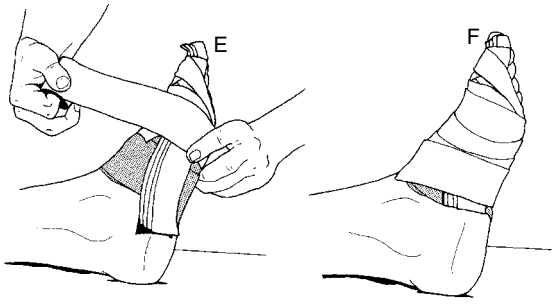

Figure 33.9E–F

no means the only methods available, as most athletic trainers use their skills and imagination to modify applications to fit the athlete, the situation, or their individual preference. The instructions and accompanying illustrations in this chapter provide the primary care physician with a basis for using athletic taping and strapping in the clinic and training room or on the sidelines.

GENERAL REFERENCES

Athletic Uses of Adhesive Tape, Johnson & Johnson Athletic Products, New Brunswick, NJ, 1985.

Rovere, G.D., Curl, W.W., and Browning, D.G., Bracing and taping in an office sports medicine practice, *Clin. Sports Med.*, 8(3), 497–516, 1989.

34 GENERAL TYPES OF INJURIES

Daniel J. Brown and John E. Hocutt

INTRODUCTION

The purpose of this chapter is to provide a broad overview of the epidemiology, mechanism of injury, relevant anatomy, diagnosis, and management of the most common types of sports injuries along with what sports commonly cause these injuries. The reader is referred to subsequent chapters for a more detailed presentation of specific injuries and their treatment.

SPRAINS

I. Epidemiology[1–4]

Sprains are ranked first along with strains among nonfatal sports-related injuries treated in the emergency department and account for 0.7% of all visits to primary care physicians. The incidence is 30 to 40 injuries per 1000 athletes; males > females; 78% acute, 20% chronic. The most common age group is 15 to 44 years, and 1.5% require admission or referral. Activity restriction is required for 80 to 85% (average is 7 days); 70 to 75% are medically attended. Ankle, wrist, and knee are most common sites.

II. Mechanism of Injury[5]

A. Acute overload from a blow or fall
B. Overuse from repetitive microtrauma

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TABLE 34.1
Practical Universal Classification of Sprains and Strains

Grade	Symptom[a]	Grade	Signs[a]	Grade
1	Pain	0–2	Edema	0–2
	Swelling	0–2	Tenderness	0–2
	Disability	0–2	Function loss	0–2
2	Pain	3	Edema	3
	Swelling	3	Tenderness	3
	Disability	3	Function loss	3
			Ecchymosis	30–50%
3	Pain	4	Edema	4
	Swelling	4	Tenderness	4
	Disability	4	Function loss	4
			Ecchymosis	70–90%
			Palpable mass — strain only	
			Palpable gap	

[a] Injuries seen during the golden period have no or minimal pain, swelling, ecchymosis, or tenderness.

[b] Sprain: ligament fibers are torn; strain: muscle–tendon unit is torn.

Note: Pain, tenderness, swelling, edema, and function loss are graded on a 0 to 4 scale, where 0 = absent, 1 = minimal, 2 = mild, 3 = moderate, 4 = severe.

III to V. Anatomy, Symptoms, Signs

See Table 34.1. Ankle sprains can occur in any sport and usually involve a partial or complete tear of the anterior talofibular ligament and/or the calcaneofibular ligament.[6]

VI. X-Rays

X-rays are indicated for grades 2 and 3 and marked pain over a bone. It is possible for a ligament attachment to pull a piece of bone covering off as the ligament is overstretched, producing an avulsion fracture associated with the sprain. Studies may be performed over tape.

VII. Special Studies

A. Magnetic resonance imaging (MRI) clearly shows complete tears of ligaments and often reveals edema and bleeding around the point of damage.[7-9]
B. Appropriate stress films reveal ligament instability.

VIII. Diagnosis

Sprain location _____, grade _____.

IX. Differential Diagnosis

Strain, avulsion fracture, hairline fracture, contusion, eccyhmosis, tendon rupture (particularly Achilles), hematoma, septic joint, inflammatory arthropathies, tendinitis.

X. Treatment/Rehabilitation[5,10–13]

A. Initially: Use RICE (rest, ice, compression, elevation), nonsteroidal anti-inflammatory drugs (NSAIDs), analgesics as needed; splint as necessary, weight bearing as tolerated.
B. Long term:
   Grade 1 — strapping/taping or orthotic for 2 to 3 weeks
   Grade 2 — weight-bearing brace/orthotic/cast for 4 to 8 weeks
   Grade 3 — weight-bearing cast for 3 to 6 weeks followed by orthotic or strapping for 3 to 6 weeks; surgical repair may be indicated for a competitive or young athlete
   Range of motion (ROM) + progressive resistance exercise (PRE) within the limits of pain
   Total recovery — Grade 1, 4 to 6 weeks; grade 2, 2 to 3 months; grade 3, 4 to 6 months
   Return to play when ROM, strength, and function of the injured side nearly equals that of the uninjured side

Consultation: grade 3 and chronic injuries

XI. Complications
Complications include recurrent sprains, completion of ligament tear, stress fracture, joint instability, further bleeding, and degenerative arthritis from chronic joint instability.

XII. Prevention
A. Preseason conditioning and strengthening exercises
B. Progressive adequate warm-up exercises (e.g., flexibility, coordination), although scientific evidence that such exercises reduce injuries is limited
C. Appropriate strapping, taping, and bracing

STRAINS AND MUSCLE TEARS
I. Epidemiology[1–4]
Strains and muscle tears are ranked first along with sprains among nonfatal sports-related injuries treated in the emergency department. Common sites include the muscles supporting the neck (which are relatively weak, especially in forward flexion), hamstrings (seen in soccer and basketball injuries), groin (seen in hockey and soccer), and ankle.[6]

II. Mechanism of Injury[5]
A. Acute overload from a blow or fall
B. Chronic overuse from repetitive microtrauma

III to V. Anatomy, Symptoms, Signs
See Table 34.1.

VI. X-Rays
X-rays are not useful but may demonstrate tissue edema or secondary complications (e.g., avulsion fractures).

VII. Special Studies
Magnetic resonance imaging provides good visualization of muscle–tendon pathology.[7–9]

VIII. Diagnosis
Strain location _____, grade _____.

IX. Differential Diagnosis
Infectious and inflammatory muscle syndromes, sprain, underlying tumor involving the muscle or its attachment, contusion, tendinitis, and fracture.

X. Treatment/Rehabilitation[10–13]
See sprains.

XI. Complications
Recurrent strains, complete muscle tears, and myositis ossificans.

XII. Prevention
See sprains.

CONTUSIONS/HEMATOMAS (BRUISES)
I. Epidemiology[1–4]
Contusions are ranked third along with abrasions among non-fatal sports-related injuries treated in the emergency department. They are very common in collision and contact sports, with 10 to 13 injuries occurring per 1000 athletes. The occurrence among males > females; 60 to 65% restrict activity (average 5 days); 75 to 80% are medically attended. The most susceptible areas depend on the sport, but the most serious injuries occur to the head and face, including the eyes and teeth.

II. Mechanism of Injury
Direct or indirect blunt force.[14]

III. Anatomy
A. Contusions cause damage to muscle, ligament, connective tissue, skin, and vessels; extravascular blood from diffuse capillary bleeding produces ecchymosis, inflammation, and edema.
B. Hematomas occur when a larger sized blood vessel is damaged, allowing a larger volume of blood to leak into surrounding tissue.

IV. Symptoms
A. Contusion — pain, swelling, increased pain with use of involved or surrounding muscle
B. Hematoma — pain, greater swelling, and increased pain with use of involved muscle

V. Signs
A. Contusion — decreased ROM, ecchymosis, and swelling
B. Hematoma — marked decrease in ROM, ecchymosis (gravity dependent), edema, and mass of firm, jelly-like consistency

Measure and record size of hematoma. A greater than 75% subungual hematoma involves a nailbed laceration until proven otherwise.

VI. X-Rays
Soft tissue mass effect may be present.

VII. Special Studies
Magnetic resonance imaging will help distinguish hematomas, calcifications, and solid tumor as well as visualize extent of soft tissue injury.[7–9]

VIII. Diagnosis
Contusion/hematoma location _____, size _____.

IX. Differential Diagnosis
Sprain, strain, tendinitis, fracture, spontaneous bleeding from coagulopathy, soft tissue tumor, lymph node pathology, or reaction.

X. Treatment/Rehabilitation[5,11,14]
A. Initially: Use compression, RICE, or NSAIDs.
   Compression with elastic bandages for the first 24 hours limits swelling due to hemostasis and interstitial pressure. Ensure distal perfusion and venous return of the injured extremity during period of compression. Ice for the first 24 to 48 hours will limit hypoxic injury and speed recovery. Early motion following 24 to 48 hours of rest will prevent the loss of function that may occur with prolonged immobilization.[15] Evaluate hemodynamic status if a large surface area is involved. One or two units of blood may be lost into the anterior thigh with significant hematoma formation (charley horse). Subungual hematomas may be evacuated by drilling or burning a hole in the nail. A nailbed laceration should be sutured with 6–0 chromic. Follow up with padded protection and soaking to keep the hematoma draining.

B. Long-term: Use ROM and PREs, within the limits of pain.
   Contusions/hematomas will be sore after heavy use, after repetitive microtrauma, or after a single significant rebumping or reinjury. Use RICE to minimize the restimulation of inflammation. Minor soreness present in the morning will decrease as the area is warmed up with activity. Recurrent microtrauma to large muscles should be avoided at all costs, as myositis ossificans can result; therefore, heat, massage, ultrasound, or steroid injections should be avoided.

Consultation: large hematoma, recurrent multiple contusions following minimal trauma

XI. Complications
Myositis ossificans, infection, continued pain, adhesions, joint damage, tenosynovitis, and ischemic necrosis.

XII. Prevention
A. Soft or hard padding (e.g., orthoplast) and/or tape
B. Review mechanism of injury with coach/trainer, correct faulty technique, and protect against high-risk movement or activity

DISLOCATIONS/SUBLUXATIONS

I. Epidemiology[3,4]
A. Dislocations are infrequent injuries (2–3 injuries per 1000 athletes) that occur in collision/contact sports: 81% present acutely, 17% chronically; 11% require admission or referral. Incidence in males is twice that of females; 90% require activity restriction (average 15 days); 75 to 80% seek medical help.
B. Subluxations are common following previous extensive injury to a joint.

II. Mechanism of Injury
Acute direct or indirect deforming force (e.g., fall, blow, strong muscle contraction) to ligaments or tendons causes partial (subluxation) or complete (dislocation) joint or tendon displacement.

III. Anatomy
Sequential tearing of ligament fibers surrounding a joint occurs (Figure 34.1). Patella alta, in which the patella rides high in the femoral groove, predisposes to instability.[16]

IV. Symptoms
A. Dislocation — pain (usually severe) that increases with movement of joint or tendon, feeling of joint or tendon weakness and not in correct position
B. Subluxation — variable pain, feeling that joint or tendon was out of position briefly; joint or tendon usually feels

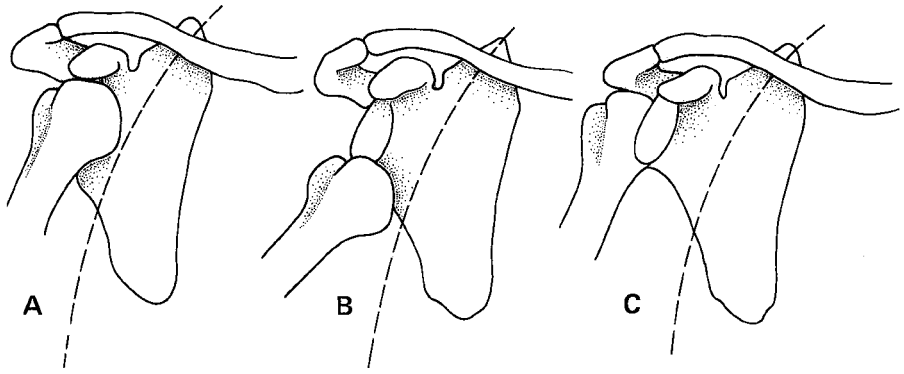


Figure 34.1 Dislocations of the shoulder: (A) anterior, (B) inferior, (C) posterior. (From Hocutt, J.E. and Walters, D.T., in *Family Medicine, Principles and Practice*, 2nd ed., Taylor, R.B., Ed., Springer-Verlag, Berlin, 1983, pp. 1884–1910. With permission.)

weak, and pain increases with use or movement

V. Signs

A. Dislocation — swelling around or below joint or tendon, complete or nearly complete loss of function of involved joint or tendon, gross deformity; check neurovascular status
B. Subluxation — often normal examination except for positive stress tests (e.g., apprehension sign), point tenderness, and edema; a snapping hip is often subluxation due to stenosing tenosynovitis of the ileopsoas tendon[16]

VI. X-Rays

X-rays are diagnostic of type and direction of joint dislocation but usually are not helpful with tendon dislocation unless avulsion fracture coexists; films are usually normal with subluxations. Skyline radiographs are recommended to exclude marginal osteochodral fractures, which can result in loose bodies.[16]

VII. Special Studies

Magnetic resonance imaging is useful for visualizing associated soft tissue damage (e.g., tendon dislocation). Computed tomography (CT) should be used to assess for associated occult fractures.[7–9] Specific stress films are useful for determining if subluxation is present.

VIII. Diagnosis

Subluxation/dislocation location _____, acute/chronic _____.

IX. Differential Diagnosis

Sprain, strain, hemarthrosis, muscle tear, fracture, inflammatory arthritis, and infectious arthritis.

X. Treatment/Rehabilitation

A. Initially: Immediate reduction is usually preferable, before inflammation and spasm become significant.
   Obtain postreduction film; if immediate reduction of joint is not practical, give intravenous analgesic and muscle relaxant before attempting to reduce. Obtain pre- and post-reduction films. Use RICE, NSAIDs, and analgesics as needed.
B. Long-term: Use ROM and PREs within the limits of pain.
   Splint, harness, or padding should be used for 4 to 8 weeks in initial dislocation or until pain is greatly reduced and muscle function provides adequate support in recurrent dislocation and subluxation.
C. Shoulder dislocations: Immediate reduction is usually preferable.
   Up to 35% of first-time shoulder dislocations are associated with axillary neuropraxia; therefore, a careful neurovascular exam is required before and after reduction. Obtain pre- and post-reduction films if possible. Immobilization should be for pain management only; it has no effect on the natural history of recovery from shoulder dislocations. A preliminary arthroscopy will determine extent of injury and can directly repair or lead to open

reconstruction repair. A quick diagnostic arthroscopy does not hinder an open reconstruction in the same sitting. Early arthroscopy will result in a lower recurrence rate than nonoperative treatment even in first-time shoulder dislocations. Nonoperative treatment includes early ROM and isometric exercises followed by isokinetic and plyometric exercises at 6 to 8 weeks. An orthosis that limits external rotation and abduction may be used for midseason athletes who return to play prematurely.[17,18]

Consultation: dislocation-associated neurovascular compromise, recurrent subluxations/dislocations

XI. Complications
Avulsion fracture, joint fracture, ligament laxity, hemarthrosis, periarticular nerve damage, tendon disruption, tendinitis, and joint calcification.

XII. Prevention
A. Compensatory muscle strengthening
B. Joint-restrictive supports (e.g., Duke–Wyre shoulder harness)

ABRASIONS (CINDER BURNS, FLOOR BURNS, FRICTION BURNS, MAT BURNS, RASPBERRY, ROAD RASH, STRAWBERRIES, TURF BURNS)

I. Epidemiology[1-4]
Abrasions are ranked third along with contusions among nonfatal sports-related injuries treated in the emergency department. Abrasions are extremely common injury in collision/contact sports (10–13 injuries per 1000 athletes), males > females. Abrasions are usually a nuisance problem unless repetitive irritation advances the injury or allows it to get infected. Knee, hand, anterior tibia, iliac crest, and elbow are most susceptible.

II. Mechanism of Injury
Abrasions are caused by excessive shear forces on skin, especially against a rough surface and with high speeds.

III. Anatomy
The abrasion may be partial or full skin thickness.

IV. Symptoms
Pain, particularly with movement, is often significant because of the concentration of pain fibers in the skin.

V. Signs
A. Bleeding and weeping of the skin are common; record size.
B. Increased skin temperature and erythema suggest infection.

VI. X-Rays
X-rays are not indicated unless looking for radiopaque foreign body.

VII. Special Studies
None.

VIII. Diagnosis
Abrasion location _____, size _____, grade _____.

IX. Differential Diagnosis
Cellulitis, dermatitis syndromes.

X. Treatment/Rehabilitation
A. Initially: Meticulous cleaning/scrubbing should be followed by the application of a thin layer of antibiotic ointment (e.g., Betadine®, Bacitracin®); local anesthesia may be necessary for thorough debridement. Use RICE and analgesics as needed, systemic antibiotics if infection present, and tetanus prophylaxis when indicated (Table 34.2).
B. Long-term: Cover with a bio-occlusive hydrophilic bandage (e.g., Duoderm®) with overlapping 3/4- to 1-inch margins; the dressing should be changed any time fluid accumulation is large or extends to within 1/4 inch of the dressing edge.

Consultation: none

XI. Complications
Infection or discoloration (tattooing).

XII. Prevention
Use proper protective equipment, modify form or technique, wear custom-molded padding (soft or hard, such as Orthoplast®), and apply skin lubricant (e.g., Skin Lube®).

TABLE 34.2
Guidelines for Antitetanus Treatment of Patients with Open Wounds

Tetanus Immunization History	Clean Minor Wounds		All Other Wounds[a]	
	Td[b]	TIG	Td	TIG
Unknown or <3 doses	Yes	No	Yes	Yes
≥3 doses	No[c]	No	No[d]	No

Note: Td = Adult tetanus toxoid + diphtheria vaccine; TIG = tetanus immune globulin.

[a] Dirty wounds include contamination with dirt, feces, soil, or saliva; puncture wounds; avulsions; and wounds from missiles, crushing trauma, frostbite, or burns.

[b] Give DPT if <7 years old (or DT if pertussis is contraindicated).

[c] Give Td booster if >10 years since last dose.

[d] Give Td booster if >5 years since last dose.

LACERATIONS

I. Epidemiology[1-4,19]

Lacerations are ranked fourth among non-fatal sports-related injuries treated in the emergency department. Cuts are the number one cause of nonfatal injuries among 10 to 17 year olds (8–10 injuries per 1000 athletes); relatively common in contact/collision sports and in off-road bicycling. Rate of occurrence for males is over twice that for females; 90 to 95% seek medical help; 50% require activity restriction (average 3 to 4 days).

II. Mechanism of Injury

Significant direct (e.g., sharp object) or indirect compression force.

III. Anatomy

Sequential tearing of the epidermis, dermis, and subcutaneous tissue.

IV. Symptoms

Pain, feeling of loss of skin support, numbness if sensory nerve is cut, loss of function distal to laceration if tendon or motor nerve is cut, and bleeding.

V. Signs

Obtain vital signs. Measure and record size, depth, and location. Note neurovascular and functional status. Check for other injuries.

VI. X-Rays

X-rays are not necessary or helpful unless looking for a radio-opaque foreign body or underlying bony damage.

VII. Special Studies

None.

VIII. Diagnosis

Laceration type _____, location _____, size _____.

IX. Differential Diagnosis

Foreign body or infection.

X. Treatment/Rehabilitation

A. Apply direct pressure, preferably with sterile material, until help arrives. If clean ice or snow is around and sterile material is not, put some directly on the wound and then reapply pressure; otherwise, use as clean a material as possible. Tourniquets are rarely needed, especially when medical help is nearby. If utilized, a tourniquet must be released every 5 to 10 minutes or so to be sure the circulation is not compromised to the extremity. If a tourniquet is needed for an extended period, apply a constrictive bandage above the wound that may be easily and conveniently released. While the tourniquet is held tight, reclean the wound and then redress with clean bandages and wrap moderately tightly.

B. Clean and debride thoroughly before suturing. Risk and rate of infection increase with delay in closure and contamination of wound. Lacerations due to crush injuries (often hand injuries) have increased rates of infection. These injuries require more extensive cleaning with higher pressure irrigation before closure.[20]

C. Methods of closure:[15]

1. Sutures — complete closure with most tensile strength and lowest dehiscence rate. Disadvantages include more pain, higher tissue

reaction rates, higher cost, and increased resources for removal of sutures.
2. Staples — rapid, low cost, low tissue reactivity, and no risk for needle stick. The main disadvantage is that staples may interfere with imaging studies. The closure is less meticulous than sutures.
3. Tissue adhesives — same advantages as staples but increased patient comfort and resistance to bacterial growth. Disadvantages are low tensile strength and increased rates of dehiscence.
4. Surgical tapes — advantages similar to tissue adhesives except that tapes offer the least tissue reactivity and highest dehiscence rates and must remain dry.

D. Suture closed if less than 8 to 12 hours after the trauma. Facial wounds, because of excellent blood supply, may be closed 12 to 24 hours following trauma if edges are excised. A contaminated wound should be left open. Hand lacerations <2 cm need not be closed.[21]
E. Anesthesia (topical):
1. Lidocaine (0.5 to 2.0%); more than 1% may cause reduced tensile strength.
2. Lidocaine with epinephrine is used in highly vascular areas to reduce bleeding unless vasospasm could compromise tissue circulation (e.g., digits, ears, penis); maximum dose is 4.5 mg/kg or 300 mg (30 cc of 1% solution).
3. TAC; 25 mg tetracaine, 2.5 mL 1:1000 adrenalin, and 0.59 g cocaine are mixed in sterile water to make 5 mL.
4. Regional blocks should be done only with plain lidocaine.

F. Needles:
1. Type — cutting needle for the skin, round tip needle for fascia and subcutaneous tissue
2. Shape — curved needles for skin and deeper tissue closure, straight needles for skin closure
3. Size — P-1 (small) to P-3 (large)

G. Suture material:
1. Colored nylon and prolene are strong with minimal tissue reaction. Prolene is quite slippery, so it is the material of choice for subcuticular applications. Nylon is often the preferred choice for surface suturing (interrupted). Silk and chromic are too reactive.
2. Rarely need greater than 5-0 on the face or hands or 4-0 on the trunk or extremities.
3. For buried sutures, Dexon® causes little reaction and is water soluble in 21 days. Vicryl® maintains tensile strength for 3 weeks and dissolves in 90 days. Clear nylon and Mersilene® are appropriate for nonabsorbable sutures. Avoid chromic cat gut because of its inflammatory reaction. Plain cat gut is absorbed in a few days and may be used to tie blood vessels and fat.

H. Closure technique:
1. Primary is reserved for clean wounds less than 6 to 8 hours old.
2. Secondary (delayed) is used for wounds that are dirty or older than 6 to 8 hours.
3. Preoperative shaving increases infection rate if skin is disrupted; use scissors or electric clippers if possible. Do not shave eyebrows.
4. Mark borders of wound for proper closure before injecting with anesthetic.
5. Bury all but skin knots.
6. Interrupted sutures should be kept close to skin edge and as far apart as long. Sutures should be deeper than wide with equal bites at same depth. Halving of wound with sutures ensures good alignment. Best cosmetic results are achieved with multiple sutures very close together.
7. Continuous (simple running) provides quicker and easier insertion and removal but higher risk for wound opening if suture breaks or knot opens. Prolene material is slippery enough to adjust tension evenly in wound after suturing and provides a hemostatic lock. Cosmetic results may be better.

8. Evert skin edges slightly with deep stitches if possible to take tension off suture line (improves scar formation).
9. Undermining decreases scar width.

I. Post-closure:
1. Cover with bio-occlusive bandage (e.g., Duoderm®) and have patient change bandage twice a day after cleaning. Patient can shower, swim, and otherwise lead a relatively normal life while keeping the wound clean and moist. Moisture improves scar formation. If not using bio-occlusive bandage, the patient must wait 48 hours before getting the wound moist unless treating infection. Soaking should be avoided until wound is well healed because of weakening of suture line.
2. When appropriate, immobilization may help decrease likelihood of infection.

J. Confirm that tetanus immunization is current (see Table 34.2).

K. Systemic broad-spectrum antibiotics (e.g., cephalosporin) are indicated in cases with a high risk for infection (e.g., patients with diabetes mellitus or who are immunocompromised or obese; vascular insufficiency; human bites; severe contamination). More specific antistaphylococcal coverage may be necessary. Routine prophylaxis is not indicated.

L. Suture removal:
1. Face, 3 to 5 days
2. Trunk or extremities without tension, 7 to 10 days
3. Extremities or joints, 10 to 14 days

Consultation: large, deep lacerations

XI. Complications
Infection, dehiscence with or without further injury to adjacent structures.

XII. Prevention
Wear padded protective equipment on appropriate playing field or arena. Remain within functional ability of athlete for activity performed.

FRACTURES

I. Epidemiology[1–4]
Fractures are ranked second among nonfatal sports-related injuries treated in the emergency department (4–5 injuries per 1000 athletes); males > females, except stress fractures (females > males);[16] 93% present acutely, 6% chronically. Fractures are not uncommon in contact/collision sports and are often associated with severe ligament or muscle injury; 5% require admission or referral; 90 to 95% are medically attended; 70 to 75% require activity restriction (average 17 days for upper extremity neck and trunk, 29 days for lower extremity). Stress fractures are relatively common in endurance sports. Pediatric patients with growing bones are predisposed to injury due to weaker epiphyseal plates.[16,22]

II. Mechanism of Injury
A. Acute overload from direct or indirect trauma

B. Overuse — repetitive microtrauma, fatigued muscle, excessive loading, mechanical stress, piezoelectric stimulus, bone remodeling, bone strain (e.g., normal or accelerated remodeling, weakened trabeculae, microfracture, macrofracture) (Figures 34.2 and 34.3)[14]

III. Anatomy
A fracture is a break of bone or cartilage. Types include linear, comminuted, impacted, incomplete, complete, oblique, spiral, transverse, and epiphyseal. The most common non-stress fractures are those of the distal radius (snowboarding, soccer, or in-line skating) and the tibia (skiing, soccer, and football). Stress fractures commonly involve the tibia, metatarsals, proximal femur, and calcaneus and often result from beginning a new sport or training regimen.[6,16]

IV. Symptoms
A. Pain over fracture site, often progressive

B. Increased pain with weight bearing or load carrying

C. Resting pain after heavy use

V. Signs
A. Swelling, distorted anatomical relationships (including irregular bone lines), bruising, abnormal posturing, and crepitus and tenderness with passive

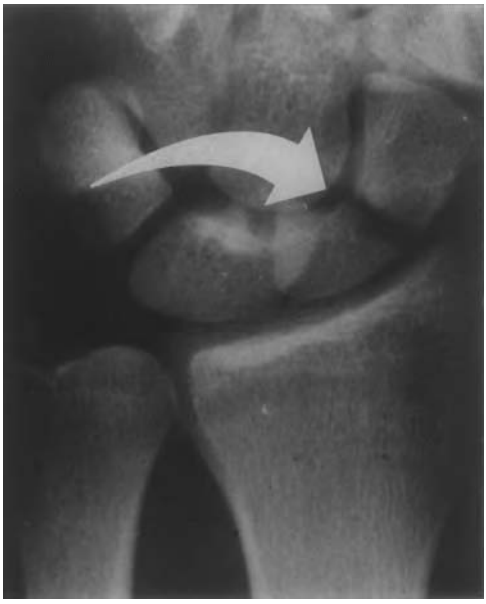


Figure 34.2 A navicular fracture. (Radiographs courtesy of Mark Glazer, M.D., and Jack Wills, M.D., Department of Radiology, Medical Center of Delaware, Wilmington.)

movement and the application of pressure at or across the site; check joints above and below involved bone.

B. Check neurovascular status. Diminishment of pulse is possible distal to displaced or angulated fracture.

C. Check for loss of sound transmission in fractured long bones (positive osteophony).

VI. X-Rays

X-rays are the standard for diagnosing fractures (high specificity). While routine anteroposterior (AP) and lateral views usually suffice, obliques and special views may be necessary. Repeat films as necessary for continued pain, as hairline fractures may become evident after several days and stress fractures after several weeks.

VII. Special Studies

A. Bone scan (99mTc) or CT scan (high sensitivity) may be necessary at any time if the clinical situation suggests a fracture (stress) and the plain films remain negative. Bone scan specificity depends on correlation with site of pain; 35 to 40% of athletes have two or more asymptomatic sites with high uptake, presumably due to remodeling stress without actual fracture.[23] MRI is useful to exclude soft tissue interposition and later to identify avascular necrosis in severe fractures.[16]

B. Computer-assisted thermography may be helpful in distinguishing soft-tissue inflammatory syndromes from stress fractures, particularly in the anterior tibial area.[24]

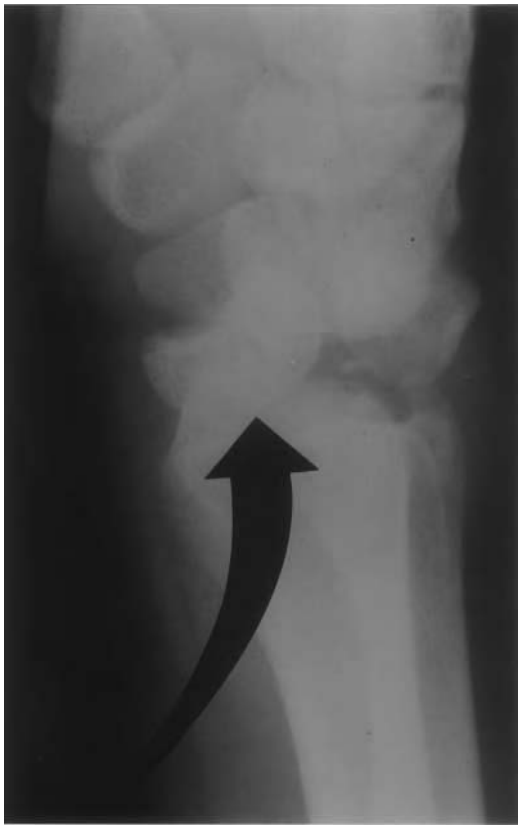


Figure 34.3 Lateral view of the wrist, showing volarward dislocation of the lunate through the space of Poirier. (Radiographs courtesy of Mark Glazer, M.D., and Jack Wills, M.D., Department of Radiology, Medical Center of Delaware, Wilmington.)

VIII. Diagnosis

Fracture type _____, location _____.

IX. Differential Diagnosis

Soft tissue contusion, bone contusion, osteomyelitis, arthritis syndromes, severe sprains and strains, dislocation, subluxation, and compartment syndrome.

X. Treatment/Rehabilitation

Nonstress Fractures

A. Initially: Reduce to proper position and splint, tape, or cast in place; verify neurovascular status. Use RICE, NSAIDs, or analgesics as needed.
B. Long-term: Use ROM + PRE within limits of pain after cast/splint removal.
C. Use electrical stimulation for resistant fractures.

Stress Fractures [25]

A. Uncomplicated stress fractures: Modify exercise prescription. Identify and remove causes of stress fracture prior to return to play. Rest 4 to 8 weeks. Maintain fitness by cycling, water running, or working out on exercise machines. Use ROM or PREs within the limits of pain to strengthen and balance muscles around site of fracture. Return to activity when no local tenderness is noted and athlete is pain free during daily activities. Promote a gradual return to walking, jogging, or running. Educate the patient regarding long-term control and therapy.
B. High-risk stress fractures:
   1. Femoral neck — distraction type more common among elderly; high risk of progression to complete fracture and displacement. Compression type more common in younger athletes. High risk of avascular necrosis and nonunion. Advise rest and serial radiographs every 2 to 3 days during first week to monitor progress indicated for both types. Immediate open reduction and internal fixation are required if fracture widens. Crutches are indicated until athlete is pain free and radiographs show evidence of healing or callus formation. Weight-bearing exercise can be resumed gradually over 6 to 8 weeks.
   2. Anterior cortex of tibia — prone to nonunion and sometimes develop into acute transverse fractures of the tibia with increased exercise. Aggressive treatment with immobilization in non-weight-bearing cast for 3 to 6 months or early surgery with insertion of an intramedullary rod, which may enable a return to play after 6 to 8 weeks.
   3. Medial malleolus — if fracture is visible on radiograph, undergo open reduction and internal fixation. Fractures seen on bone scan but not on plain radiograph should be treated with immobilization in pneumatic brace for 6 weeks.
   4. Talus — usually only seen on bone scan; immobilize in non-weight-bearing cast for 4 to 6 weeks if diagnosed early. For longstanding symptomatic fractures, excision of the lateral process of the talus through the fracture is recommended.
   5. Tarsal navicular — conservative treatment with rest; high incidence of delayed union and nonunion. Recommended treatment of tarsal navicular stress fractures is immobilization in non-weight-bearing cast for at least 6 weeks. When the patient is pain free upon palpation over the navicular bone, a 6-week rehabilitation program is started. This program consists of joint mobilization, muscle strengthening, and gradual return to activity.
   6. Fifth metatarsal — high incidence of delayed union and nonunion. Most heal with immobilization in non-weight-bearing cast for 3 to 6 months. Those who cannot tolerate prolonged immobilization require surgery with screw fixation or bone grafting.

Growth Plate Fractures[26]

A. Salter types I and II are usually treated by closed manipulation and adequate immobilization.
B. Salter types III, IV, and V often require surgery to achieve satisfactory alignment.

Consultation: physeal, unstable, nonreducible, open or nonhealing fractures or those associated with a major dislocation or possible compartment syndrome or neurovascular injury

XI. Complications
Extension of hairline to complete fracture, nonunion, joint damage, circulatory compromise, nerve damage.

XII. Prevention
Use balanced muscle strengthening, proper protective equipment and playing surfaces, proper progression within sport, and adequate conditioning to maintain technique.

GANGLIA

I. Epidemiology
Ganglia are synovial cysts occurring as the most common tumor of the hand or wrist. The knee, ankle, and foot are less common sites.

II. Mechanism of Injury
Individual predisposition, but repetitive trauma or overuse may increase development.

III. Anatomy
Circumscribed synovial cyst of a joint capsule or tendon sheaths.

IV. Symptoms
A. Occasional pain with variable lump at site
B. Increased pain with use, especially when cyst is inflamed

V. Signs
A. Firm cystic structure of variable size (several millimeters to 3 cm)
B. May be soft, rubbery, or bony hard; nonpulsatile

VI. X-Rays
X-rays are not normally helpful and are needed only occasionally to rule out tumor.

VII. Special Studies
Magnetic resonance imaging is helpful when needed to identify occult cyst or rule out tumor or vascular lesion.[6]

VIII. Diagnosis
Ganglion (or synovial) cyst location _____.

IX. Differential Diagnosis
Soft tissue tumor, bony tumor, and aneurysm.

X. Treatment/Rehabilitation
A. Initially: Use RICE, NSAIDs, and analgesics as needed; watchful waiting; aspiration with or without steroid injection.
B. Long-term: Surgically excise ganglia and stalk (recurrence rate 20 to 40%).

Consultation: intractable pain, cosmesis, decreased function, neurovascular compromise

XI. Complications
Interference with joint function secondary to mass effect, inflammation, or pain; neurovascular compromise, fascial dissection.

XII. Prevention
None.

MYOSITIS OSSIFICANS

I. Epidemiology
Infrequent complication of contusions, most frequent in anterior thigh and upper arm.

II. Mechanism of Injury
Repeated trauma to local tissue in susceptible individual (e.g., active bone turnover).

III. Anatomy
New bone formation from calcification and osteogenesis 2 to 6 weeks after significant trauma. Usually has mature bone at periphery compared to malignant osteogenic sarcoma, which has wild bone at its periphery.

IV. Symptoms
Pain, muscle weakness, soreness, decreased function, and swelling.

V. Signs
Palpable firmness and tenderness interspersed in muscle and decreased ROM.

VI. X-Rays
X-ray indicates simple bony exostosis with sharp extension into affected muscle or ectopic calcification within muscle (Figure 34.4). Initial X-rays may be negative.

VII. Special Studies
Ultrasound may be positive before any x-ray changes develop.[27] MRI and CT are usually positive and more sensitive than x-rays.[7–9]

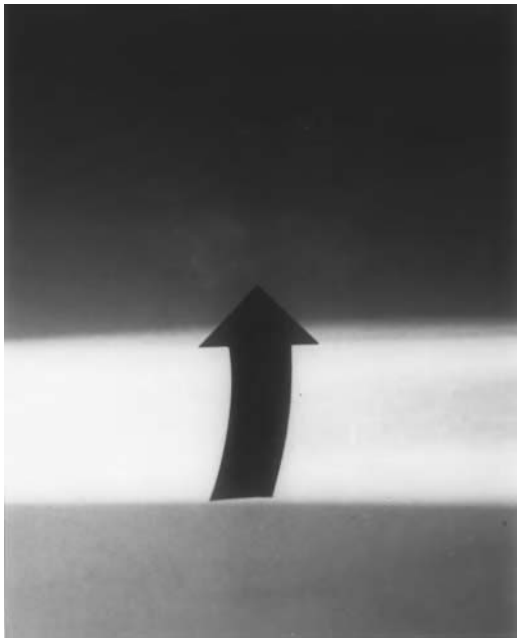


Figure 34.4 Example of myositis ossificans. (Radiographs courtesy of Mark Glazer, M.D., and Jack Wills, M.D., Department of Radiology, Medical Center of Delaware, Wilmington.)

VIII. Diagnosis
Myositis ossificans location _____.

IX. Differential Diagnosis
Soft tissue tumor, malignant bone tumor (osteogenic sarcoma), contusion, or noncalcified hematoma.

X. Treatment/Rehabilitation[11,13]
A. Initially: Use RICE, NSAIDs, and analgesics as needed.
B. Long-term: Use physical therapy (active), ROM, PREs within limits of pain, contrast baths, and ultrasound.
Avoid passive manipulation and early surgery. Surgery is only appropriate to remove calcification after the lesion is completely stable and all pain and inflammation have been controlled for many months or years or if distinction from osteogenic sarcoma is not clear. Excisional biopsy is more accurate in myositis ossificans the longer the hematoma is present. Confusion with malignancy has occurred with immature lesions! Return to play when full ROM and strength have returned.

Consultation: intractable pain, impaired function, unclear diagnosis

XI. Complications
Extensive calcification with resultant significant functional impairment.

XII. Prevention
Wear proper protective equipment (padding) and use appropriate playing surfaces.

OVERUSE

I. Epidemiology[5,28–31]
Overuse occurs in 25 to 50% of all sports injuries; 80 to 90% involve endurance sports; 80% involve the lower extremity, with the knee followed by heel, ankle, and foot being the most frequent locations. Males are affected 85% of the time, females 15%; most common age group is 20 to 29 for competitive athletes and 30 to 49 for recreational athletes. Common examples include patellofemoral syndrome, plantar fasciitis, lateral/medial epicondylitis (elbow), rotator cuff syndrome, anterior/medial tibialis strain, and stress fractures. The majority of overuse injuries involve the muscle–tendon unit. Risk factors can be extrinsic (e.g., training errors, surfaces, footwear and equipment, environmental conditions) or intrinsic (e.g., malalignment, muscular imbalance and insufficiency, leg length discrepancy).

II. Mechanism of Injury[31]
Repetitive microtrauma, overload, sprain, strain, etc. (see Table 34.3, Figure 34.5).

III. Anatomy
Medial tibial stress syndrome (shin splints) seen in runners is due to overuse of the soleus muscle on its attachment to the tibia.[16]

IV. History
See Chapter 27.

V. Physical Exam
See Chapter 27.

VI and VII. X-Rays and Special Studies

VIII. Diagnosis
Overuse syndrome type _____, grade _____.

IX. Differential Diagnosis
Diagnoses include occult tumor/cancer, inflammatory arthritis, and cellulitis.

TABLE 34.3
Musculoskeletal Overuse Injuries: A Clinical Guide

Grade	Anatomy	History	Physical Exam	Diagnostic Studies
1	Mild tissue inflammation	No pain or less than 2 weeks of transient pain 1 to 2 hours after extreme activity; soreness; normal or slightly decreased sports performance; usually experienced by a new athlete or due to a new training program	Generalized tenderness	None
2	Moderate tissue inflammation	Two to 3 weeks of long-standing pain late in activity or immediately after vigorous activity; performance somewhat restricted; technique, equipment, or environment could be source of problem	Localized pain; no discrete point tenderness	None
3	Major tissue inflammation, periostitis, bone microtrauma (pre-stress-fracture syndrome)	Three to 4 weeks of pain in early or middle of activity; performance significantly restricted	Point tenderness; percussion tenderness; pressure elsewhere produces pain at point; other evidence of inflammation (heat, erythema, swelling, crepitation)	X-ray may show edema, usually negative; bone scan (+) 75%.
4	Degeneration of soft tissue, stress fracture, compartment syndrome (especially if swelling is major finding)	Greater than 4 weeks of chronic, unremitting pain, even at rest	All of the grade 3 signs plus disturbance in function, decreased ROM, muscle atrophy	X-ray positive 2–6 weeks after stress fracture. Bone scan (+) 95% in 1–4 weeks.[12] MRI indicates edema.[4–6] Computer assisted thermography distinguishes stress fracture from soft tissue inflammation.[13] Isokinetic testing may demonstrate functional deficits.

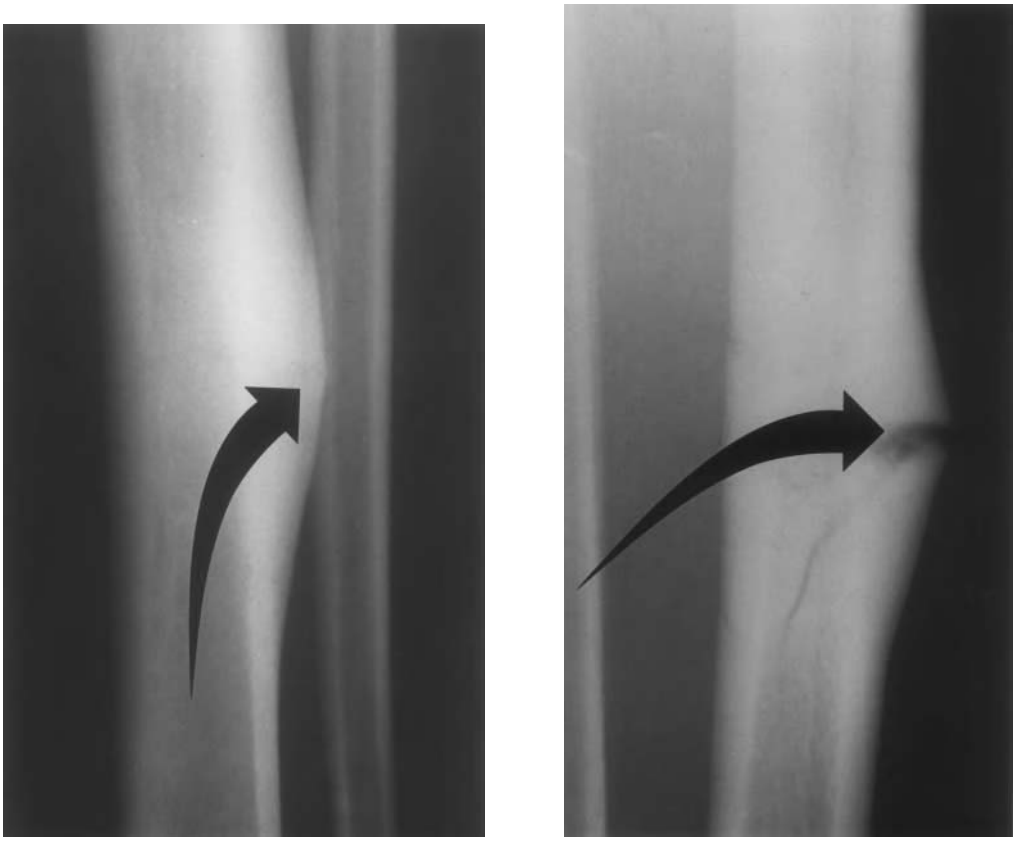
Source: Adapted from McKeag, D.B. and Dolan, C., *Phys. Sportsmed.*, 17(7), 108–123, 1989. With permission.

X. Treatment/Rehabilitation[11–13,29-31]

See Table 34.4.

Grade 1 — Ice, no change in activity level
Grade 2 — Modification (cross training) and titration (10 to 25% reduction) of offending activity, education, and anti-inflammatory measures
Grade 3 — RICE and NSAIDs for 5 to 7 days, modification and 25 to 75% reduction of activity until grade 2 or lower is achieved (usually days to weeks)
Grade 4 — RICE, NSAIDs, or analgesics as needed with possible immobilization; weeks to months of rehabilitation before return

Consultation: chronic disability, history, and exam conflict



(a) **(b)**

Figure 34.5 (a) Stress fracture of the mid third of the tibia; (b) chronically separated stress fracture of the tibia. (Radiographs courtesy of Mark Glazer, M.D., and Jack Wills, M.D., Department of Radiology, Medical Center of Delaware, Wilmington.)

TABLE 34.4
Physiologic Effects of Temperature on Injured Tissue

Injury	Cold	Heat
Muscle spasm	Marked decrease by direct action on muscle and afferent nerve fibers	Mild decrease initially; possible secondary increase after 30 minutes
Metabolism	Decreases	Increases
Pain	Initial few minutes of discomfort, then numbness and anesthesia	Soothes initially; secondary rebound increase after 30 minutes
Edema	Decreases	Increases
Penetration	Usually very efficient; varies with method used	Normally shallow; fair with moist heat; better with ultrasound
Circulation	Vasoconstriction, decreased blood flow, decreased hematoma formation	Vasodilation, increased blood flow, increased hematoma formation

Source: Hocutt, J.E., *Am. Family Phys.*, 12, 141–144, 1981. With permission.

XI. Complications
Grade 3 sprain or strain, muscular atrophy, progression to frank fracture with or without displacement.

XII. Prevention
A. Adequate, but most importantly balanced, muscle power greatly protects all areas of the body from overstretching, overstraining, and overuse.
B. Progressive training program is helpful.
C. Optimum aerobic conditioning helps reduce the risk of overuse and acute sports injuries by delaying onset of fatigue and imbalance.[32] The 10% rule may be applied in endurance sports (i.e., increasing activity by 10% each week).[16]

BLISTERS
I. Epidemiology
Blisters are common overuse injuries due to new or stiff equipment, wrinkles in socks, or sudden increase in training schedule. Weight-bearing and equipment-contact areas are most susceptible.

II. Mechanism of Injury
Excessive single or repetitive shear force.

III. Anatomy
Separation of the epidermis and dermis with fluid extravasation.

IV. Symptoms
Pain or sensitivity of skin.

V. Signs
Vesicle formation, weeping, and localized tenderness.

VI. X-Rays
X-rays are not indicated.

VII. Special Studies
Cultures if signs of infection are present.

VIII. Diagnosis
Blister location _____, size _____.

IX. Differential Diagnosis
Infection, particularly viral; allergic reaction; bullous pemphigoid; epidermolysis bullosa; Weber–Cockayne syndrome; other dermatitis syndromes.

X. Treatment/Rehabilitation
A. Initially: Use RICE and analgesics as needed.
B. Long-term: Sterile aspiration should be followed by circumferential application of a moleskin or foam rubber donut and debridement of roof if vesicle ruptures.

XI. Complications
Infection and scarring.

XII. Prevention
Use appropriate padding with 1/2-inch margins over an adherent such as tincture of benzoin or quick-drying adherent (e.g., tape, moleskin, "second skin"); wear protective equipment and appropriate training clothes (e.g., two pairs of socks or double-layered socks such as Runique®, gloves); apply skin lubricant (Skin Lube®, Vaseline®); utilize progressive skin toughening through graduated training or skin hardening with 10% tannic acid soaks.

MUSCLE SORENESS
Muscle soreness is a mild form of muscle strain resulting from overuse appropriate to the specific muscle involved. (See muscle strain and overuse injuries.)

CRAMPS
I. Epidemiology
Cramps are common involuntary muscle spasms that occur during vigorous activity in a relatively hot environment.

II. Mechanism of Injury[33]
No single factor has been identified as the cause of muscle cramps. Possible causes of cramps during exercise include metabolic factors such as acids, ions, proteins, and hormones. Another possibility is damage to the connective tissue in muscle due to repetitive eccentric muscle contractions associated with prolonged weight-bearing exercise. Cramps after exercise may be due to fatigue-induced neuromuscular changes. These changes have increased baseline electromyographic activity.

III. Anatomy
Systemic, metabolic syndrome involving one or more specific muscles.

IV. Symptoms
Pain and impaired function of involved muscle.

V. Signs
Painful obvious muscle spasm is accompanied by extreme shortening of muscle and resultant posturing.

VI. X-Rays
X-rays are not indicated.

VII. Special Studies
Muscle biopsy and enzymes with unusual predisposition. An elevated creatine kinase–muscle band (CK–MB) is expected after prolonged endurance sports. This is due to protein leakage from damaged muscle fibers and does not indicate a diagnosis of a myocardial infarction.

VIII. Diagnosis
Muscle cramp(s) location _____.

IX. Differential Diagnosis
Strain, heat illness, disc syndrome, and electrolyte abnormality.

X. Treatment/Rehabilitation
Athlete should rest and maintain muscles in extended position. Active contraction of the antagonist of the cramped muscle is useful in reducing muscle spindle and motor neuron activity through reflex inhibition. Massage and stretching of involved muscles has no evidence-based efficacy in treatment, although many athletes consider this helpful. Most muscle cramps will resolve within 30 minutes, regardless of the treatment given.

XI. Complications
Strain or fall from loss of muscle function.

XII. Prevention
A. Progressive conditioning
B. Adequate fluid replacement (see Chapter 11)
C. Acclimatization

BURSITIS

I. Epidemiology[3,4]
Bursitis accounts for 0.9% of all visits to primary care physicians; it is a very common inflammation associated with repetitive activity (e.g., throwing, rowing, running, swimming).

II. Mechanism of Injury
Acute trauma or repetitive extremity movement irritates the bursae. This trauma may be to nearby cartilage or bone or directly to the bursae sac. Inflammation causes increased collagen production, leading to thickening of the bursae sac, increased fluid production, and increased capillary permeability, causing fluid and proteinaceous exudates to enter the bursae. Chronic inflammation leaves irregular areas of scar tissue, and blood may fill the bursae sac.[3,4]

III. Anatomy
A bursa is a closed sac lined with a synovia-like membrane and containing fluid. Bursae are found or formed in areas subject to friction (e.g., shoulder, elbow, hip, knee). Recurrent trauma leads to inflammation, calcification, adhesions, and infection.

IV. Symptoms
Localized pain over bursa and increased pain with use of area muscles and tendons.

V. Signs
Point tenderness over bursa, associated signs of inflammation, and decreased ROM.

VI. X-Rays
X-rays are usually unremarkable but may show some edema; calcifications are seen with chronic bursitis.

VII. Special Studies
A. Bone scan may indicate area inflammation.
B. Computed tomography or MRI may reveal enlargement, thickening, or calcification of bursa.[7–9]
C. Thermography is usually suggestive of inflammation when bursa is inflamed.

VIII. Diagnosis
Bursitis location _____.

IX. Differential Diagnosis
Tendinitis, myositis, sprain, strain, occult tumor or cancer, inflammatory arthritis, or cellulitis.

X. Treatment/Rehabilitation

A. Initially: Use RICE, NSAIDs, aspiration, compression, and padding; analgesics as needed. Steroid injections may be used (see Chapter 27).
B. Long-term: Use ROM and PREs within the limits of pain; modify exercise prescription.

Consultation: chronic calcific bursitis that does not respond to physical therapy and injections

XI. Complications

Bursa rupture or calcification, muscle atrophy.

XII. Prevention

A. Develop adequate, balanced muscle power.
B. Use proper equipment and technique to reduce localized repetitive loads (e.g., orthotics).
C. Follow a graduated training program.

CONCUSSIONS

I. Epidemiology[35,36]

Concussions or mild traumatic brain injuries (MTBIs) are the most frequently encountered traumatic head injuries that present in a primary care setting. Concussions include any trauma-induced alteration in mental status, with 300,000 sports-related concussions per year being reported in the United States; most common among individuals 15 to 30 years old, with twice as many men as women.

II. Mechanism of Injury

Diffuse injury is due to shearing forces produced by acceleration/deceleration with angular rotation (diffuse axonal injury). Possible indirect brain injury can be the result of trauma to torso or axial skeleton, with a secondary effect of impaired cerebral autoregulation. A second blow to a patient with impaired vascular regulation results in a rapid rise in intracranial pressure and cerebral edema (second-impact syndrome; see Chapter 37).

III. Anatomy

Diffuse brain injury is traditionally diagnosed with brainstem involvement.

IV. Symptoms

Headache and fatigue; witnesses are most valuable for history and diagnosis.

V. Signs

Alteration of consciousness, disturbance of vision or equilibrium, suppression of reflexes, photo/phonophobia. Symptoms occur immediately following traumatic head injury and last variable periods of time (seconds, minutes, hours, or days); Glasgow coma scale score of 13 to 15.

VI. X-Rays

Little benefit is obtained from skull radiographs.

VII. Special Studies

Special studies are not indicated due to normal findings on CT and MRI. Use CT only for patients with prolonged loss of consciousness or neurologic deterioration and MRI only to exclude more severe intracranial pathologic conditions. Extensive abnormal findings lead to different diagnosis.

VIII. Diagnosis

Concussion grade_____. Concussion grading is controversial and complicated by the fact that multiple grading systems exist; Chapters 36, 37, and 61 detail commonly employed concussion guidelines.

IX. Differential Diagnosis

Cerebral contusions, epidural/subdural hematomas, or cranial vault injury.

X. Treatment/Rehabilitation

Standardized assessment of concussion (SAC) evaluates orientation, immediate and delayed memory, and concentration. A neurologically stable patient with no deterioration is managed as an outpatient with a reliable observer. The observer (parent, spouse, roommate) should be instructed on how to perform a serial neurological exam, paying particular attention to changes in mental status, worsening headache, vomiting, or visual deterioration. Allow return to play only when patients is asymptomatic both at rest and with exertion. Repeated concussions require a longer period of asymptomatic rest (see Chapters 37 and 61).

XI. Complications

A. Prolonged loss of consciousness, amnesia, or focal neurological deficits indicating extensive craniocerebral injury
B. Second-impact syndrome
C. Convulsions (antiepileptic therapy not indicated)

D. Postconcussive syndrome
E. After sustaining initial concussion, increased risk for additional concussions

XII. Prevention

A. Use of properly fitting headgear in contact sports.
B. Knowledge of proper heading technique in soccer.

FATIGUE

A common presenting sports problem, fatigue occurs from overtraining, nonanemic iron deficiency, depression, and a variety of illnesses (e.g., subclinical mononucleosis, hepatitis, chronic fatigue syndrome). It is typically seen in female distance runners and male runners, as well as rowers and swimmers. The history should focus on the description and timing of fatigue (e.g., is it present with athletic activity or throughout the day?), training history, dietary history, school- and home-related stress, and drug or alcohol use. A minimal exam consists of the cardiorespiratory, neck (including thyroid), HEENT, and additional specific areas requiring elaboration. The laboratory evaluation includes CBC, thyrotropin, and a ferritin level. Spirometry should be considered if asthma is suspected. A variable period of rest is usually indicated in terms of overtraining. A course of iron supplementation (324 mg p.o. daily for 6 weeks) is appropriate for nonanemic iron deficiency. Medication and counseling are appropriate for the anxious or depressed athlete. An appropriate period of convalescence is essential for acute illnesses.

SPORT-SPECIFIC INJURY

Sports medicine in primary care requires heightened awareness of sport-specific injuries for patient education. Training and conditioning with injury-specific prevention in mind can eliminate many common injuries (see Table 34.5).[37] Sports with the highest incidence of sports-related injury include:[2]

TABLE 34.5
Incidence of Injury Sites by Sport

Sport	Injuries
Baseball and softball	Shoulder >> elbow, forearm, wrist, fingers > ankle, hip, back (softball has less injuries than back)
Basketball and volleyball	Ankle, knee >> hip, thigh > Achilles tendon, heel, thigh, back, shoulder, elbow, eye, wrist, fingers (girls' knee injury rate is three to four times that of boys)
Football	Knee > ankle > head, shoulder, neck > back, face, wrist, fingers
Ice hockey	Head, neck > ankle, knee, shoulder/arm > eye, wrist, hand
Field hockey	Knee, ankle > face, hand
Gymnastics	Shoulders > torso, back, hip, wrist > elbow > ankle, head, neck
Roller-blading	Distal forearm, wrist > lower leg > elbow > knee > head (roller skating has a similar but less severe injury profile; skateboarding has a similar profile but the injuries are more severe)
Snowboarding	Wrist > head >> upper extremity, shoulder, forearm > knee, ankle, hip, back
Snow skiing	Lower extremity, leg, knee >> head >> thumb, shoulder, ankle, wrist, back, neck, thigh
Soccer	Knee, ankle >> shin, foot, hip, thigh >> shoulders, cervical spine, head (knee injuries are three times more common in girls than in boys)
Swimming	Shoulder > knee > elbow, head, spine (diving)
Track and running	Knee, shin (shin splints) > foot, hip, back, thigh
Field	Knee, shoulder, hip, back > ankle, spine, elbow
Wrestling	Shoulder >> knee >> back, neck, foot, ankle, wrist, hand >> head, ear, torso, elbow

Note: >>, Higher incidence; >, lower incidence.
Source: Adapted from Luckstead, E.F. and Satran, A.L., *Pediatr. Clin. N. Am.,* 49(4), 753–767, 2002.

- *Group sports* — basketball > football > baseball or softball > soccer > volleyball, hockey, lacrosse.
- *Individual sports* — pedal cycling > ice or roller skating or skateboarding > gymnastics or cheerleading > playground > snow sports > water sports > exercising–track; combative > recreational

SUMMARY

Most sports injuries fall into several well-defined generic categories: overuse, abrasions, lacerations, sprains, strains, fractures, dislocations, and concussions. While acute trauma from a blow or fall is an obvious etiology, chronic insult from repetitive stresses in excess of the reparative process is more commonly the culprit and is responsible for the greatest amount of morbidity in the sports and recreational arena. The sports physician must be circumspect with regard to diagnosis and management. Biomechanical abnormalities and training errors must be corrected and the athlete encouraged to adopt aerobic fitness practices that do not substitute pain for progress. The long-term outlook is optimized by an adequate and aggressive rehabilitation process.

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35 SPORTS MEDICINE EMERGENCIES

E. James Swenson, Jr.

FIELD MANAGEMENT OF SPORTS MEDICINE EMERGENCIES

The majority of injuries resulting from participation in sports and fitness activities are minor sprains, strains, contusions, and abrasions that do not prevent continued participation. More severe injuries require the team physician to make a decision about the return to play, and the principles outlined in Chapter 36 provide objective guidelines for making such a decision. The purpose of this chapter is to discuss those rare sports-medicine emergencies that must be recognized and managed appropriately to save the life or limb of the athlete. A successful outcome results from an awareness and familiarity with each type of emergency and from adequate preparation in terms of skill, equipment, and other necessary resources. The contents of this chapter will serve a variety of individuals with varied backgrounds in many different circumstances. Sufficient depth is given so that individuals trained in the techniques can deliver definitive care to athletes in an emergency. It will often be the case that an ambulance with emergency personnel is available and that the certified athletic trainer (ATC) or physician will perform only the basic life support until the ambulance arrives and can take control of the emergency.

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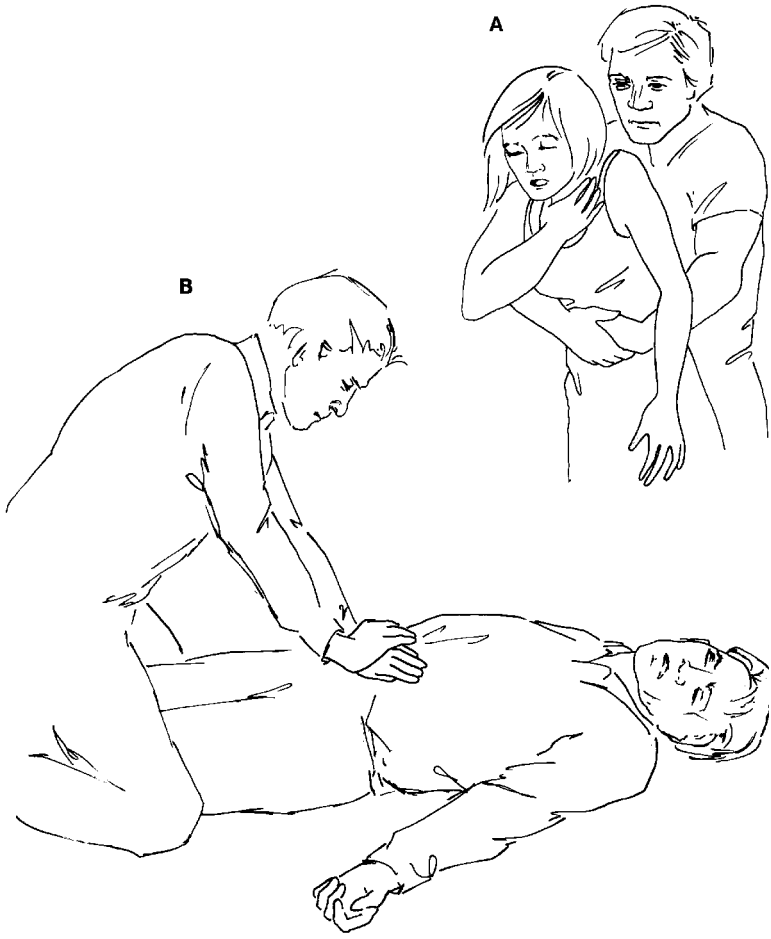

Figure 35.1 Heimlich maneuver to dislodge a foreign object from the airway. Repeated quick thrusts are applied, with the patient in the upright (A) or supine (B) position.

Basic Strategies and Principles

Outcome is contingent upon rapid, effective evaluation and intervention; therefore, adequate preparation and training are critical. A team leader (physician or ATC) must be identified who is appropriately trained (e.g., basic cardiac life support, or BCLS) and who is responsible for the on-field effort. An integrated system of ambulance transportation, telephone link-ups, necessary emergency equipment, and appropriately trained ancillary healthcare personnel must be coordinated, preferably through rehearsed injury scenarios done at least annually.

Assessment and Stabilization

Primary assessment and stabilization require coordinated, skilled, rapid evaluation and management based upon close serial observation (looking, listening, and feeling) using the mnemonic ABCDE:

Airway

- Check for spontaneous breathing.
- If absent, establish airway access and clear any obstruction (e.g., tongue or foreign body such as mouthpiece) (Figure 35.1).
- Stabilize C-spine — in-line immobilization with no traction, chin or jaw lift (Figure 35.2), or log roll (Figure 35.3). Do not remove helmet (if mechanical airway control is needed due to apnea, remove face mask for access). Do not use ammonia capsules.
- Provide supplemental oxygen.

Breathing

- Establish artificial ventilation with supplemental oxygen if no spontaneous breathing:
  - Mouth-to-mouth.
  - Mouth-to-mask (to reduce risk of infectious diseases).

Figure 35.2 Jaw thrust maneuver to open airway of patient who is unconscious and/or may have C-spine injury. From a position above the patient's head, the fingers are placed behind the mandible, lifting and displacing the jaw forward. An oral airway can then be inserted by retracting the lower lip. (Modified from Simon, P.R. and Brenner, B.E., *Emergency Procedures and Techniques*, Baltimore, William & Wilkins, 1985, p. 35.)

  - Bag-valve-mask (to reduce risk of infectious diseases).
  - Cricothyrotomy — indicated in situations of (1) emergent airway compromise and the above methods are either contraindicated or unsuccessful, or (2) massive facial trauma. Use 14-gauge catheter over needle puncture (Figures 35.4 and 35.5).
- Check respiratory status (e.g., rate and difficulty if spontaneous, symmetry). If tachypnea, dyspnea, or asymmetry is detected, consider:
  - Pneumothorax (tension, open, closed)
  - Hemothorax
  - Flail chest
  - Pulmonary contusion
  - Tracheobronchial injury

Circulation

- Check pulse.
  - Systolic BP is approximately 60 mmHg if carotid pulse is present.
  - Systolic BP is approximately 80 mmHg if radial pulse is palpable.

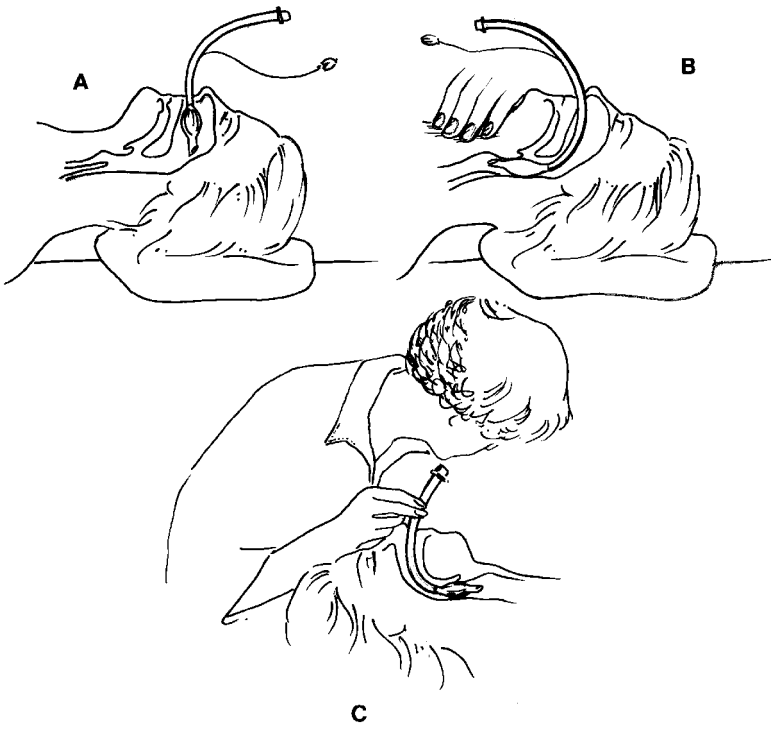

Figure 35.3 When a C-spine injury is possible, the athlete may be log-rolled into a supine position after the neck is placed in a neutral position. (Courtesy of AirCast Corp., Summit, NJ.)

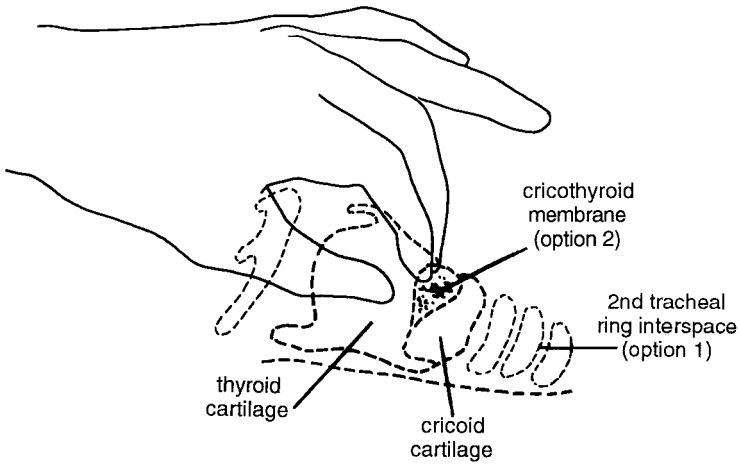

Figure 35.4 Laryngeal anatomy. "Option 1" and "Option 2" refer to treatment of patients with laryngeal trauma, as detailed on Table 35.5. (Courtesy of AirCast Corp., Summit, NJ.)

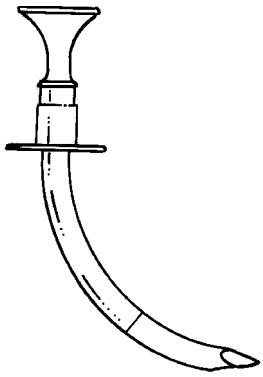

Figure 35.5 Abelson emergency cricothyrotomy cannula, used to relieve upper airway obstruction in patients with laryngeal injury. (Courtesy of AirCast Corp., Summit, NJ.)

- Begin cardiopulmonary resuscitation if pulse is absent, and follow advanced cardiac life support (ACLS) algorithms.
- Check capillary blanch test if pulse is present but weak (>2 seconds required for color return suggests shock state) (Table 35.1).
- Check for hemorrhage sites and apply direct pressure.
- Consider cardiac tamponade for a chest injury with: (1) shock, (2) distended neck veins, (3) muffled heart sounds, or (4) narrowed pulse pressure.
- Monitor cardiac rate and rhythm with electrocardiogram (EKG).

Disability

Perform a serial mini neurologic exam. Record findings utilizing approved grading system (Table 35.2).

Exposure

Remove equipment/clothing and carefully inspect, palpate, percuss, and auscultate entire body for injury sites.

IMMEDIATE CATASTROPHIC SPORTS MEDICINE EMERGENCIES

Sports medicine emergencies are divided into two groups: *immediate catastrophic* (Table 35.3) and *delayed onset* (Table 35.4).

Sudden Death[1–5]

Sudden death occurs occasionally in officials and fans at sporting events but rarely in athletes. In officials, fans, and athletes over 35, the cause is almost always atherosclerotic coronary artery disease. In cases under 35 (10 to 25 per year), the common causes of sudden death are hypertrophic cardiomyopathy, congenital coronary artery anomalies,[6] and, less frequently, valvular insufficiency, commotio cordis,[7–9] arrhythmogenic right ventricular dysplasia (ARVD),[10] or other conduction system disorders[10,12] and aortic rupture from Marfan's syndrome.[11] A key is prevention through appropriate identification of susceptible individuals using risk factor analysis when possible. In athletes under 40, the appropriate historical questions and a careful physical and cardiac exam may pick up Marfan's syndrome, hypertrophic cardiomyopathy, and valvular insufficiency, while conduction system disorders and coronary artery anomalies are difficult to identify. The fact remains that a chance exists that someone at an athletic event will suddenly collapse. For management, see Cardiac Arrest section below.

TABLE 35.1
Estimated Localized Blood Loss (Liters) from Adult Fractures

Pelvis	1.5–4.5
Hip	1.5–2.5
Femur	1.0–2.0
Humerus	1.0–2.0
Knee, tibia, ankle, elbow, forearm	0.5–1.5

Respiratory Arrest

Respiratory arrest can occur in both contact and non-contact athletic activities. Management depends on the specific etiology (Table 35.5).

Cardiac Arrest (Ventricular Fibrillation)

The management of cardiac arrest must follow the latest guidelines of the American Heart Association. After assessment of the airway, breathing, and circulation (ABC), cardiopulmonary resuscitation (CPR) is initiated as necessary. Early (within 5 minutes) on-scene electrical counter-shock of ventricular fibrillation is mandatory and can now be available with the use of automated external defibrillators (AEDs).[13]

Myocardial Infarction (MI)

Any middle-aged or older recreational athlete who presents complaining of precordial chest pressure or tightness with radiation to the left

TABLE 35.2
Glasgow Coma Scale

Best motor response		
To verbal command	obeys	6
To painful stimulus	localizes pain	5
	flexion — withdraws	4
	flexion — abnormal (decerebrate)	3
	extension (decerebrate)	4
	no response	1
Best verbal response		
Arouse patient with painful stimulus if necessary	oriented and converses	5
	disoriented and converses	4
	inappropriate words	3
	incomprehensible sounds	2
	no response	1
Eyes		
Open	spontaneously	4
	to verbal command	3
	to pain	2
	no response	1
	TOTAL	3–15

Note: Lowest score is 3; highest is 15; score of 7 or less indicates coma; falling score over short period indicates a progressive acute injury requiring immediate definite action.

TABLE 35.3
Immediate Catastrophic Sports Medicine Emergencies

Sudden death
Respiratory arrest
Cardiac arrest
Myocardial infarction
Commotio cordis
Anaphylactic shock
Near drowning
Heat stroke/hyperthermia

TABLE 35.4
Delayed-Onset Sports Medicine Emergencies

Head injury
Neck injury
Abdominal injury
Genital injury
Seizures
Acute compartment syndrome
Acute elbow dislocation
Acute knee dislocation
Acute fracture care

jaw or shoulder represents myocardial ischemia (MI) until proven otherwise. If the pain is unrelieved by nitroglycerin, infarction is suspected. Exercise-related diaphoresis and shortness of breath may confuse the diagnosis. Low blood pressure, the presence of an S3 heart sound, and moist rales at the base of the lungs indicate compromised left ventricular function. Any athlete, coach, or fan suspected of having an acute MI should be transported to the hospital for immediate evaluation. The initial treatment of an MI is reasonably standard, with only minor differences in protocol from one hospital to the next. Early administration of thrombolytics has become a mainstay of treatment and has significantly reduced mortality.

TABLE 35.5
Respiratory Arrest in the Athlete

Etiology	Specific Examples	Signs/ Symptoms	Physical Exam	Initial Treatment	Emergency Equipment or Technique
Upper airway obstruction (may be complete or incomplete)	Loss of consciousness: In supine position with neck flexed, obstruction results from tongue occluding oropharynx	Absent or diminished airflow from mouth and nose in spite of exaggerated respiratory efforts	Tachycardia; absent or diminished breath sounds in spite of intercostal, suprasternal retractions; cyanosis	Unconscious athlete and/or if C-spine injury possible: Place in supine position and establish airway by forward jaw thrust without neck tilt (Figure 35-2)	Jaw thrust maneuver; oropharyngeal or nasopharyngeal airway
	Foreign body such as gum or mouthpiece lodged in oropharynx, most often as result of trauma	Conscious athlete may give "choke" sign, with inability to talk, inability to breathe. May include thrashing about, cyanosis	Snoring, stridor, or wheezing may occur with partial obstruction. Care must be taken not to push the object further down	Incomplete obstruction moving adequate air (if conscious and no C-spine injury): Place in sitting position with neck extended and transport to ED	Magill forceps for removal of foreign body
				Complete obstruction or inadequate ventilation (from foreign body aspiration): Heimlich maneuver (Figure 35-1)	Heimlich maneuver
	Maxillofacial trauma may result in teeth or blood occluding airway	See above	Oral exam; absent teeth, blood occluding airway	See above for complete or incomplete obstruction	Oral suctioning equipment
	Direct trauma may crush larynx, preventing airflow	See above	Swelling, ecchymosis, deformity of anterior upper neck. Possible subcutaneous emphysema. Hoarseness stridor	A. If complete obstruction *Option 1:* Enter trachea at level of 2nd or 3rd tracheal ring with scalpel, being careful not to perforate esophagus (see Figure 35-4), insert a temporary rigid airway (e.g., pen barrel) and secure with tape	Scalpel, rigid endotracheal (ET) tubes (pen barrel), adhesive tape

TABLE 35.5 (CONTINUED)
Respiratory Arrest in the Athlete

Etiology	Specific Examples	Signs/ Symptoms	Physical Exam	Initial Treatment	Emergency Equipment or Technique
				Option 2: Use commercial cannula which consists of 7-gauge curved trocar with a sharp tip that is pushed through the cricothyroid membrane. The tip is then retracted and the plastic cannula advanced.[30]	Abelson emergency cricothyreotomy cannula[30] (see Figure 35-5)
				B. If incomplete obstruction follow above plan. ENT referral for controlled airway evaluation (laryngoscope) and control (tracheostomy)	
	Laryngospasm from anterior neck trauma, or reflexively with traumatic stimulation of visceral nerve endings in pelvis, abdomen, or thorax	See above	Contours of larynx normal, no subcutaneous air	Force the chin forward by strong pressure behind the angles of the jaw (see Figure 35-2). When possible, all patients with laryngeal injury should be referred for immediate laryngoscopy[30]	Jaw thrust maneuver
Acute respiratory failure; $pO_2 \leq 50$ mmHg; hypoxia; hygercapnia	Exertional syncope associated with prolonged or intense exercise (most often in unconditioned individuals) [26,31]	Little or no respiratory effort. Exertional syncope usually brief, but if prolonged, convulsions may result	Brief loss of consciousness. Tachycardia; breath sounds absent or diminished; increasing cyanosis. Temp <39° C (rectal). May be hypotensive pale	Establish airway by tilting head back and opening jaw, insertion of oropharyngeal airway. If breathing does not immediately resume, begin mouth-to-mouth resuscitation	Oropharyngeal airway, BCLS

	Craniocerebral or spinal cord injury	Transient paralysis or oropharyngeal musculature or respiratory arrest	See above	Establish airway by ("blind") nasotracheal insertion if still breathing (see Figure 34-3), otherwise endotracheal intubation with in-line immobilization	Endotracheal tubes
	Fractured ribs, flail chest	Acute respiratory distress	Paradoxical breathing (flail segment out with expiration and in with inspiration)	Stabilization of flail fragment with hand or sandbag; immediate transport to hospital	
	Drug-induced;[32] cocaine, narcotics	Little or no respiratory effort	Tachycardia	Ventilation, transport to ED	Ambu®Bag (BCLS)
	Asthma	Respiratory distress	Tachycardia, tachypnea, poor air exchange, wheezing with intercostal retractions	Epinephrine 1:1000, 0.3–0.5 cc SQ, Rpt 20 min.[33] Transport to ED. Inhaled beta-agonists if available	Epinephrine 1:1000, needle syringe, beta-agonists (Ventolin®, Proventil®)
Pneumothorax	Traumatic, may be secondary to rib fracture	Sudden-onset chest pain, dyspnea; may have referred pain to shoulder	Tachypnea, tachycardia. May have decreased breath sounds, hyperresonance to percussion, decreased fremitus on involved side	Transport to hospital for X-rays, possible chest tube	Stethoscope
	Spontaneous: young, tall, thin individual. May be associated with exertion. (Progression to tension pneumothorax rare.[34])	As above. 5% have no symptoms	As above	X-ray evaluation	Stethoscope
	Tension pneumothorax[18]	Acute respiratory distress, nausea, shock	Will have above findings; tachycardia, distention of neck veins, syncope, hypotension	Insertion of 14-gauge needle into 2nd ICS at mid-clavicular line; should hear air release	14-gauge catheter over needle

TABLE 35.5 (CONTINUED)
Respiratory Arrest in the Athlete

Etiology	Specific Examples	Signs/ Symptoms	Physical Exam	Initial Treatment	Emergency Equipment or Technique
Pulmonary embolism[33,36]	Traumatic: deep venous thrombosis (DVT) in lower extremities	Tachypnea, dyspnea, pleuritic pain, apprehension, cough. Hemoptysis (28% only)	Tachypnea, tachycardia, rales over involved area	Oxygen; transport to hospital	Ambu®Bag, oral airway
	Spontaneous	May present with syncope, shock	Signs of DVT: swelling, tender extremity	As above	
High-altitude pulmonary edema (HAPE)[37]	Occurs in 1–2% of healthy individuals of any age traveling above 10,000–11,000 ft. (Prevent by proper acclimatization.)	(1) Headache, insomnia, dyspnea, lightheadedness, confusion, nausea, malaise, fatigue; then (2) chest pain, cough, confusion, leghargy, profound dyspnea, orthopnea, death	Tachypnea, tachycardia, fever, cyanosis, rales	Rapid descent minimum 1000–2000 ft. Oxygen therapy	Oxygen source: respiratory

Note: BCLS = basic cardiac life support, ED = Emergency Department, ICS = intercostal space, NTG = nitroglycerin, RPt = repeat, SQ = subcutaneous.

TABLE 35.6
Treatment of Anaphylactic Shock

Etiology	Signs/Symptoms	Initial Treatment	Required Equipment	Prevention
Hymenoptera[38] allergy (most common, yellow jacket)	Local urticaria, bronchospasm, angioedema wheezing, SOB, dyspnea, hypotension	SQ injection, 0.5 cc 1:1000 epinephrine O_2 therapy as available Inhaled beta-agonists if available	Syringe, needles, 1:1000 epinephrine (Anakit®)	Identify at preparticipation physical. Carry "Bee sting" kit, med alert tag
Exercise-induced anaphylaxis;[5,6] age 12–54 years Exertion: running, tennis, racquetball, basketball; possibly food related	Prodrome: fatigue, warmth, itching Early: urticaria Late: headache, dizziness, hypotension; may have wheezing, nausea, vomiting, colic	SQ 0.5 cc 1:1000 epinephrine Inhaled beta-agonists if available	As above (Anakit®)	Run with buddy. Have Anakit® available Avoid sensitivity-producing foods, celery, shrimp Avoid eating within 2 h of exercise and stop workout if prodromal signs occur

Note: SOB = shortness of breath, SQ = subcutaneous.

Commotio Cordis[7–9]

Cardiac injuries are rare in sports events but can occur in such collision or contact sports as baseball, football, and race car driving. Pitchers hit by a batter's ball and batters hit by a pitched ball while attempting to bunt are the most frequent victims. Sudden death has resulted rarely from these events. Autopsy findings indicate that the impact of the ball resulted in no detectable myocardial damage and only sometimes a mild, superficial contusion of the pericardium. Commotio cordis may be defined as "instantaneous cardiac arrest produced by nonpenetrating chest blows in the absence of heart disease or identifiable morphologic injury to the chest wall or heart." Case reports of sudden death indicate an initial traumatic apnea, most likely associated with ventricular fibrillation or asystole. Initial treatment includes CPR and evaluation of the cardiac rhythm, followed by administration of the appropriate antiarrhythmic drugs and early countershock for ventricular fibrillation. Mortality can be lowered with the use of AEDs.[13]

Anaphylactic Shock[14]

The preparticipation physical examination is the time to identify athletes with a significant bee sting allergy. Exercise-induced anaphylaxis (EIA) been described in the literature (Chapter 60). No fatalities from EIA have been reported to date but the potential does exist. Table 35.6 provides an outline of the appropriate treatment to be used for anaphylactic shock.

Near Drowning

Remember that the apparent duration of submersion, especially in cold water, is an unreliable guide to the condition of the victim. Ventilate the victim's lungs as soon as possible using as much oxygen as possible. If no pulse is present, begin chest compressions. Continue CPR during transport to the hospital. Remember the dictum that, "A patient is never dead until warm *and* dead."

Heat Illness/Heat Stroke[15,16]

Adequate conditioning, acclimatization, and hydration during an event are the keys to preventing heat illness. It is important to remember that, especially in distance runners, the sweating mechanism may be operational during the initial presentation of heat stroke. An additional key is to take a rectal, not oral, temperature if heat injury is suspected, as the oral temperature can be normal while the rectal temperature exceeds 106°F. Treatment consists of immediate removal of most clothing and application of iced or cool water. No time should be lost initiating body cooling measures, which must continue en route to the treatment center.

DELAYED-ONSET SPORTS MEDICINE EMERGENCIES

Head Injuries[17]

Head injuries are possible in many sports but occur most often in football, hockey, and boxing. The most serious head injuries cause increased intracranial pressure, such as subdural or epidural hematoma (see Chapter 37). These injuries are most often but not always associated with immediate loss of consciousness (LOC). LOC is usually greater than 5 minutes and in 20% of cases (epidural and subdural hematomas) is associated with a lucid interval prior to a relapse into a decreased level of consciousness. In general, prolonged post-traumatic amnesia (PTA) is a more sensitive marker of significant head injury than brief LOC. The key to a successful outcome is safe and rapid transport to a facility where neurologic evaluation and intervention can take place.

On the field, the unconscious athlete is assumed to have a cervical spine injury. Initially, the airway must be evaluated and established. It may be necessary to perform a jaw thrust to open the airway safely in the unconscious athlete (see Figure 35.2). C-spine injury above the level of C5 results in paralysis of the diaphragm and requires mouth-to-mouth resuscitation.

An on-field, mini-neurologic exam, including C-spine and cranial evaluation, is performed. The neurologic exam includes questions regarding any numbness, tingling, or weakness of the extremities. The ability of the athlete to move hands and feet, the athlete's level of consciousness, and the pupils are next assessed. A more detailed examination can take place on the sideline, to include cranial nerves, cerebellar function, and presence of an abnormal plantar reflex. In cases of any neurologic deficit or evidence of skull fracture, the athlete should be immediately transported for neurologic evaluation. If a fully equipped medical facility is not readily available, the physician must be prepared to establish an airway, hyperventilate the patient, start an IV, and administer 20% mannitol (1.0 g/kg) (if impending herniation) for a suspected serious head injury. For the athlete with known structural damage to the brain each case must be evaluated individually to assess whether to return to contact sports in spite of a full recovery.

Neck Injuries[18]

Spinal cord injury, most often a fracture dislocation of C5 on C6, is a result of axial loading of the C-spine in slight flexion. Few fatalities result from neck fractures, and not all neck fractures result in paralysis. Improper transport technique, however, can cause paralysis. It has been estimated that between 10 and 25% of permanent neurological deficits occur after the initial accident. The most common etiologies include diving into unknown waters (often associated with alcohol use), and team sports such as football or hockey. Injury occurs in football when the defensive backs tackles the ball carrier with his head down or in hockey when the player is checked into the side of the rink. Table 35.7 provides guidelines for the assessment of the conscious and unconscious athlete suspected of having a neck injury.

TABLE 35.7
Management of C-Spine Injury

Unconscious Athlete
Assume neck injury.
Place in supine position with head and neck stabilized.
If present, remove face mask. (Do NOT remove helmet.)
Check airway; use jaw thrust and insert airway as needed.
Check pulse; begin CPR as needed.
Check pupils.
Transport on spine board with head and neck stabilized.
Conscious Athlete
Ask athlete: Do you have any neck pain? Do you feel any numbness, tingling, or burning sensation? Can you move your fingers and feet?
Physical exam: palpate C-spine.
If above are positive, transport from field on spine board.
If above are negative, ask athlete to put neck through a voluntary ROM.
If above are satisfactory, athlete can stand and walk off the field.
If any findings are in question, transport athlete from field on spine board.

It is important when transporting an athlete with a suspected neck injury to always maintain control of the cervical spine. Initially, the neck, if rotated, is moved into a neutral position by gentle rotation and in-line traction, and the athlete, if lying prone, is log rolled into a supine position (Figure 35.6). If possible, a rigid cervical collar is fitted. All subsequent movement is done with the neck in the neutral position.

For respiratory arrest, the jaw thrust method for opening the airway prevents further injury to

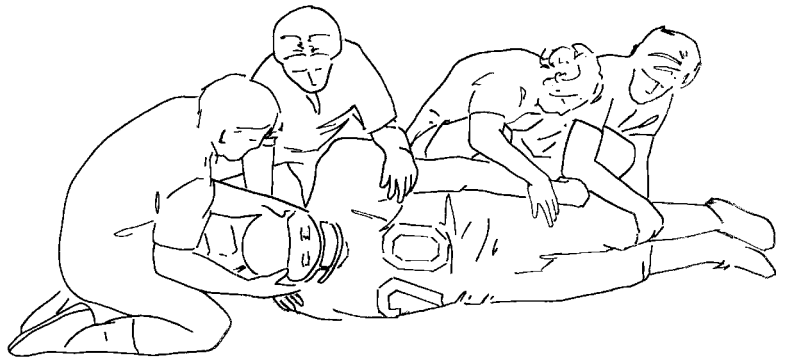

Figure 35.6 Nasotracheal intubation. (A) With the patient in the supine position, the neck is protected in slight flexion as the nasotracheal tube is inserted. (B) The tube is rotated as it passes the nasopharynx into the orphopharynx. The larynx is posteriorly displaced. (C) Advance the tube while listening for maximum breath sounds. The tube is then passed rapidly during the beginning of inspiration. (Modified from Simon, P.R. and Brenner, B.E., *Emergency Procedures and Techniques*, Baltimore, William & Wilkins, 1985, p. 65.)

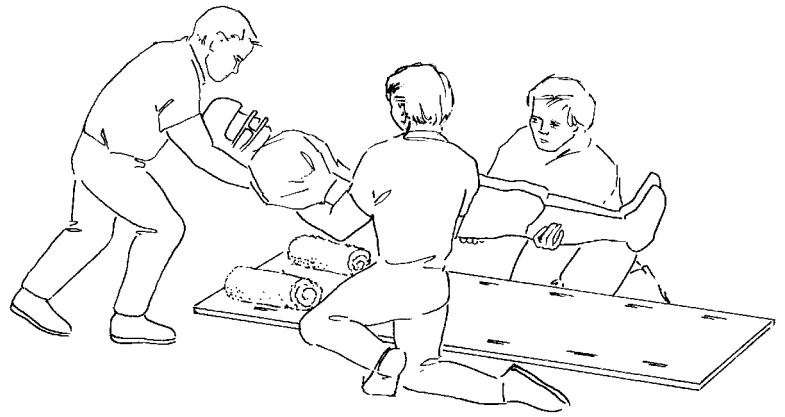

Figure 35.7 Proper method of airlifting onto a spine board. Participation by 5 to 7 individuals may be preferable. (Courtesy of AirCast Corp., Summit, NJ.)

the cervical spine and spinal cord (see Figure 35.2). The helmet should never be removed, but bolt cutters or a knife may be used to remove all obstructive athletic gear (e.g., face mask). If impending respiratory arrest persists, following the jaw thrust, a "blind" nasotracheal intubation (see Figure 35.6) is carried out to establish the airway without risking additional injury to the spinal cord. In the apneic patient, endotracheal intubation with in-line immobilization should be utilized. In the case of cardiac arrest, CPR is initiated as soon as the individual has been placed into the supine position with the neck stabilized.

Following airway and cardiac assessment and stabilization, the spine board is then placed beside the athlete. At least three (preferably five to seven) individuals assist with transportation. With one to three helpers on either side to support the trunk and limbs (multiman carry), the physician controls the head and neck by cradling the head and shoulders between his forearms while clasping the scapula and trapezius (or shoulder pads, if present) and gives the commands to lift and place on the spine board (Figure 35.7). Once the individual is placed on a spine board, appropriate sand bags, taping, and supportive strapping are used to secure the patient (Figure 35.8). Rapid transportation to an appropriate neurosurgical facility occurs only after optimal immobilization.

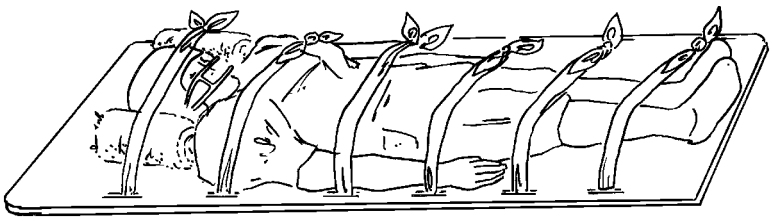

Figure 35.8 Stabilization on the spine board. Sand bags or towels around the neck and strapping adhesive tape are used to secure the head, neck, body, and extremities firmly. (Courtesy of AirCast Corp., Summit, NJ.)

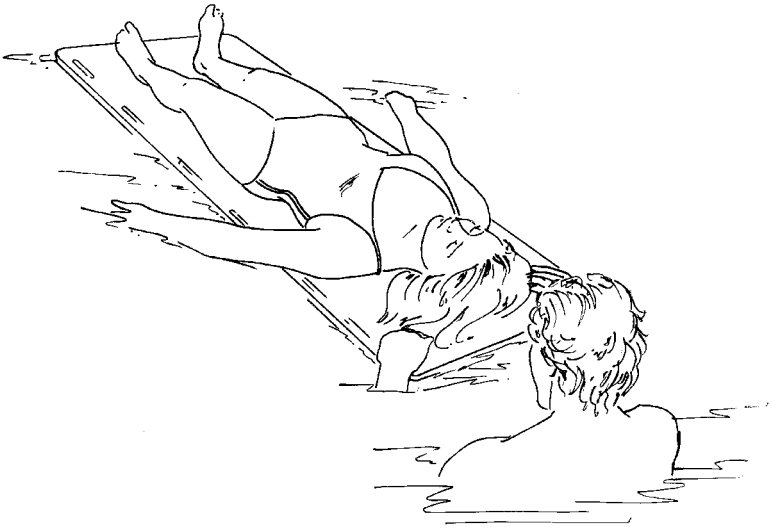

Figure 35.9 Management of a C-spine injury in water. A spine board is floated under the patient. (Courtesy of AirCast Corp., Summit, NJ.)

When a suspected cervical spine injury takes place in the water, a spine board is floated underneath the individual prior to transport from the water (Figure 35.9). Again the individual is secured to the spine board with appropriate sand bagging or towels as well as straps and/or adhesive tape as needed. Transport to the emergency facility should occur as soon as possible. Evaluation in the emergency department (ED) includes routine C-spine films and, if negative, patient-assisted flexion and extension views, looking for cervical subluxation. Appropriate orthopedic or neurologic consultation follows any positive findings.

Abdominal and Renal Injury[19-23]

(See Chapter 43.) The initial exam is not always reliable, as a serious injury may have few initial findings. Pain may be present in the left or right upper quadrant in the case of a spleen or liver injury, respectively. The majority of spleen injuries tend to be brisk bleeders, but the remainder are slow bleeders; therefore, symptoms of shock may be dramatic from the onset or occur more slowly over several hours. Rebound and guarding are often delayed. With any suspicion of abdominal injury, the athlete is denied fluids to drink, and serial vital signs and abdominal exams are monitored. With signs of shock, the athlete is placed in a Trendelenburg position, medical antishock trousers (MAST) applied, and emergency transport arranged. Two or more large-bore IVs are started as soon as possible. Definitive diagnosis is made by magnetic resonance imaging (MRI). Early surgical consultation is advised.

Genital Injuries[24,25]

A severe blow to the scrotum, such as from a kick or straddle injury, can result in testicular dislocation, rupture, or a less severe contusion or hematocele. Testicular dislocation can occur in the inguinal, crural, or perineal regions and is associated with testicular swelling and scrotal hematoma. If closed reduction is not possible, a urologist is consulted for surgical relocation and orchioplexy. Testicular rupture also presents with swelling and hematoma. Little pain may be noted. The diagnosis can be made with ultrasound; early repair increases survival of the testicle. Transillumination also assists with the diagnosis if major swelling makes palpation difficult. Aspiration is of little diagnostic value. The differential diagnosis must include torsion.

Seizures[26]

Although uncommon, a grand mal seizure can result from head trauma in contact or collision sports. A seizure also can occur in the athlete with a known seizure disorder, precipitated by hyperventilation, contact, or a subtherapeutic level of medication. In any case, emergency management is the same: maintenance of an open airway and prevention of self-injury. Prolonged seizures (greater than 2 minutes) are treated with IV diazepam 5 to 10 mg, phenytoin 50 mg/min up to 1 g (15 to 18 mg/kg), or phenobarbital 150 to 200 mg.

Acute Compartment Syndromes[27,28]

Blunt trauma or severe contusion to the leg may cause an acute compartment syndrome with permanent damage to nerve and muscular tissue. The anterior compartment is the most common site. Increasing pain associated with an extremely tense anterior compartment, with or without paresthesias or foot drop, should be evaluated with compartment pressures in the ED. Compartmental pressure in excess of 30 mmHg requires immediate surgical decompression.[27]

Acute Elbow Dislocation[29]

The majority of elbow dislocations are posterior and result from an abduction and hyperextension force as the athlete (most commonly 10 to 25 years old) falls on the outstretched arm. The olecranon process applies leverage against the olecranon fossa, and the coronoid process slips posteriorly and comes to rest in the olecranon fossa. The anterior capsule, medial collateral ligament, and brachialis muscle may be torn and may be accompanied by an avulsion fracture of the coronoid process. Diagnosis can be made from clinical presentation (e.g., deformity, limited range of motion, crepitus) and must be distinguished from a supracondylar fracture.

It is important to assess and record neurovascular status beginning at the initial exam. Closed reduction is possible in most cases. The sooner after the injury the reduction is attempted, the easier the reduction will be. The reduction should always involve gentle manipulation. A simple method of reduction involves countertraction on the humerus by an assistant with simultaneous distal traction on the wrist and proximal forearm (Figure 35.10). After disengaging the coronoid process and radial head from their positions behind the distal humerus by forearm supination, the elbow is gently flexed to 70 to 80°. Following the reduction, a neurovascular assessment is repeated. The elbow is iced and placed in a sling for transport to the hospital or orthopedic office.

If the reduction is not accomplished soon after the injury, swelling and pain will make the procedure more difficult and local anesthesia may be required. Under these circumstances, the elbow should be splinted in a position of comfort and the athlete immediately transported to the ED.

Acute Knee Dislocation[30]

Dislocation of the knee is a rare injury, and immediate orthopedic referral is required. A history involving major joint trauma and global laxity on clinical exam are the keys to diagnosis. Spontaneous reduction is the rule, although an on-site reduction may occasionally be necessary. Anterior dislocation of the tibia relative to the femur is most common. Reduction is accomplished by axial distraction on the tibia followed by gentle extension. A "clunk" can be expected as the tibia falls back into place. Vascular injury to the popliteal artery or a traction injury to the posterior tibia may be present and can compromise the viability of the lower leg if referral is delayed. A neurovascular exam is performed initially, and serial measurements should continue en route to the definitive care facility. The knee can be splinted with a straight leg immobilizer for symptomatic relief.

Acute Fracture Care

Serious fractures do occur in sports and can result in neurovascular compromise. Fractures of the long bones, including femur, the humerus, or the bones of the lower leg or forearm, present with

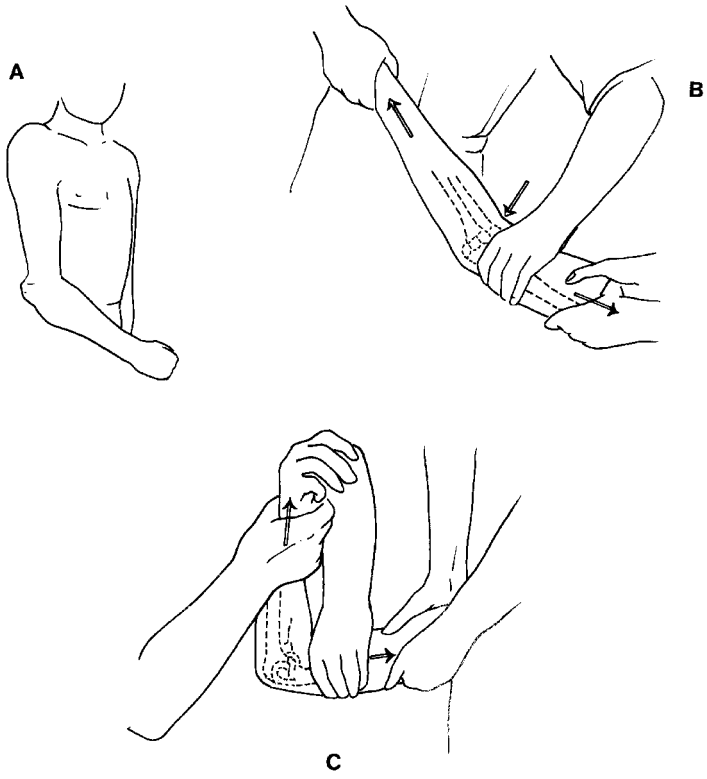

Figure 35.10 Reduction of a posterior elbow dislocation. (A) Typical deformity. (B) An assistant provides countertraction while traction is applied to the forearm. (C) Gentle flexion follows reduction. Traction is maintained on the upper arm. (Courtesy of AirCast Corp., Summit, NJ.)

obvious pain, swelling, positive osteophony, and possible deformity. Neurovascular status should be noted and followed. Icing, splinting, and transport to an acute care facility are essential. While commercial splints such as air splints are available, it is quite possible to provide adequate immobilization with wooden splints placed on opposite sides of the extremity and secured with tape or appropriate pieces of cloth if necessary (Figure 35.11).

SUMMARY

True sports medicine emergencies are infrequent. The physician must be prepared to recognize and provide emergency care for a variety of injuries. It is also important to know about the available resources such as ambulances, emergency transport, and those facilities and personnel that could provide the needed care. Knowing he or she is prepared will allow the physician to enjoy the game.

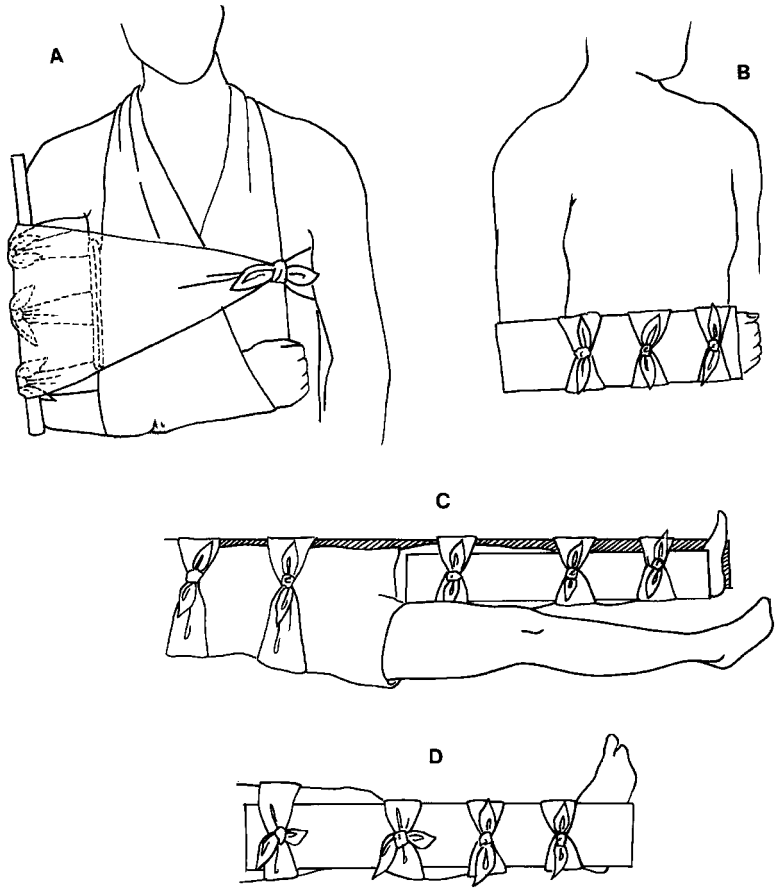

Figure 35.11 Splinting of acute fractures: (A) humeral fracture; (B) forearm fracture; (C) femoral fracture; (D) lower leg fracture. (Courtesy of AirCast Corp., Summit, NJ.)

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36 SPORTS INJURIES: A GUIDE FOR THE ON-FIELD MANAGEMENT OF INJURIES

E. James Swenson, Jr.

INTRODUCTION

The key to success in the evaluation and management of on-site injuries is *preparation*. A knowledge of the injuries that can occur coupled with an approach to systematically evaluate each injury including an appropriate functional assessment is essential. The team physician will ideally be working hand in hand with a certified athletic trainer (ATC), who will have the equipment and plan in place to treat emergencies that will need to be transported by ambulance to the medical facility of choice. If no ATC is available, the team physician must make sure the appropriate equipment and emergency phone numbers are available and that a plan is in place to transport serious injuries.

RETURN TO PLAY GUIDELINES: MUSCULOSKELETAL INJURY[1]

Severe pain, swelling, bony deformity, or joint instability preclude a return to sport. If no disqualifying factors are identified, it is appropriate to carry out sport- and joint-specific functional testing. A functional progression involves slowly and progressively stressing the joint in a series of drills until the same sport-specific forces are equaled. If the athlete completes the functional progression, both the athlete and the team physician will have confidence in the athlete's ability to perform safely under game conditions, minimizing risk. If the athlete fails to complete the functional progression because of pain or giving way of the joint, the team physician can say to the coach, "John cannot play because his knee collapses when he is running and cuts to the right at anything greater than 50% speed." The athlete will most likely relate to such objective testing and will not continue to question why he or she cannot return to the game. This builds confidence so that when a functional progression is not appropriate and the team physician must simply say, "No, I cannot allow you to participate," it is more likely that coaches and players will be receptive. Return-to-play guidelines for the most frequently injured joints — knee, ankle, and shoulder — will now be reviewed (Level of Evidence C, consensus/expert opinion).[1] The mechanisms of injury (MOI) and mechanics of other exams are found in other chapters.

The Knee

With an injury to the knee, it is particularly important to determine the MOI if a "pop" was heard or felt and any previous significant injury to either knee has occurred. The physical exam must identify any effusion, ligamentous laxity, patellar instability, meniscal tear, or any other condition at risk for additional injury should play continue. On the

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TABLE 36.1
Functional Progression for the Knee

1. No absolute contraindications on initial examination.
2. Lean Hopping — athlete hops on one foot while leaning forward and turning 360º, both directions.
3. Duck waddle.
4. Forward run — half speed progressing to full speed.
5. Run and cut — half speed progressing to full speed, to the right and the left.
6. Backwards running — half speed progressing to full speed.
7. Figure 8s — large to small pattern while increasing speed.
8. Return to game after successful completion of above sequence.

TABLE 36.2
Functional Progression for the Ankle

1. No absolute contraindications on initial examination.
2. Ability to walk with minimal to no pain.
3. Forward run — half speed progressing to full speed.
4. Run and cut — half speed progressing to full speed, to the right and the left.
5. Backwards running — half speed progressing to full speed.
6. Hop and jump — short height and distance progressing to maximal effort.
7. Figure 8s — large to small pattern while increasing speed.
8. Return to game after successful completion of above sequence.

field, the knee is exposed and inspected for any obvious bony deformity or swelling. Unless present before the injury, any effusion represents an immediate effusion or hemarthrosis. Any effusion is identified, and the Lachman, apprehension, and varus and valgus stressing are completed. Range of motion (ROM) is then evaluated, looking for a locked knee (lacking full extension), and full flexion, looking for a torn meniscus. The ATC in consultation with the team physician will decide how best to transport the athlete from the field. If in doubt as to the best method of transportation, have the athlete carried from the field. If the initial exam is negative, the athlete can walk off the field of play, where ROM, strength, and any areas of pain or tenderness can be reviewed in more detail. If no disqualifying injury is identified, the athlete can then be taken through a functional progression (Table 36.1). If the testing is completed at 85 to 90% capacity without significant pain or a limp, the athlete may return to play.

The Ankle

For ankle injuries, the physician must identify fractures and severe ligamentous laxity that would prevent return to play. Less frequently, peroneal tendon subluxation or dislocation or Achilles rupture may occur that would also prevent return to play. Often, it is very difficult to distinguish between a severe contusion and a nondisplaced ankle fracture or an ankle fracture from a severe sprain. Always err on the side of getting radiographs. In the absence of bony deformity, severe swelling, severe tenderness, a positive anterior drawer, or talar tilt and with a functional ROM the athlete may attempt to bear weight and walk as tolerated. After additional taping as necessary, the athlete may attempt to complete a functional progression (Table 36.2).

The Shoulder

Shoulder injuries that prevent continued participation include clavicular fractures, anterior subluxation or dislocation (possible torn labrum), grade II or III acromioclavicular (AC) sprains, grade II or III sternoclavicular (SC) sprains, and moderate to severe muscle strains. It is, however, common to have mild strains, sprains, and contusions that may not prevent return to play. After considering the MOI and any previous injury, the exam proceeds by having the athlete identify the site of pain. Bony or soft tissue deformity is identified and relevant structures are palpated. Passive then active ROM is completed followed by specific muscle testing. The exam concludes with stability testing. If no disqualifying conditions are identified, the athlete is ready to proceed to a sport-specific functional progression. The athlete my return to play if minimal pain, full ROM, good resistance in all muscle groups, and the ability to complete a sport-specific functional test are observed.

BRACHIAL PLEXUS INJURY[2]

A unilateral injury to the brachial plexus (commonly referred to as a "stinger" or a "burner") can present as a shoulder injury. Etiology may represent a stretch of the brachial plexus or a compression of the dorsal nerve root ganglion within the neural foramina. If the unilateral pain, burning, tingling, and weakness down into the arm completely resolve within minutes and the physical exam is normal, the athlete is allowed

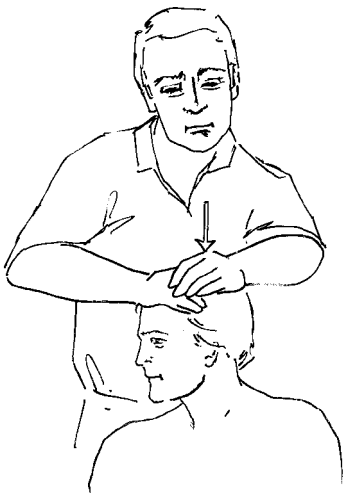

Figure 36.1 Axial compression test (see text for details). (Courtesy of Aircast Corporation, Summit, NJ.)

to return to competition. Bilateral symptoms should alert one to the possibility of a spinal cord contusion and/or spinal stenosis and should prevent continued participation until further evaluation. Significant negative findings on exam include non-tender spinous processes and a full, pain-free ROM of the C-spine, pain-free active lateral flexion to the contralateral side of the injury, and a negative Spurling test. This text is performed by application of an axial compression to the top of the head when the chin is placed in the ipsilateral supraclavicular fossa, noting any radiation of pain into the arm (Figure 36.1).

In the event of a recurrent injury, the athlete is allowed to return to competition if symptoms resolve within minutes, but every attempt is made to prevent chronic recurrence by using properly fitted shoulder pads and a protective collar, by avoiding spearing, and by strengthening neck muscles. The more proximal brachial plexus injuries are the most worrisome and difficult because of the prolonged sensitivity to even minor trauma. The athlete (and parents, as the case may be) must be made aware of the small but real risk of permanent damage should recurrent injury continue. In the event of recurrent brachial plexus injury after the above measures have been taken, it is important to consider suspension of play until the cervical muscles are strengthened. The athlete who sustains a brachial plexus injury that not only fails to resolve quickly but also shows an increasing neurologic deficit requires immediate evaluation by an orthopedic surgeon, as this condition may represent an expanding hematoma.

SPINAL INJURY[3]

The incidence of serious neck injury has been dramatically reduced since rule changes in 1976 prohibited spearing. Cervical spine instabilities (C5–C6 most frequent) produce greater morbidity than mortality through improper handling, converting an unstable condition without neurologic deficit to permanent disability. After initial assessment and stabilization, the patient is asked about neck pain, numbness, tingling, or weakness in the extremities. A positive answer for any of these questions must be taken seriously and an adequate exam completed. Palpation for C-spine tenderness is essential. Tenderness alone should prompt immobilization and transportation on a spine board for radiographic evaluation. In the absence of neurologic symptoms and without C-spine tenderness, the athlete may attempt an active ROM. With a full ROM and good strength, the athlete may return to play after successful completion of a sport-specific functional test.

OTHER MUSCULOSKELETAL INJURIES[1]

The same principles are applied to any joint by consideration of the MOI, identifying those injuries that would prevent return to play and as appropriate having the athlete complete a sport-specific, joint-specific functional test or progression prior to return to play. Muscle strains and contusions can be evaluated using these same principles. Any athlete allowed to return to play should be evaluated after the game to assess the athlete's condition and facilitate any rehabilitation or follow-up as needed. While some physicians inject professional athletes to allow continued participation,[4] the majority of physicians feel no muscle, tendon, joint, or ligament should be injected in order to allow the athlete to compete because of the risk of additional injury.

CONCUSSION[5–8]

Concussions occur in many sports. The incidence is difficult to determine with certainty in part because of the subjective nature of symptoms but also because of the multiple grading systems used to evaluate concussions. The standardized assessment of concussion (SAC) is used by some athletic trainers as another tool to assess concussion. It is felt that, for the evaluation of an acute injury and decision about return to play, the Maddocks' questions[8] are adequate to identify

the disorientation associated with concussion. These questions have been shown to be much more sensitive than the standard orientation questions (time, person, place) and include the following;

- Which field are we at?
- Which team are we playing today?
- Who is today's opponent?
- Which quarter is it?
- How far into the quarter is it?
- Which side scored the last goal?
- Which team did we play last week?
- Did we win last week?

Most team physicians feel that if an athlete is concussed and returns to play before recovery is complete, it would take less impact to result in a subsequent concussion and that the symptoms may be more severe and prolonged than if recovery from the initial concussion were complete. A head injury involving a prolonged loss of consciousness (LOC) (<1 to 2 minutes) or prolonged post-traumatic amnesia (PTA) may result in a subdural or epidural hematoma. In these instances, an immediate return to play is out of the question. In any athlete with LOC at the time of the on-field evaluation, it must assumed that there is an associated C-spine injury. Also, a subset of athletes may be subject to the so-called second-impact syndrome, thus making it all the more important that all symptoms resolve prior to return to play.

Which clinical symptoms predict injury severity? As noted above, questions challenging recently acquired memory are much more sensitive in identifying PTA in a concussed athlete than the standard orientation questions.[8] The presence or absence of retrograde amnesia (loss of memory prior to the moment of impact) is not considered a reliable indicator of injury severity or outcome. The duration of PTA has been found to correlate with the severity and outcome of severe concussions but has no prognostic value for mild concussions. The presence or absence of related symptoms (headache, dizziness, blurred vision, or nausea) as a predictor of severity or outcome remains unclear to date. The relative importance of LOC as compared to PTA or other symptoms remains unclear, as it does not correlate with severity of injury or neuropsychologic performance.[5]

While widespread controversy exists about return-to-play guidelines following a concussion, most authors are in agreement that, for a first concussion with no LOC and with any confusion, PTA, or other symptoms clearing in less than 15 to 20 minutes, the athlete can safely return to competition. While it would seem prudent to inform the athlete that another even mild concussion during that contest or during that sport season would result in a more delayed return to play, it could also have the effect of having the athlete say nothing, fearing that participation would not be allowed to continue. The proximity, severity, and total number of concussive episodes are variables that will influence the return-to-play decision (Level of Evidence C, consensus/expert opinion).[5–7] The Cantu return-to-play guidelines have remained popular since they were published in 1986 (Table 36.3). The Colorado and American Academy of Neurology concussion guidelines are discussed in Chapter 61. Most experts on concussion now agree that each case must be individualized, with the greatest emphasis placed on the clinical presentation.

Any concussion that prevents an athlete from a same-day return to play requires further evaluation. The following steps are suggested:

- All post-concussive symptoms must resolve. It is very useful to use a comprehensive checklist of possible symptoms as part of the initial assessment of the athlete. A 0 to 6 severity grading scale can help to quantify symptoms. The list is completed as soon after the injury as possible. It is very convenient if the ATC has a supply of such sheets on hand for games as well as for practices. Answers are recorded (1) at the time of injury, (2) after 24 hours, (3) after 5 days, and thereafter at intervals deemed appropriate. A column for recording baseline data should be included on the sheet.
- Symptoms must not return with exertion. If the athlete experiences a return of symptoms with exertion, that athlete can engage in low and no impact activities for 1 to 3 days and then repeat exertional testing. The longer the symptoms persist before resolution, the longer the symptom free-phase should be before return to full activity. For post-concussive symptoms lasting for more than 3 to 4 weeks, consultation with a neurologist should be consulted. For initial symptoms that are becoming more intense with time (e.g., headache and lethargy), a computed tomography (CT) scan should be considered. For less intense but prolonged symptoms (>1 to 2 weeks), magnetic resonance

TABLE 36.3
Guidelines for Concussions in Athletes

Concussion	Signs/Symptoms	Management	Return to Play
Grade I (mild)	No LOC, PTA < 30 min. May have transitory headache, blurry vision, dizziness, or nausea.	Remove from play. Physical examination immediately.[a] Observation q 5 min, examine next day.	First concussion — may return to play 20 min after symptoms clear. Second concussion — may return to play 1 week after symptoms clear. Third concussion — terminate season. May participate in non-contact sport and may return next season in contact collision sport of asymptomatic.
Grade II (moderate)	LOC < 2 min, PTA < 30 min, < 24 h; symptoms as noted above.	Prompt physical examination.[a] Observe for lucid interval. Consider transport to nearest hospital. CT/MRI/Neuro consult if symptoms/signs last longer than 2 days or return after clearing.	First concussion — may return to play 1 week after symptoms clear. Second concussion — terminate season or return to contact after asymptomatic 1 month. Third concussion — terminate sport.
Grade III (severe)	LOC > 2 min or PTA > 24 h. Symptoms as noted above.	Prompt physical examination.[a] Observe for lucid interval. Immediate transport to appropriate treatment facility. Admit if signs of pathology detected.	First concussion — consider return asymptomatic 1 month. Second concussion — terminate season or return following year if asymptomatic. Third concussion — terminate sport. Work-up includes negative CT scan and clearance from neurosurgeon.

Note: LOC = loss of consciousness; PTA = post-traumatic amnesia.

[a] Assume neck injury and immobilize C-spine.

imaging (MRI) and consultation with a neurologist should be considered.

- Neuropsychologic testing should also be considered. Even without baseline testing, a knowledge of academic performance and the presence of attention deficit hyperactivity disorder (ADHD) or learning disabilities in consultation with a neuropschologist will allow for a meaning test to be interpreted.[5,6] Written as well as computerized tests are available.
- After symptoms clear, the athlete is guided through a progressive exercise session, often by the ATC to see if symptoms return. Without a return of symptoms, the athlete is ready for neuropsychological testing. Following successful completion of neuropsychological testing, the athlete is cleared to begin a return to his or her sport. No universal guidelines exist for return to sport. Consider a functional progression that will be dependant on the level of play and experience of the athlete and would allow the athlete to phase back into full play in 3 to 7 days.
- No uniform guidelines exist to account for recent or remote concussions nor for the total number of concussions sustained. Each concussion must be evaluated individually. For an athlete who has sustained a mild to moderate concussion within the previous 3 months, consider waiting an additional symptom-free week prior to exertional testing. For the athlete who has sustained a moderate concussion in the previous month, consider lengthening that time to 2 weeks. For the athlete who sustains three concussions in any 3-month period, it would seem prudent to wait at least 3 months following the clearing of all symptoms before return to play. The more involved the scenario, the stronger

the consideration of more in-depth neuropsychological testing and a possible consultation with a neurologist. It is not only convenient but also critical for sports medicine physicians to have a team composed of athletic trainers, a neuropsychologist, and a consulting neurologist in order to optimize the evaluation and management of concussions.

- The cumulative effect of concussions must be considered when an athlete complains of academic difficulty in school. In-depth neuropsychological testing should be considered.

How many concussions are too many? Opinions vary. One thought is that an athlete who has a total of five minor concussions in consecutive years or six in any number of years should not continue participation in contact/collision sports. Three moderate concussions in consecutive years or four severe concussions over any number of years would disqualify the athlete from further participation in contact or collision sports. Any combination of the above should be considered on an individual basis. The severity of symptoms at the time of injury and the persistence of postconcussive symptoms should be considered. An MRI, in-depth neuropsychological testing, and a consultation with a neurologist can add additional insight in difficult cases. If an athlete is asked to stop participation in a contact or collision sport, alternative sports participation should be suggested.

LACERATIONS

The recent introduction of a nontoxic mixture of a hydrophilic polymer and a potassium salt has impacted in a significant way the return to play following a laceration. It is now possible to place this nonprescription powder into the laceration, stop the bleeding, return immediately to play, and wait for the conclusion of the contest to clean, debride, and suture the wound. Once applied to the wound, the powder adheres to the wound tissue to form a temporary scab. This powder seems to be quicker and more practical than steri-strips. Following the contest, simple small lacerations (<2 cm) are sutured in the training room if sterile instruments are available. Larger or contaminated lacerations are best handled in the emergency department.

SUMMARY

Management of additional injuries can be found in those chapters dealing specifically with those body areas. By utilizing the guidelines provided, the team physician should be able to manage the majority of injuries effectively on the sidelines, neither allowing the athlete to return with undue risk or restricting play unnecessarily. If, however, any question ever arises regarding whether or not to allow an athlete to participate, it is better to err on the side of conservatism and withhold the athlete from competition.

The team physician must be familiar with common MOI and be able to recognize injuries that prevent return to play. Appropriate use of functional testing in less severe injuries is key to instilling confidence in the athlete, the physician, the ATC, and others that the athlete is able to return to play and compete with minimal risk of injury. While written guidelines are available, each case must be individualized. While the preference is to return the athlete to immediate play, if possible, it is always wise to err on the side of conservatism and not allow return to play if any doubt exists in the mind of the team physician or the athlete.

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PART II
SPORTS INJURIES: THE HEAD

37 HEAD INJURIES*

Leanne C.S. Mihata

GENERAL BACKGROUND

Head injuries occur most commonly as the result of direct and forceful blunt trauma to the skull. Fortunately, catastrophic sports-related head injuries are uncommon, occurring in highest rates in bicycling, downhill skiing, hang-gliding, skydiving, mountaineering, and race-car driving. These deaths result from high-speed collisions. Mild traumatic brain injuries (MTBIs), or concussions, are far more common throughout a wide variety of sports. In the United States, football ranks first in both the number of participants and the incidence of head injuries in organized sports.[7] An excess of 6000 deaths occur annually as a result of acute head trauma, with 3 to 5 million individuals sustaining non-lethal disabling injuries of varying degrees of severity. The majority of injuries occur in young, otherwise healthy individuals, with males between the ages of 15 and 24 constituting the largest group at risk.[1] Athletes participating in collision sports and sports with a high risk of collision (e.g., football, rugby, ice hockey, soccer, boxing) are at greatest risk for head injury. In addition, long-term neurologic sequelae have been observed in athletes who sustain repetitive low-intensity head trauma (e.g., boxing, soccer, football, ice hockey, martial arts).[8]

Anatomy

The brain is covered by the three layers of tissue known as the meninges. The outermost layer consists of thick, fibrous tissue and is known as the dura. Beneath this layer is a thinner layer of tissue known as the arachnoid. Moving internally, directly overlying the brain tissue is a thin, fragile layer of vascular tissue known as the pia mater.

Enclosing the brain tissue are a series of bones that are joined together by rigid sutures to form a protective casing known as the skull. The major bones, which comprise the cranial vault, are the frontal, ethmoid, sphenoid, parietal, temporal, and occipital bones. Together these form a smooth bony vault that encloses and protects the brain tissue. Structurally, the skull contains an inner and outer

* In the second edition, this chapter was authored by Timothy Robinson, Michael D. Greenberg, and Richard B. Birrer.

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TABLE 37.1
Anatomic Makeup of the Scalp

S = skin
C = connective tissue
A = aponeurosis epicranalis
L = loose connective tissue
P = periosteum

TABLE 37.2
AVPU Mini Mental Status Assessment

A = alert
V = responds to verbal stimuli
P = responds to painful stimuli
U = unresponsive

table of hard compact bone separated by a layer of softer cancellous bone. The thickness of the skull varies in different locations and tends to be the thinnest over the temporal regions.

Covering the bones of the skull are five layers of soft tissue that make up the scalp (Table 37.1). The outermost three tissue layers (i.e., skin, connective tissue, and aponeurosis epicranalis) tend to remain fused and move as a single layer. The aponeurosis epicranalis, also known as the galea, is a thick, membranous sheet from which arise the frontalis and occipitalis muscles. A collection of loose connective tissue separates the first three layers of the scalp from the underlying periosteum.

General Mechanisms of Injury

The three types of stresses that result from an applied force are compressive, tensile (negative pressure), and shearing (force applied parallel to surface). While uniform compressive forces are tolerated fairly well by neural tissue, shearing forces are poorly tolerated. Blunt trauma to a stationary head results in injury at the immediate site of impact (coup). This is due to the transmission of force directly to the underlying brain. Trauma to a head in motion results in injury at a point opposite from the site of impact (contrecoup) (Figure 37.1). This is because the cerebral spinal fluid (CSF) allows the brain to move slightly within the cranial vault. When a moving head strikes a stationary object, the brain lags slightly, allowing a collection of CSF to cushion the brain immediately under the site of impact and dissipating the energy to the opposite side of the brain, resulting in the contrecoup injury. Additionally, the undersurface of the brain can sustain injury when sliding across the rough surface of the skull, thereby sustaining a shearing injury. When the athlete's neck muscles are tensed at the moment of head impact, functionally more mass is available through which to dissipate the force of impact; in this setting, the athlete is much less likely to sustain brain injury. Conversely, when the athlete's neck muscles are relaxed, the mass of the head is its own weight, resulting in greater risk of brain injury.[3] Penetration is the last primary and least common mechanism of injury.

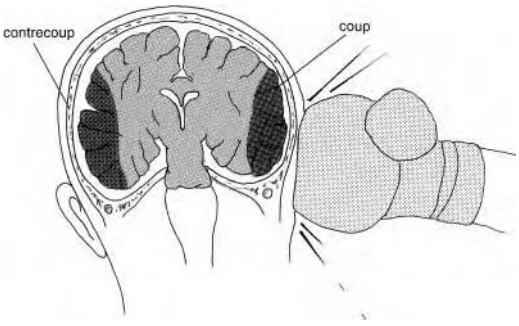

Figure 37.1 Injury types associated with blunt cerebral trauma.

Clinical Evaluation

History

A clear understanding of the mechanism of injury is important in all cases of head trauma, as it allows the examiner to anticipate associated injuries. For instance, cervical spine injury should be considered in all head traumas. Any pertinent preexisting medical or surgical conditions should be clearly documented, as well as any medications that the athlete has been using.

Physical Examination

On-the-field initial evaluation must include an assessment of the athlete's airway and cervical spine, followed by a determination of the adequacy of the athlete's respiratory and circulatory status and a rapid neurologic evaluation using the AVPU mnemonic (Table 37.2). If the athlete is conscious, has no motor or sensory deficits, and has no cervical spine point tenderness, he or she can be moved to a seated position. Slowly, the athlete can be raised up and helped off the field. Further evaluation of the athlete should take place either on the sideline or in the locker room. Increasing headache, pupillary inequality, seizures, persistent nausea or vomiting, inequality

Tests of Orientation (1 point each)	Tests of Immediate Memory (complete 3 trials regardless of score on trial 1 and 2; 1 point per word per trial)
	List Trial (circle if correct)
Month:	Word 1 1 2 3
Date:	Word 2 1 2 3
Day of week:	Word 3 1 2 3
Year:	Word 4 1 2 3
Time (accurate within 1 hour):	Word 5 1 2 3
Total Orientation score (max 5):	Total Immediate Memory Score (max 15):

Tests of Concentration	Test of Delayed Recall (same word list, 1 point each)
Digits Backward (Continue with next string length for each correct answer; if incorrect, read trial 2. Stop if incorrect on both trials. 1 point per string length, regardless if 1st or 2nd trial)	Word 1 Word 2 Word 3 Word 4 Word 5
2-5-4 7-4-3	
6-9-3-4 8-6-3-1	
5-3-7-9-2 3-7-9-2-5	
4-6-2-8-5-3 8-5-9-3-6-7	
Months Backward (must have entire sequence correct for 1 point): Dec-Nov-Oct-Sep-Aug-Jul-Jun-May-Apr-Mar-Feb-Jan	Total Delayed Recall Score (max 5):
Total Concentration Score (max 5):	TOTAL SCORE (max 30):

Neurological Screening:
-Loss of consciousness (duration):

-Recall of pre- and post-injury events (retrograde/posttraumatic amnesia):

-Strength:

-Sensation:

-Coordination:

Exertional Maneuvers (note any neurological symptoms with exercise):
5 jumping jacks
5 sit-ups
5 push-ups
5 knee-bends

Figure 37.2 Standardized assessment of concussion (Adapted from McCrea M. Standardized mental status assessment of sports concussion, *Clin. J. Sport Med.,* 11(3): 176-81, 2001.)

or deficiency of motor activity, alterations of the respiratory pattern, or progressive impairment of consciousness demand detailed and emergent neurologic evaluation.

A formal evaluation of the neurologic status then should include an assessment of the mental status with the use of the Glasgow coma scale for incidents of severe head injury, and the standardized assessment of concussion (SAC) for mild injuries (Figure 37.2), along with a full evaluation of motor and sensory function. Evaluation of motor function requires that each extremity and

TABLE 37.3
Recommended Selection Criteria for Routine Skull X-Rays[3,4]

Suspected skull penetration or depressed fracture
Suspected child abuse
Suspected opaque foreign body
Age <2 years (unless trivial injury)
Alcohol or drug intoxication
Amnesia
Multiple trauma
Vomiting
Signs of basilar fracture
Progressive headache
Unreliable history of the injury
Post-traumatic seizures
Severe facial injury
Change of consciousness at time of injury

the major muscle groups be assessed for strength and the presence or absence of any specific abnormalities such as hypotonia, spasticity, or hypertonia. Sensory evaluation should include testing each major body dermatome to determine whether the patient is able to discriminate between sharp and dull stimuli; proprioceptive function should also be assessed, and the patient should be questioned regarding any numbness or abnormal sensation.

Radiologic Studies

The yield of information from plain-film skull radiographs (AP, lateral) that affects clinical management of head-injured patients is very low (0.4 to 2%).[2] Clinical yield, however, can be enhanced by selection criteria based on the patient's history and physical exam (Table 37.3). Nevertheless, skull films are not routinely indicated for low-risk (e.g., no symptoms except dizziness or headache, minor physical findings) or high-risk (e.g., penetrating trauma, focal neurologic signs, decreasing/depressed level of consciousness) groups. Certainly, clinical judgment is paramount in rendering a final decision.

Computed tomography (CT) scanning has become the imaging modality of choice in most cases of head injury, particularly for those patients in the high-risk group. It allows for the rapid diagnosis of subdural, epidural, and intracerebral hemorrhages (Figure 37.3). In addition, the use of special "bone windows" makes it possible to demonstrate the presence of facial and skull fractures in many cases. Although magnetic resonance imaging (MRI) has a higher sensitivity than CT in the diagnosis of parenchymal abnormalities, CT scanning is preferred over MRI due to the longer time required to obtain an MRI scan, the difficulty in monitoring a trauma patient within the confines of an MRI machine, and the development of utilization criteria in cases of minor head injury.[3,4]

Prognosis

Patients with high entry Glasgow coma scores (GCSs) and those with low or normal intracranial pressures have better outcomes.[1] The percentage of patients with a GCS on entry of 3 to 4 who die or persist in a vegetative state is 80%. If the GCS is 5 to 9, the percentage drops to 50 to 55%, while 23 to 30% of those with scores of 8 to 10 have this outcome, as do only 5% of those with a GCS on entry of 11 to 15. However, the GCS has been shown to correlate poorly with outcome in cases of mild traumatic brain injury, as it is not sensitive to slight deficits in memory, attention, concentration, and other neurologic complications after sports concussion. In determining prognosis for mild traumatic brain injury, the SAC along with formal neuropsychological testing has been shown to be more useful.[10] Delayed complications of head trauma include meningitis, abscess, post-traumatic epilepsy, CSF fistula, leptomeningeal cysts, cerebral infarction, communicating hydrocephalus, and brain death.

SPECIFIC TYPES OF HEAD INJURIES

Head trauma can produce any one of a number of types of injury depending upon the specific mechanism of injury and the magnitude of the traumatic force. Types of head injuries include:

- Scalp trauma
- Skull fractures
- Cerebral concussion
- Cerebral contusion
- Intracranial hemorrhage
- Epidural hemorrhage
- Subdural hemorrhage
- Subarachnoid hemorrhage
- Intracerebral hemorrhage
- Dementia pugilistica

Traumatic Injuries of the Scalp

Scalp lacerations, abrasions, contusions, and hematomas are among the most common form

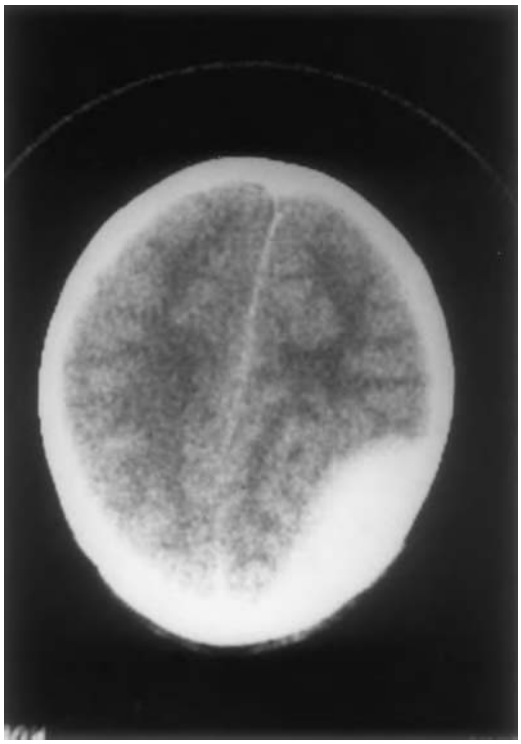

Figure 37.3A

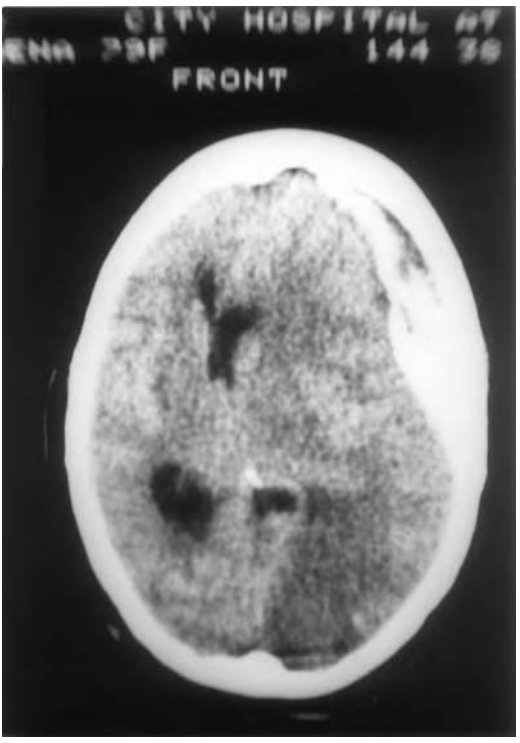

Figure 37.3B

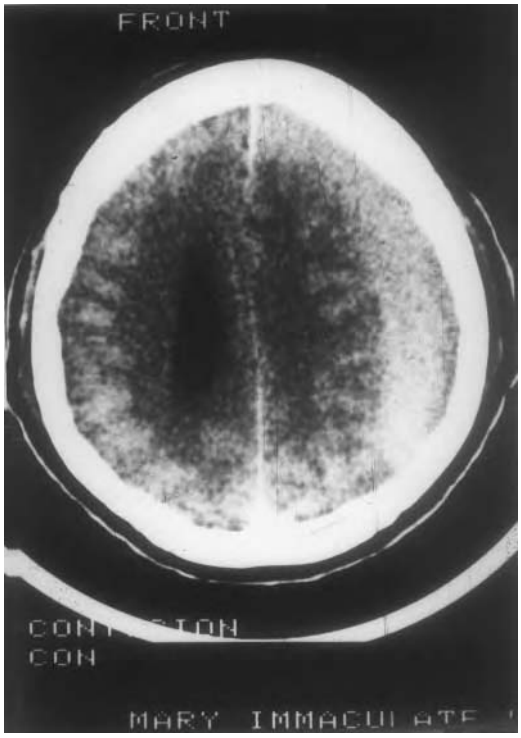

Figure 37.3C

Figure 37.3 (A) CT scanning reveals an epidural hematoma following a motocross accident. (B) Subdural hematoma, intracerebral hemorrhage, and midline shift following a diving accident. (C) This subdural hematoma appears as a crescent-shaped, hyperdense (white) area.

of head injury. Both blunt and penetrating trauma can lacerate the vascular tissues of the scalp.

Signs and Symptoms

When the scalp laceration is the result of multiple systems trauma or exists in conjunction with more serious underlying head injury (e.g., intracranial hemorrhage), the patient may offer no specific complaints. The marked vascularity of the scalp tissue and the inability of lacerated vessels to retract within the rigid confines of the galea aponeurotica often result in significant blood loss, including hypovolemic shock.

Treatment

The hair surrounding a scalp laceration should be shaved and the wound area fully exposed. Hemostats should be used to control bleeding. Meticulous irrigation and cleansing of the wound with copious amounts of normal saline are

essential. The wound should be fully explored, and a careful search for any embedded foreign body should be made. Following infiltration with an appropriate local anesthetic agent and vasoconstrictor (e.g., 1% lidocaine and 1/100,000 epinephrine), the wound should be closed in the standard fashion. In most cases, a one-layer closure utilizing a strong, nonabsorbable suture is the preferred means of wound management. If the galea aponeurotica has been cut, it is important to make sure that the lacerated ends are approximated. Finally, the tetanus status of the individual should be determined and tetanus toxoid given if appropriate.

Complications

While most patients with isolated scalp lacerations pursue an uneventful course, infection, wound dehiscence, and hemorrhage at the site of the injury are always possible. It is important to keep in mind that a scalp laceration may represent only the tip of the iceberg in a patient with head trauma. The possibility of more serious injury such as cerebral contusion or intracranial bleeding should always be kept in mind.

Prevention

The use of proper protective gear and technique during participation in sporting activities can greatly reduce the incidence of scalp lacerations as well as more serious forms of head injury.

Concussion

Concussion has been defined as "a clinical syndrome characterized by immediate and transient posttraumatic impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to brainstem involvement."[11] Confusion and amnesia are the hallmarks of concussion. These symptoms may appear immediately or may be delayed for several minutes. A list of frequently observed symptoms in concussion is provided in Table 37.4.

TABLE 37.4
Frequently Observed Symptoms of Concussion

Early symptoms (minutes to hours)
Headache
Dizziness
Impaired concentration (cannot remember plays)
Disorientation
Late symptoms (days to weeks)
Headache (especially with exertion)
Labyrinthine disturbance
Fatigue
Irritability
Impaired memory
Impaired concentration

Source: Adapted from Proctor, M.R. and Cantu, R.C., *Clin. Sports Med.*, 19(4), 693–715, 2000.

To properly care for an athlete with a concussion, the physician must first grade the injury. Much debate exists regarding the grading of severity of concussion in sport; however, the two most widely used systems are those developed by the American Academy of Neurology and by Robert Cantu, an experienced neurosurgeon and team physician. These two systems are shown in Table 37.5.[9,12] The main difference between these two systems is the emphasis placed on loss of consciousness (LOC) as a marker of severity versus duration of concussive symptoms. It is possible for an athlete to experience a brief LOC but be symptom free within a few minutes. Conversely, it is also possible for an athlete to have had no LOC but still be symptomatic the next day. Cantu maintains the second athlete has experienced a more severe concussion, despite no LOC.[9] Team physicians are encouraged to learn

TABLE 37.5
Grades of Concussions

	Grade 1	Grade 2	Grade 3
Cantu[a]	No LOC; PTA < 30 min	LOC < 5 minutes or PTA > 30 minutes	LOC > 5 minutes or PTA >24 hours
AAN[b]	Transient confusion; no LOC; PTA < 15 minutes	Transient confusion; No LOC; PTA > 15 minutes	Any LOC

Note: AAN = American Academy of Neurology, LOC = loss of consciousness, PTA = post-traumatic amnesia.

[a] Adapted from Proctor, M.R. and Cantu, R.C., *Clin. Sports Med.*, 19(4), 693–715, 2000.

[b] Adapted from Report of the Quality Standards Subcommittee, *Neurology*, 48(3), 581–585, 1997.

at least one grading system well and apply it uniformly to the athletes under their care. These management guidelines for concussion are based on expert opinion (class III).

Grading an athlete's concussion helps guide the team physician in further management and return-to-play considerations. Regardless of the grading system used, no athlete should be allowed to return to play while symptomatic from a head injury. The following return-to-play guidelines are based on the American Academy of Neurology's grading system:[12]

- Grade 1
  1. Athlete should be removed from contest.
  2. Athlete should be examined immediately and at 5-minute intervals for the development of mental status abnormalities or post-concussive symptoms at rest and with exertion.
  3. Athlete may return to contest if mental status abnormalities or post-concussive symptoms clear within 15 minutes.
  4. A second grade 1 concussion in the same contest eliminates the player from competition that day, with the player returning only if asymptomatic for 1 week at rest and with exercise.
- Grade 2
  1. Athlete should be removed from the contest and not allowed to return that day.
  2. Athlete should be examined on-site frequently for signs of evolving intracranial pathology.
  3. A trained person should reexamine the athlete the following day.
  4. A physician should perform a neurologic examination to clear the athlete for return to play after 1 full asymptomatic week at rest and with exertion.
  5. Computed tomography or MRI scanning is recommended in all instances where headache or other associated symptoms worsen or persist longer than 1 week.
  6. Following a second grade 2 concussion, return to play should be deferred until the athlete has had at least 2 weeks symptom free at rest and with exertion.
  7. Terminating the season for that player is mandated by any abnormality on CT or MRI scan consistent with brain swelling, contusion, or other intracranial pathology.
- Grade 3
  1. Transport the athlete from the field to the nearest emergency department by ambulance if still unconscious or if worrisome signs are detected (with cervical spine immobilization, if indicated).
  2. A thorough neurologic evaluation should be performed emergently, including appropriate neuroimaging procedures when indicated.
  3. Hospital admission is indicated if any signs of pathology are detected or if the mental status of the athlete remains abnormal.
  4. If findings are normal at the time of the initial medical evaluation, the athlete may be sent home. Explicit written instructions will help the family or responsible party observe the athlete over a period of time.
  5. Neurologic status should be assessed daily thereafter until all symptoms have stabilized or resolved.
  6. Prolonged unconsciousness, persistent mental status alterations, worsening post-concussion symptoms, or abnormalities on neurologic examination require urgent neurosurgical evaluation or transfer to a trauma center.
  7. After a brief (seconds) grade 3 concussion, the athlete should be withheld from play until asymptomatic for 1 week at rest and with exertion.
  8. After a prolonged (minutes) grade 3 concussion, the athlete should be withheld from play for 2 asymptomatic weeks at rest and with exertion.
  9. Following a second grade 3 concussion, the athlete should be withheld from play for a minimum of 1 asymptomatic month. The evaluating physician may elect to extend that period beyond 1 month, depending on clinical evaluation and other circumstances.
  10. Computed tomography or MRI scanning is recommended for athletes whose headache or other

associated symptoms worsen or persist longer than 1 week.

11. Any abnormality on CT or MRI consistent with brain swelling, contusion, or other intracranial pathology should result in termination of the season for that athlete, and return to play in the future should be seriously discouraged in discussions with the athlete.

The use of the SAC gives the physician a simple way to track an athlete's recovery from concussion and adds to the overall clinical picture. By itself, the SAC is not intended to be used to clear athletes for return to play; rather, it is an additional tool to use when making an assessment of the athlete's condition. The SAC is most useful when pre-season baseline scores are obtained on all athletes to use as preinjury comparison data. The test includes measures of orientation, immediate memory, concentration, and delayed recall with a maximum of 30 points. Included is a basic neurological screening as well. A decrease in an athlete's score on the SAC of 1 point from baseline testing has been shown to be a sensitive measure of brain injury, based on class I evidence. Further studies in the use of formal neuropsychological testing and correlation to recovery from concussion are currently underway. These studies should help physicians to treat athletes more appropriately, allowing less injured players to return sooner and more seriously injured players to be protected.[10]

Skull Fracture

Skull fractures result most frequently from blunt trauma. It is not the fracture itself that poses the most serious problem but rather the associated injuries such as intracranial bleeding, brain injury from embedded bone fragments, and infection secondary to contamination of the cranial vault.

Signs and Symptoms

If a history is obtainable, a complaint of severe headache, nausea, and other related symptoms is often noted. Soft-tissue injuries to the scalp are common and in some cases the fracture site may be found by careful palpation of the cranial vault. Cases of a depressed skull fracture may reveal a marked deformity in the continuity of the calvaria, along with loose bone fragments. A careful examination should be made for clues that suggest an underlying skull fracture. Physical findings suggestive of skull fracture include:

- Hemotympanum
- Postauricular ecchymosis (Battle's sign)
- Periorbital ecchymosis (raccoon eyes)
- Otorrhea
- Rhinorrhea

Fractures involving the basilar skull frequently result in cerebrospinal fluid otorrhea and rhinorrhea. The seepage of blood into the surrounding subcutaneous tissues that occurs in these injuries gives rise to the characteristic raccoon eyes (periorbital ecchymosis) and Battle's sign (postauricular ecchymosis). Blood behind the tympanic membrane is also evidence of a basal skull fracture until otherwise proven. An altered neurologic exam (e.g., lateralizing signs, coma) helps indicate the injury severity and location.

Radiographs and Special Studies

(See earlier Radiologic Studies section.) A blood-air level in the sphenoid sinus may be the only radiographic manifestation of a basal skull fracture. Because of the magnitude of the force required to produce a skull fracture, an associated cervical spine fracture must be assumed until proven otherwise.

Treatment

Consultation with a neurosurgeon is mandatory. Treatment depends upon the mechanism of injury and the extent of damage to the underlying brain tissue.

Complications

The complications associated with skull fracture include infection, direct injury to the underlying brain tissue, and damage secondary to associated intracranial bleeding.

Cerebral Contusion

A cerebral contusion indicates true anatomic injury to the brain matter itself. Unlike a concussion, where no gross anatomic injury can be demonstrated, a definite focal area of brain injury is characterized by varying combinations of cell damage, edema, and hemorrhage.

Signs and Symptoms

The clinical manifestations of cerebral contusion are highly variable and depend to a large extent upon the magnitude of the trauma as well as the area of the brain that has sustained injury. An alteration in mental status (loss of consciousness, confusion, dizziness, or abnormal behavior) is the most common manifestation of cerebral contusion.

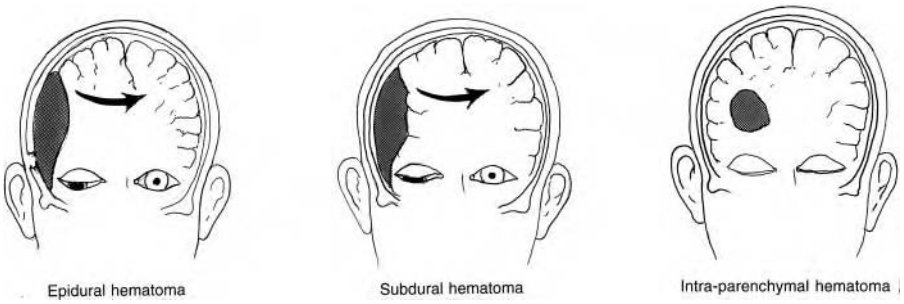

Figure 37.4 Intracranial hematomas.

Focal neurologic deficits may be present, with the particular type depending upon the location of injury.

Special Studies

Diagnostic imaging modalities (CT and MRI) can assess the extent and location of a cerebral contusion and identify varying combinations of hemorrhage and edema.

Treatment

Hospital admission and serial neurologic exams are indicated for patients with documented cerebral contusions. Further treatment is based upon the clinical status of the patient as well as the findings on CT or MRI.

Complications

The extent of injury in the patient who has sustained a cerebral contusion is variable. The exact mechanism of injury, the magnitude of the trauma, and the area of brain tissue that is injured will determine the patient's clinical course. Significant hemorrhage, edema, and mass effect may pose significant complications in these patients.

Prevention

As with all forms of head injury, wearing proper protective headgear and using appropriate technique during participation in sporting events are absolute musts.

Intracranial Hemorrhage

Bleeding within the cranial vault may be classified with respect to its anatomic relation to the brain tissue and the three meningeal layers that overlie it (Figure 37.4). Intracranial hemorrhage is classified as:

- Intracerebral hematoma
- Epidural hematoma
- Subdural hematoma
- Subarachnoid hematoma

Intracerebral Hemorrhage

Direct hemorrhage involving the brain tissue proper may either occur on the cerebral surface, as a result of a contusion, or take place beneath the surface, deep within the brain. The frontal and temporal lobes are the most frequent sites of intracerebral hemorrhage (see Figure 37.3B).

Signs and Symptoms

Alterations in mental status along with focal neurologic findings are common, with the size of the hemorrhage and the location determining the specific clinical presentation.

Treatment

Hospital admission, serial neurologic exams, and consultation with a neurosurgeon are indicated for all patients with a documented intracerebral bleed.

Epidural Hemorrhage

When traumatic injury results in hemorrhage that is confined to the space between the inner table of the calvaria and the outer meningeal layer (i.e., dura mater), the term *epidural hemorrhage* is used (see Figure 37.4).

Signs and Symptoms

The classic presentation of epidural hemorrhage (20 to 30%) is immediate loss of consciousness at the time of trauma followed by a return of consciousness (i.e., a lucid interval) and then progressive alteration in mental status with lethargy, loss of consciousness, and coma occurring over the next several hours. Many patients may not demonstrate an initial period of unconsciousness, however, whereas other individuals may lose consciousness immediately after the injury and remain in this state without any lucid interval.

Radiographs

(See earlier Radiologic Studies section.) Fractures in the temporal region should always raise suspicion of an associated epidural bleed from laceration of the middle meningeal artery.

Special Studies

Computed tomography imaging is an absolute requirement if an epidural bleed is suspected. Hemorrhage results in an expanding hematoma between the inner table of the calvaria and the dura mater. This forces the dura to bulge inward toward the brain tissue. On CT, the epidural bleed assumes a hyperdense, biconvex appearance (see Figure 37.3A).

Treatment

Immediate neurosurgical consultation is required in all cases of suspected or documented epidural bleeds. Definitive therapy is surgical. If surgery can be done before herniation occurs, the athlete has a reasonably good chance of recovery, as underlying brain injury in cases of epidural hemorrhage is mild, if present at all.[9]

Complications

Rapid expansion of an epidural hemorrhage results in marked increases in intracranial pressure and can lead to herniation and death in a relatively short time unless appropriate corrective measures are taken.

Subdural Hemorrhage

A subdural hemorrhage occurs when trauma results in bleeding localized beneath the dura mater. In contrast to epidural bleeds, which usually result from arterial injury, subdural hemorrhage is most commonly caused by a torn vein running from the surface of the brain to the dura or from diffuse injury to the surface of the brain. Unlike the epidural hematoma, where the brain substance is usually injury free, brain tissue injury is often associated with a subdural hemorrage.[9]

Signs and Symptoms

The clinical presentation of subdural hemorrhage is variable. A period of loss of consciousness usually occurs following the traumatic episode, followed by a partial, if any, improvement in mental status. Headache, neck stiffness and discomfort, changes in behavior, and pupillary abnormalities are common findings.

Radiographs and Special Studies

During the initial stage, the area of hemorrhage appears as a hyperdense, crescent-shaped region (see Figures 37.3B and C). With time, the hemorrhagic area becomes less dense on CT; by the third week, it has a density similar to that of surrounding tissue (i.e., isodense) and subsequently appears as a hypodense area.

Treatment

Immediate neurosurgical consultation is required in all cases of documented subdural hematoma. Treatment is based on the size and location of the bleed as well as the patient's clinical status. Intubation with controlled hyperventilation, along with the use of mannitol and loop diuretics, may provide temporary stabilization pending neurosurgical intervention.

Complications

The mortality rate for acute subdural hematomas is in excess of 60%, due to the associated injury to the brain tissue itself. Early recognition followed by neurosurgical consultation and intervention within the first 4 hours can reduce mortality by 50%.

Subarachnoid Hemorrhage

Bleeding in the subarachnoid space is not uncommon following head trauma. It is a result of disruption of the tiny surface brain vessels and is analogous to a bruise. It can also result from a ruptured cerebral aneurysm or arteriovenous malformation. Patients may present with variable complaints, including headache and neck stiffness. The irritative properties of the blood may precipitate a seizure. If this occurs, the athlete should be rolled onto his side to allow the tongue and any blood or saliva to roll out of the mouth and not obstruct the airway. Usually, this type of seizure lasts for only 1 to 2 minutes. The athlete should then be transported to the nearest medical facility. The diagnosis is confirmed by CT scan and treatment is generally supportive. These patients are usually given prophylactic anticonvulsant therapy for 1 week following injury, or longer if the patient actually experienced a seizure.[9]

Dementia Pugilistica

Chronic, repetitive, closed head trauma has been shown to result in permanent impairment in higher intellectual functioning.[5,6] Originally described among boxers in 1928 (punch drunk syndrome), such damage may be seen with a number of sporting activities that result in repetitive

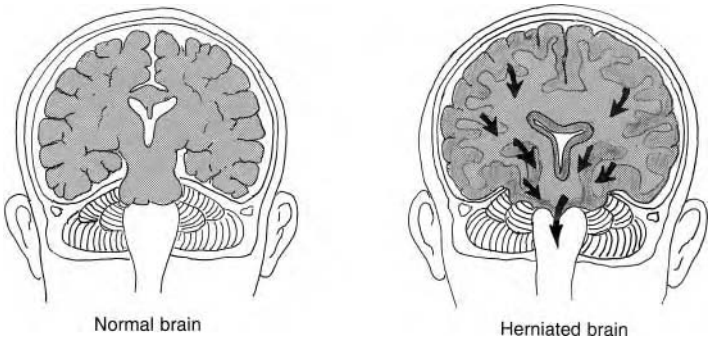

Figure 37.5 Cerebral herniation may underlie second-impact syndrome.

blunt trauma to the head. A number of neuroanatomic changes have been documented, including hydrocephalus, cavum septum pellucidum, and cortical scarring.[1,5] Symptoms and signs include cerebellar or Parkinson-like disturbances, organic mental syndrome, personality disturbances, and dysarthria. A detailed neurologic examination, electroencephalogram, CT scan, and neurologic testing are recommended. Immediate cessation of the activity with supportive care is also recommended.

Second-Impact Syndrome

A poorly understood and much debated entity, second-impact syndrome has been described as rapid brain swelling and herniation following a second head injury. In most cases described, an adolescent male athlete returned to competition while still symptomatic from a previous head injury. The athlete then sustained a relatively minor head injury. Rapid decline in the athlete's mental status ensued, ultimately resulting in brain herniation and death. The proposed mechanism for this syndrome is a disruption of the normal autoregulation of the brain's blood supply. The loss of autoregulation leads to rapid vascular engorgement, causing increased intracranial pressure and resulting in herniation of the uncus of the temporal lobe(s) through the foramen magnum (Figure 37.5). The time from second impact to brainstem failure is rapid, taking only 2 to 5 minutes. After brainstem failure, respiratory failure ensues and death is not preventable. Given the rapid and catastrophic outcome of this syndrome, prevention is paramount; therefore, no athlete should be allowed to return to play while still symptomatic from a head injury, based on class III evidence. Stricter enforcement of this rule is needed with youth sports, as adolescents seem to be more susceptible to second-impact syndrome, presumably due to an immature autoregulatory system.[9,13–15]

Concussive Convulsions

Concussive convulsions are infrequent but dramatic consequences of concussions in sports. They have been reported in Australian Rules football and rugby but can occur in any sport with a risk of concussion. Concussive convulsion is defined as a convulsive episode that begins within 2 seconds of impact and is associated with concussive brain injury. Following impact, usually a phase of brief tonic stiffening is followed by myoclonic movements. Movements are usually bilateral but can be asymmetrical. Convulsions are transient but can last up to 3 minutes in some cases. This phenomenon is more commonly seen following facial impacts than other anatomical sites of impact. These convulsions are not associated with permanent brain injury and are not epileptic phenomenon. The incidence in Australian Rules football is 131 injuries per 1000 player hours, or approximately 1 convulsive episode for every 70 cases of concussion. Immediate treatment for the athlete includes maneuvers to protect the athlete's airway and other appropriate first-aid techniques. It is the role of the physician to exclude other possibilities for cerebral injury in cases of convulsive concussions. After other head injuries have been excluded, the athlete can expect to recover fully and suffer no long-term sequelae from a simple convulsive concussion. Physicians should be aware of this entity, although it is uncommon.[16]

SUMMARY

A wide range of sporting activities, including boxing, football, martial arts, auto racing, and soccer, pose an increased risk for head injury as a result of either blunt or penetrating trauma. Because

injured persons are frequently first seen by primary care physicians, it is important that such individuals be well versed in the recognition and management of head trauma that may result in injury to the external soft tissues, underlying bony structures, or the brain matter itself. Lacerations and skull fractures are among the more common forms of extracranial injury. Intracranial injury may result in bruising of the underlying brain tissues or frank hemorrhage may occur. CT scanning provides the single best imaging study for early recognition and diagnosis of intracranial injury.

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38 EYE INJURIES*

Leanne C.S. Mihata

INTRODUCTION

Sharp, penetrating (e.g., shattered protective eyewear) and blunt, non-penetrating (e.g., fist, stick, ball) trauma causes potentially serious ocular and orbital injury in sports and recreational activities. Indirect ocular injury from rapid acceleration and deceleration also occurs when the face or head is struck without actual contact with the globe.

GENERAL EPIDEMIOLOGY

Approximately 2 to 3% of all eye injuries in the United States are sports related, with children and young adults at the highest risk of injury. Males are more frequently injured than females, at a ratio of approximately 3–4:1. In children, baseball accounts for the most eye injuries, followed by pool and swimming sports. In the adult population, basketball is the most common cause of ocular injury followed by racquet sports.[1] Sports-related eye injuries are responsible for 16% of all enucleations following ocular trauma.[2] The incidence of eye injury in sports is increasing, despite its well-recognized potential morbidity and the fact that the majority of these injuries are preventable.

MECHANISM OF INJURY

The eye, despite its fragile structure, is well protected by its bony orbit. Further protection against ocular injury is provided by rapid reflex eyelid closure. Depending on its size, speed, and direction, the initial point of contact by an offending object is often the bony orbit. With continued force, the globe is contacted, causing sequential damage to the structures in the anterior segment

* In the second edition, this chapter was authored by Timothy Robinson and Richard B. Birrer.

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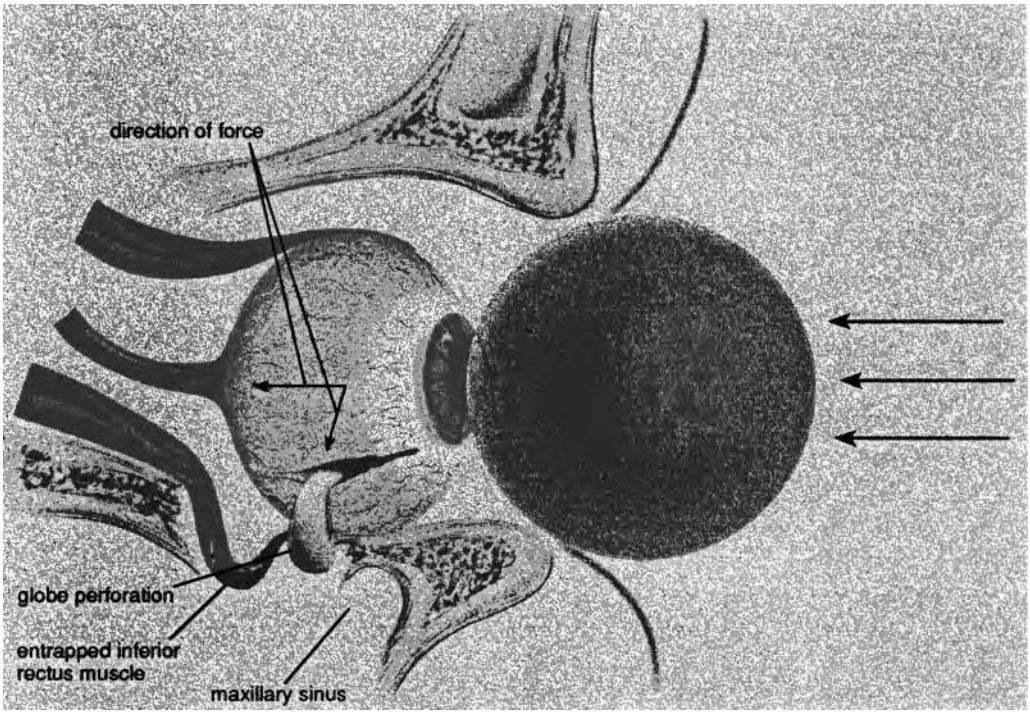

Figure 38.1 Anatomy of a globe perforation, orbital blowout fracture, and entrapment.

of the eye (e.g., conjunctiva, iris, lens), globe expansion in the horizontal and vertical planes due to anteroposterior shortening with resultant stretching and tearing of structures in the equatorial region of the eye (e.g., ciliary body, lens zonules, peripheral retina), and eventual shearing of posterior-segment ocular tissues (e.g., retina, optic nerve, and choroid) (Figure 38.1).[7] The striking end of the badminton shuttlecock, squash balls, and racquetballs are particularly hazardous because they are small enough to fit into the orbit of the eye. In addition, squash and racquetballs can easily travel faster than 100 miles per hour. Penetrating objects can cause globe rupture and can also result in an intraorbital foreign body. Lacerations of the eyelid can have important consequences for the health of the eye, as well. Corneal abrasions are frequently encountered in sports, especially those played outdoors.

HISTORY

Traumatic eye injuries should be handled with a sense of urgency and triaged along with other life- and limb-threatening injuries; therefore, it may be necessary to initiate treatment while simultaneously obtaining a thorough history, as is the case with chemical exposures. Additionally, coexisting head, neck, and spine injuries must be considered in patients presenting with ocular trauma. Important historical details to consider are mechanism of injury, amount of energy imparted, and material involved, especially with penetrating trauma. Organic material increases the risk for infection, while metallic objects can cause a reaction within vitreous and ocular tissues. Visual symptoms — floaters, decreased visual acuity, flashing lights, and diplopia — along with pain and discharge should be documented. Past ocular history — previous trauma, surgeries, corrective lenses — should also be noted. Finally, it should be noted if the athlete was wearing any eye protection and what type.[3]

EXAMINATION

During examination of the eye, care should be taken not to put pressure on the globe itself; if rupture is present, increased pressure can herniate intraocular contents. Visual acuity is the vital sign for the eye and must be assessed and recorded for each eye. If glasses are normally used, they should be worn for the testing. Visual acuity should be recorded as ability to read printed material, count fingers, perceive hand motion, or perceive light. External inspection looks for exophthalmos, enophthalmos, or deformity of external tissues. Palpation of the bony

orbit will detect step-off of a rim fracture, and the presence of crepitus and epistaxis can indicate a medial orbital fracture. Inspection of the conjunctivae and sclera should look for blood, chemosis (swelling), pigmented uveal tissue, and foreign bodies. The cornea should be inspected for clarity, irregularity, foreign bodies, and evidence of abrasion with fluorescein uptake under a Wood's lamp. Extraocular movements need to be assessed to rule out muscle entrapment or cranial nerve injury. A blowout fracture may cause hypesthesia in the distribution of the infraorbital nerve. This can be tested by sensation on the ipsilateral cheek, upper lip, and upper gum. The internal eye structures should be examined next, including observation of the size, shape, symmetry, and reactivity of the pupils. The presence of an afferent pupillary defect (paradoxical pupillary dilation with the swinging flashlight test) indicates optic nerve damage and is a poor prognostic indicator. Fundoscopy should be performed, noting the presence and intensity of the red reflex. Decreased red reflex may be a sign of a cataract, vitreous hemorrhage, or a large retinal tear.[3] Use of 1 to 2 cc of 2.5% phenylephrine hydrochloride and 1% tropicamide drops is good for pupillary dilation, which should be performed only after the acuity and pupillary responses have been documented and associated neurologic injuries have been ruled out. Retinal edema appears as a localized area of retinal pallor, whereas a detachment appears as an out-of-focus grayish billow. A thorough ocular exam should also include slit lamp assessment and intraocular pressure measurements. An ophthalmologist should be consulted for all difficult (e.g., excessive edema), unclear, or incomplete exams (e.g., possible globe ruptures precluding tonometry).

LABORATORY/RADIOLOGY

Plain orbital x-rays including Waters and Caldwell projections should be ordered for all major blunt injuries. Orbital rim, medial wall, and floor fractures will be evident, as well as associated air–fluid levels in the sinus. CT scanning of the orbit with 1- to 5-mm cuts (bone windows) are excellent for optic canal fractures, foreign bodies within the eye and orbit, and retrobulbar hemorrhage.

COMMON EYE INJURIES

Lacerations[1]

Eyelid lacerations are common, as the skin in this area is the thinnest and often absorbs the initial contact. Two important types of lacerations are full-thickness eyelid lacerations and eyelid margin lacerations. Eyelid margin laceration repairs require an ophthalmologist because proper function of the lid depends on proper repair. Malposition of the eyelid can cause chronic corneal irritation. In addition, if the laceration involves the medial one third of the eyelid, the tear drainage system will be affected. Repair of these lacerations is done in the operating room with microsurgical equipment. Full-thickness lacerations of the eyelid require special attention not only to rule out penetrating globe injuries but also to ensure future function of the levator muscle. To identify a full-thickness laceration, the eyelid can be everted to look for fat protruding through the wound. If this is seen, consultation with an ophthalmologist is a must.

Orbital Fractures[1]

The bony orbit not only houses the globe but also cranial nerves, blood vessels, periocular muscles, and the lacrimal gland. Fractures to the orbit, as a result of trauma with objects larger than the orbit itself, can damage all of these structures. Orbital roof fractures are uncommon, as the thick rim protects this part of the orbit; however, these fractures require immediate surgery, as communication with brain contents can occur through the frontal sinus. The lateral wall of the orbit is most susceptible to trauma and is usually associated with zygomatic arch fractures. Signs of these fractures, known as tripod or trimalar fractures, are depression of the cheek bone area and difficulty with opening the jaw, secondary to disruption of the tempero–mandibular joint. These fractures are usually repaired for cosmetic reasons. The medial wall of the orbit, the thinnest wall, is usually fractured as a result from damage to the orbital floor and rarely from direct trauma. Periorbital crepitus or subcutaneous emphysema may be present with medial wall fractures.

Fracture of the orbital floor is more common and can result in three main problems. First, enophthalmos can result from loss of the support structure for the globe and intraorbital tissues. This may be an immediate finding, if the intraorbital contents actually sink into the maxillary sinus. Conversely, this may not be apparent for weeks to months after the initial trauma, as orbital edema or a hematoma may initially support the globe in its position. Because enophthalmos complicates approximately 50% of large orbital fractures, many surgeons opt to repair these injuries. Second, double vision may result from a variety of causes. Periocular tissues can become trapped

in the maxillary sinus due to negative pressure, restricting mobility of the extraocular muscles. Cranial nerves can be directly damaged or edema and hematomas can trap muscles. Persistent diplopia is another indication for surgical repair. Finally, a cosmetic defect results when the anterior maxillary wall is crushed, leading to surgical correction.

An orbital blowout fracture occurs when an object larger than the orbit strikes it in such a way as to cause the orbital floor to buckle. This can cause enophthalmos, diplopia, and paresthesias over the cheek or upper teeth due to nerve injury. Whenever an orbital fracture is diagnosed, the athlete should be placed on oral antibiotics for prophylaxis against flora from disrupted sinuses.

Retrobulbar Hematoma[3]

This is a rare but sight-threatening condition that can be seen with blunt and penetrating trauma. It results from hemorrhage of the vessels in the orbital plexus, which leads to increased orbital pressure transmitted to the globe. This can cause rapid elevation of intraocular pressure, central retinal artery occlusion, and optic nerve ischemia unless treatment is initiated. Symptoms and signs include pain, nausea, vomiting, diplopia, proptosis, dissection of blood anteriorly under the conjunctiva, decreased visual acuity, elevated intraocular pressure, and restriction of ocular motility. Treatment is conservative and includes head elevation, ice, and avoidance of aspirin-containing products. Elevated intraocular pressure or an afferent pupillary defect necessitates emergent decompression with a lateral canthotomy done by an ophthalmologist. This condition must be suspected in an athlete with blunt trauma and these presenting signs and symptoms.

Subconjunctival Hemorrhage[1]

Subconjunctival hemorrhage is extremely common after blunt ocular trauma, requires no specific treatment, and gradually resolves in 2 to 3 weeks. Blood will not cover the cornea, as the conjunctiva stops at the limbus (the transition between sclera and cornea). When associated with chemosis (edema), an occult scleral perforation may be present. Additionally, a traumatic subconjunctival hemorrhage is a sign of less obvious further injury, and thorough evaluation is necessary.

Hyphema

Traumatic hyphema results from blood pooling in the anterior chamber caused by tearing of small ciliary or iris vessels. Hyphemas are graded based on size, from microscopic (grade zero), seen only with a slit lamp, to grade four, which involves the entire anterior chamber. An "eight-ball" hyphema describes a total hyphema in which the blood has begun to clot, appearing as a black ball. Symptoms of a hyphema are similar to those of iritis — pain, photophobia, and blurry vision. In cases of an associated head injury, the athlete may also appear lethargic and somnolent. Signs of hyphema include layering of blood in the anterior chamber with the athlete upright. Traumatic mydriasis or meiosis may also be observed. Hyphemas do not cause an afferent papillary defect, and the presence of this finding indicates injury to the optic nerve or the posterior segment of the eye. Complications of hyphema include rebleeding, which can occur within 2 to 5 days of the initial injury. This happens in 4 to 34% of cases. Elevated intraocular pressure due to obstruction of the outflow canal of Schlemm by red blood cells is another complication. The pooled blood can actually permanently stain the cornea; this is accentuated by increased intraocular pressure. Athletes with sickle cell disease or trait are at increased risk for complications as their red blood cells will more easily deform in the hypoxic and acidic environment of the anterior chamber. This increases the obstruction of the outflow tracts, thereby increasing the intraocular pressure. These athletes may require early surgical intervention for these injuries. All athletes with hyphema require consultation with an ophthalmologist. Immediate treatment consists of quiet activity, head elevation to 45°, and a Fox shield over the affected eye. In addition, analgesics and antiemetics can be given, but aspirin and nonsteroidal medications should be avoided. Mydriatics may be used for patient comfort as well as to prevent the formation of synechiae between the iris and the lens or the iris and the cornea. These athletes may require admission for close observation and ensured quiet activity. This is especially true for young athletes.[3] The first 5 days after injury pose the greatest risk of re-bleed. After this time, a gradual increase to the previous level of activity can be allowed. When the athlete returns to sports, appropriate use of polycarbonate protective lenses should be directed.[1]

Traumatic Uveitis[3]

Traumatic uveitis follows almost any ocular trauma. Patients complain of photophobia, headache, and blurred vision. An aqueous flare and cells are often visible on slit lamp examination, and the intraocular pressure can be decreased due to decreased aqueous production by the inflamed ciliary body or increased secondary to obstruction of the trabecular meshwork. A cycloplegic such as 2% homatropine 2 gtts 4 times a day for 1 to 2 weeks provides symptomatic relief. After consultation with an ophthalmologist, steroid drops may also be used.

Corneal Abrasion[1]

Corneal abrasions are one of the most common injuries sustained in sports-related trauma. Up to 35% of all emergency room visits for sports injuries are due to corneal abrasions. The cornea is an extremely sensitive part of the eye, and even minor disruptions of the epithelium cause great discomfort. Small abrasions give the sense of a foreign body in the eye, whereas larger abrasions can cause extreme pain, blurring vision, and tearing. The athlete may not be able to open the eye secondary to the pain. Management involves thorough inspection of the eye after instilling topical anesthetic drops to facilitate patient comfort. A retained foreign body under the eyelid must be identified and removed. This can be done with a bent paper clip to evert the lid. After removal of any foreign bodies, the extent of damage to the cornea can be assessed using fluorescein staining and a slit lamp with cobalt blue light. Most superficial abrasions heal quickly without long-term sequelae. Oral narcotics are often needed for one to two days for pain control. Topical antibiotic ointment is preferred for prophylaxis against corneal infection, based on class I evidence.[8] In some cases, a pressure patch can be placed over the affected eye for added comfort, although this has not been shown to improve healing time. Contact lens wearers require special attention in the setting of a corneal abrasion. These patients are more prone to corneal infections with Gram-negative organisms as well as being more prone to abrasions themselves. These patients should be prescribed topical antibiotics with Gram-negative coverage, such as gentamycin, tobramycin, and fluoroquinolones; additionally, contact lenses should not be worn until complete healing has been documented.

While corneal abrasions are very common in sports, associated ocular injuries should be ruled out as well. One study revealed 17% of athletes with corneal abrasions also had posterior segment hemorrhages. Microhyphema was found in 16% of athletes in this study. This highlights the importance of a thorough examination and consultation with an ophthalmologist in cases of sports-related eye injuries.

Globe Rupture[3]

Rupture of the globe is an ophthalmologic emergency. It occurs from blunt force that abruptly causes an increase in intraocular pressure. The globe ruptures at its weakest points, usually at the insertion of the extraocular muscles or the limbus. Diagnosis is usually obvious but occasionally may be subtle. Signs and symptoms include a deep or shallow anterior chamber, decreased visual acuity to light perception, hemorrhagic chemosis, hyphema, and vitreous hemorrhage. Manipulation of the eye should be avoided in all cases of suspected rupture and emergent ophthalmologic consultation is required. A Fox shield should be placed over the eye, and antiemetics, analgesics, and prophylactic antibiotics should be administered. Globe ruptures are managed with surgical intervention.

Vitreous Hemorrhage[3]

Vitreous hemorrhage in the setting of sports injuries results from blunt trauma causing tearing of the retinal vessels. These injuries are frequently associated with injuries to the choroid and retina themselves. Symptoms include floaters or dark streaks in the visual axis that move with movement of the eye. These symptoms are due to blood streaks within the vitreous humor. The athlete may also complain of blurred vision and have decreased visual acuity. Decreased red reflex will be noted with direct ophthalmoscopy. Such an athlete should keep his or her head elevated and be referred to an ophthalmologist for further treatment.

Chorioretinal Injuries

Retinal injuries can result from blunt trauma and may not present immediately. A common form of retinal injury is commotio retinae, which is acute edema of the retina that leads to blurred vision. It appears as a whitened area of the retina on fundoscopic examination. This injury is self-limited and completely resolves with no long-term visual affects.[1]

Retinal tears result from sudden shifting of the vitreous that place shearing forces on the retina. Traumatic retinal tears usually occur on the periphery and may be missed unless the eye is

dilated. If the fovea is involved, central vision will be affected. The most common place for retinal tears is the temporal superior fundus quadrant.[4] These injuries themselves are painless, as no sensory innervation of the retina is involved. Athletes may complain of seeing flashing lights due to continued traction of the vitreous on the retina; they may also see floaters if blood and/or retinal tissue have been liberated in the injury.[1]

Retinal tears may lead to retinal detachment when fluid tracks behind the tear and separates the retina from the subretinal tissues. This entity requires surgical treatment to prevent loss of vision. It is important for the team physician to remember that retinal detachment can occur long after the initial injury; therefore, they must ensure their athletes receive appropriate ophthalmologic follow-up for monitoring.

Choroidal rupture can also occur with blunt trauma. This injury causes a sheering force in the choroid, which can then disrupt the overlying retina. It is seen as concentric white scars from the optic nerve. If the injured area involves the fovea, vision will be affected. This visual loss is permanent. The greatest strategy for treatment consists of prevention with the appropriate use of protective eyewear.[1]

Lens Dislocation[1]

Traumatic lens dislocation can occur from blunt trauma in the sports setting. The lens is held in place by tiny zonules attached to the ciliary body. When these are torn in blunt trauma, the lens can be subluxed or completely dislocated. Athletes with high myopia or Marfan's syndrome are at increased risk for lens dislocation. The athlete may not have any symptoms initially with a lens subluxation; however, with a complete dislocation, an immediate loss of vision will occur in the affected eye, as the lens accounts for about one third of the focusing power of the eye. The dislocated or subluxed lens may not be apparent until the pupil is dilated.

Like the retina, no sensory nerves are located within the lens, and injury to this part of the eye is painless. However, if the dislocated lens causes blockage of aqueous flow through the pupil, an acute rise in intraocular pressure can result in symptoms of intense pain, nausea, headache, vomiting, blurred vision, and fixed pupil. This constitutes an ophthalmologic emergency and immediate treatment to lower the pressure is needed. Eventual surgical repair of dislocated and subluxed lenses is required for definitive treatment.

A late complication of lens injury is cataract formation. This may take years to develop, and regular ophthalmologic examinations are required.

GENERAL PREVENTION

Most eye injuries in sports can be prevented with the proper use of appropriate protective eyewear. Polycarbonate lenses provide the most protection and should be mandated in racquet sports and for functionally one-eyed athletes or those with previous ocular trauma, including surgery. Ice hockey and racquet sports have seen a dramatic decrease in the number of eye injuries due to the increasing number of athletes being required to wear eye protection.[1] Other sports, such as women's lacrosse, are reluctant to mandate protective eyewear, arguing that it may increase the amount of contact in this non-contact sport.[5,6] In other sports, such as rugby, martial arts, boxing, and wrestling, protective eyewear is impractical; therefore, functionally one-eyed athletes should be strongly discouraged from participating in these sports. The risks of complete visual loss should be discussed with the athlete (and their parents, as appropriate) prior to participation in the sport.

SUMMARY

Of all sports injuries, 1% involve the eye — an organ that makes up only 0.1% of the erect frontal silhouette and represents 1/375 of the body surface area. Direct and indirect (shearing) trauma from contact and collision sports can produce significant injury to the eye and its surrounding structures. Such injuries involve corneal abrasions and lacerations, levator trauma, lens dislocation, intraocular hemorrhage, retinal detachment, and globe perforation and rupture. Properly worn protective gear can prevent many of these injuries.

ACKNOWLEDGMENT

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39 EAR INJURIES*

Christy Tharenos and Richard B. Birrer

INTRODUCTION

The protuberant nature of the ear exposes it to direct and indirect trauma from a variety of contact and collision sports. Direct compressive forces from wrestling, boxing, or diving can cause hematomas, tympanic membrane perforations, and barotitis. While hearing loss has been well documented in prolonged high-noise environments, such as in riflery, the condition also has been noted to follow chronic, repetitive blows.

HEMATOMA

I. Epidemiology

Hematoma is a common injury in boxing, rugby, and wrestling and in sports utilizing small flying projectiles. It is more common in males and in participants who do not use protective head gear.

II. Mechanism of Injury

Direct trauma (single or multiple) to the auricle.

III. Anatomy

Hemorrhage into the cartilage and perichondrium.

IV. Symptoms

Pain, swelling, and bleeding if integument is disrupted.

V. Signs

Gross deformity is not unusual; swelling, discoloration, and cauliflower ear result if not treated.

VI. Laboratory/Radiology

Not usually helpful.

VII. Special Studies

Not necessary.

VIII. Diagnosis

Auricular hematoma (cauliflower, scrum, wrestler's ear).

IX. Differential Diagnosis

Rule out cyst/congenital malformation.

X. Treatment/Rehabilitation

A. Initially: Use RICE and analgesics as needed.
B. Long-term: Sterile surgical drainage should be followed by compression dressing (e.g., plaster of Paris cast, silicone mold, mineral-oil-soaked cotton balls, collodion pack) for 24 to 48 hours.

XI. Complications

Permanent distortion of external ear anatomy.

XII. Prevention

Regular use of protective headgear or ear guards, application of lubricant (Vaseline®) to ears (not allowed during matches).

* In the second edition, this chapter was authored by Timothy Robinson and Richard B. Birrer.

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RUPTURE OF THE TYMPANIC MEMBRANE

I. Epidemiology
Tympanic membrane (TM) rupture is usually seen in contact and collision sports (e.g., boxing, martial arts) but can occur in water polo, diving, surfing, and water skiing.

II. Mechanism of Injury
Indirect (fall) or a direct slapping blow to the unprotected ear.

III. Anatomy
Perforation of the tympanic membrane.

IV. Symptoms
Audible pop at the time of injury is followed by pain and hearing loss, as well as nausea, vomiting, and dizziness due to cold-water caloric effect on the labyrinth.

V. Signs
Hearing loss and visible perforation on otoscopy.

VI. Laboratory/Radiology
Not usually indicated.

VII. Special Studies
Not indicated.

VIII. Diagnosis
Tympanic membrane rupture.

IX. Differential Diagnosis
Ossicle disruption, barotrauma, skull fracture, or round window rupture.

X. Treatment/Rehabilitation
A small or moderate perforation should have conservative serial follow-up; antibiotics (drops and systemic) are reserved for documented infection. Avoid steroids, as they retard healing, which usually occurs spontaneously in 1 to 2 weeks. Large perforations should be referred to an ear, nose, and throat specialist for possible surgical grafting. Little evidence exists supporting the routine use of prosthetics for TM rupture.

XI. Complications
Permanent hearing loss, infection.

XII. Prevention
Wear protective headgear and utilize proper training techniques.

BAROTRAUMA

I. Epidemiology
Barotrauma is seen almost exclusively in smokers and sport scuba divers.

II. Mechanism of Injury
An inability to equilibrate pressure differentials across the tympanic membrane leads to barotrauma. *Ear squeeze* develops during scuba diving descent when the middle ear and sinus tissues occlude the ostea, causing underpressurization. *Reverse ear squeeze* occurs when the middle ear becomes overpressured in relation to the closed cavity between the TM and obstruction (e.g., ear plug, diving hood, cerumen, tumors).

III. Anatomy
Rigid walls and small openings of the air-filled structures of the middle ear and sinuses.

IV. Symptoms
Otalgia, blood-tinged sputum, tinnitis, decreased hearing, and ear fullness.

V. Signs
A. Decreased hearing
B. Inability to move TM with Valsalva maneuver
C. Erythematous depressed (ear squeeze) or bulging (reverse ear squeeze) TM
D. Masses, foreign bodies, or other obstruction found in sinuses, pharynx, and ear canals

VI. Laboratory/Radiology
None.

VII. Special Studies
None.

VIII. Diagnosis
Barotrauma.

IX. Differential Diagnosis
Allergy, rhinitis, sinusitis.

X. Treatment/Rehabilitation
A. Initially: Stop activity.

B. Long-term: Treat any underlying problem (cerumen impaction, sinus polyps, osteoma, exostoses, pharyngeal masses).

XI. Complications

Disability.

XII. Prevention

A. Prophylactic administration of antihistamine or decongestant (exercise caution, as a rebound phenomenon of edema and barotrauma may occur in *chronic* users of medication).
B. Keep ears (remove impacted cerumen), pharynx, and sinuses open.
C. Avoid diving with sinusitis or allergy flare-up.

OTITIS EXTERNA

For a discussion of otitis externa, see Chapter 23.

OSTEOMA/EXOSTOSIS

Osteocartilaginous exostosis and osteoid osteomas are benign, progressive neoplasms of the mesodermal tissues of the bony ear canal. Seen in coldwater swimmers and surfers, they are usually multiple and bilaterally symmetrical. Anatomically, they are bony masses covered with cartilage. Normally asymptomatic, pain can be elicited by palpation. Conductive hearing losses can occur with progressive obliteration of the canal. X-rays can define the bony contours of the mass. Surgical excision is advised for large growths. Regular use of headgear that protects the ear canals is preventive.

HEARING LOSS

Repetitive acoustical trauma (>150 dB) from the sudden explosive force of gunfire (e.g., marksmen, starting officials) can produce hearing loss. With the exception of smaller caliber 0.22 cartridges, all center-firing and rim-firing weapons and shotguns exceed the peak sound pressure level (PSPL) of 150 dB. Maximum damage occurs at the near ear, as it is closer and at a more direct angle to receive the trauma. Hearing loss can be prevented by firing in open flat terrain, regularly wearing protective ear muffs, and modifying the gun barrel.

SUMMARY

The delicate external nature of the ear subjects it to frequent insult, most of which is superficial, mild, and benign; however, repetitive insult from direct or indirect forces can result in permanent hearing loss. The regular use of protective gear is essential.

GENERAL REFERENCE

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40 NASAL INJURIES*

Charles W. Webb and Richard B. Birrer

INTRODUCTION

Facial injuries are among the more common injuries in athletics, and injuries involving the nose are the most common of these facial injuries. It is the role of the primary care sports medicine physician to determine which nasal injuries can be treated on the sideline or in the training room or must be referred to an ear, nose, and throat specialist. The onsite physician must also be able to render judgment on return to play at the time of the injury and evaluate the athlete for prevention of further injury.

MECHANISMS OF INJURY

The two mechanisms of injury common to nasal injuries are *penetrating trauma* (a stab-like wound) or, more commonly, *blunt trauma*. Blunt trauma can be caused by high-speed objects (e.g., hockey puck, baseball, bat, motor vehicle accident) or low-speed objects (e.g., another player's fist or elbow). Blows to the nose can be direct, end-on blows (resulting in comminuted injuries) or lateral blows (usually leading to dislocation injuries).

ANATOMY

The nose forms the uppermost portion of the respiratory track and serves several functions: olfaction, filtration of dust and pollens, and humidification of inspired air. The nose is composed of bone and cartilage and is divided into right and left passages. Both bony and cartilaginous tissues form the nasal septum. The bony elements are the vomer (thin flat bone) and the anterior portion of the ethmoid, which extends perpendicularly from the cribiform plate. The fleshy part of the nose is composed of cartilage. The nasal mucosa is a highly vascular tissue, with an extensive arterial and venous network. The arterial blood is provided mainly by the sphenopalatine artery, which is a branch of the maxillary artery. Kiesselbach's plexus (Little's area) is the highly vascularized portion of the antero–inferior nasal septum that is highly susceptible to epistaxis from minor trauma.[1]

EVALUATION

Evaluation of an athlete with a nasal injury, as with all aspects of medical triage, begins with

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assessment of the ABCs (airway, breathing, and circulation). Bleeding, avulsed teeth, mouth guards, or other objects can obstruct the airway in the injured athlete. Cervical spine precautions must be observed in all head injuries, particularly, if the athlete is unconscious. A thorough history must be obtained to include the mechanism of injury. The primary care physician must determine the type of injury (penetrating or blunt) and the speed of the injury (high or low), as this will give some idea as to the extent of the injuries that may be present. A brief review of systems should be performed, including questions about mental state (concussion), teeth alignment (does the athlete feel the same when he bites down? — suggests mandibular fractures or dislocation and/or avulsed teeth), and obstructions in vision (eye or orbital injury). The on-site physician should also ask about preexisting medical problems, such as hemophilia or thrombocytopenia; previous surgeries, especially rhino/septoplasty; and previous nasal fractures, as these would indicate using radiologic studies to aid in diagnosis and prognosis of injuries. Also, information regarding drug use (coumadin, aspirin, cocaine), and medical allergies should be documented. This brief history contains important information that affects the patient's immediate treatment options and potential return to play.

PHYSICAL EXAMINATION

A careful and direct examination of the nose and related structures should be conducted. It must include observation, palpation, and imaging if the diagnosis is in question. Observe for facial asymmetry, bruising, and swelling. Careful attention should be paid around the orbits; asymmetry in this area can indicate orbital or nasal fracture. The nose, orbital rim, and maxillary bones should be palpated to detect tenderness, subtle deformity, or crepitance (diagnosis of fracture). The nares should be inspected for any type of fluid drainage. This fluid might simply be blood, as in anterior epistaxis, or it could be a more ominous sign of leaking of the cerebral spinal fluid (CSF). Cerebral spinal rhinorrhea may signify fracture of the cribiform plate. The ring test can be done on the sideline or in the training room, ER, or office if cerebral spinal rhinorrhea is suspected.

> *Ring test:* Place a drop of blood from the nose on a piece of gauze or paper; CSF will form a halo (clear fluid ring) around the blood drop. If this occurs, the athlete should be transported to the nearest ER for further evaluation and treatment.

Intranasal examination should then be done using a nasal speculum or an otoscope to visualize the anterior nasal septum and turbinates. A septal hematoma must be considered if the septum is bulging into the nasal cavity. The physician must also search for any foreign bodies, lacerations, and blood clots within the nares.

IMAGING

Radiographic studies of the nose are of limited value in treatment decisions regarding nasal injuries. They are often difficult to interpret because of overlying suture lines of the facial bones and the possibility of previous trauma; however, they do provide some objective data and are an important part of chart documentation for medico-legal purposes. With this in mind, x-rays of the nasal bones should always be correlated with the clinical findings. Computed tomography (CT) scans provide the gold standard for detecting the presence of facial fractures, including nasal fractures, but are rarely of use for isolated nasal injuries.

SPECIFIED NASAL INJURIES

Nasal Fractures

Nasal fractures are commonly seen in baseball, rugby, boxing, soccer, and wrestling. These fractures result in varying degrees of deformity, based on the mechanism of injury and the direction of the blow. Direct end-on blows or inferior blows usually result in comminuted fractures of the bone and nasal cartilage. Lateral blows (side blows) usually result in displacement of the nasal bones and simple fractures with deviation to the contralateral side.[10]

Signs and Symptoms

The signs and symptoms of nasal fractures are similar to other nasal injuries: epistaxis, tearing, severe pain, facial swelling, and ecchymosis. The first visual clues to a potential nasal fracture are asymmetry, swelling, and epistaxis. Epistaxis at the time of injury is common with nasal fractures secondary to mucosal disruption.

Examination

Examination of the potential nasal fracture must include visualization (looking for asymmetry) and palpation (crepitance), as well as intranasal inspection. Visualization is best done at the time of injury before swelling has occurred. The primary care physician is looking for any obvious deformity, displacement, asymmetry, and ecchymosis. Palpation of crepitance or mobility of skeletal parts on palpation is usually diagnostic for

fracture.[2] The intranasal examination is done to evaluate for compound fracture, appearance of septal hematoma, and presence of cerebral spinal fluid. The ring test is a quick way for the primary care physician to detect cerebral spinal fluid leakage. Imaging of the nasal bones is rarely helpful in determining the presence of fracture and is not usually required; however, it may prove useful as a medico-legal record of injury.[2,3,10]

Treatment

If done immediately, the primary care physician may be able to reduce the minimally displaced nasal fracture; however, once swelling has started, the ability to assess the amount of deformity declines and relocation should not be attempted. If relocation is not possible, the athlete will require referral to an ear, nose, and throat specialist within the next 5 to 7 days. Relocation is increasingly difficult and usually requires osteotomies for adequate repair after 7 to 10 days.[2,4]

Return to Play

Return to play is not recommended for at least 1 week after nasal fractures, then external protective devices are recommend for a minimum of 4 weeks after the injury.[5]

Septal Hematoma

A septal hematoma is an accumulation of blood between the septal cartilage and the nasal mucosa (perichondrium). In children, these structures are loosely adhered, facilitating hematoma and potential abscess formation. Hematomas are mainly caused by minor trauma (no fracture, no pyramid deformation, and no laceration of the soft tissues).[6,9] A study done by Alvarez et al.[6] determined a strong male predominance, usually in the pediatric age groups. Hematomas of the nasal mucosa are prone to abscess formation and can lead to a "saddle nose" deformity, as well as other sequelea if not treated appropriately. In the study done by Alvarez et al. on pediatric patients sustaining a septal hematoma, they found that all of the patients with hematoma sustained at least some minor sequelae. Minor sequelae include minor esthetic deformities, and minimal septal and vault alterations without airway compromise. Their classification of major sequelae includes nasal deformation causing important esthetic impairment, deviation of the septum with nasal obstruction, and swelling of the cartilage.[6,9]

Signs and Symptoms

Septal hematomas usually present with a bluish nasal obstruction in one nare, but they can be bilateral. Pain and facial swelling are usually present with theses injuries, and, like nasal fractures, they are usually caused by a traumatic blow to the nose.

Examination

On physical examination, a bluish bulge is found on the nasal septum on the affected side.

Treatment

Prompt aspiration is the key to successful treatment of septal hematoma. This can be done with an 18- to 20-gauge needle. Once the hematoma has been drained, the nare is packed for 4 to 5 days to prevent reoccurrence. Prophylactic antibiotics are used for 10 to 14 days to aid in the prevention of abscess. The antibiotic should include coverage of *Staphylococcus aureus*, as it is the most common pathogen isolated.[7] Other pathogens include *Haemophilus influenzae*, *Streptococcus pneumoniae*, and group A β-hemolytic streptococcus. Because these are the primary pathogens involved, an antistaph penicillin or clindamycin is the agent initially recommended.[6] If bleeding recurs or abscesses form, prompt ear, nose, and throat referral is required.

Return to Play

Return to play is not recommended until the nasal packing is removed and the chance of rebleeding has diminished.

Epistaxis

The most common nosebleeds originate from the nasal septum and are caused by a disruption of the nasal mucosa overlying the septal vessels (Kiesselbach's plexus). Anterior epistaxis accounts for 90 to 95% of nosebleeds. The primary care physician must always consider bony or septal fracture with any traumatic epistaxis, as any force sufficient enough to cause mucosal disruption and bleeding may be sufficient to cause a fracture or dislocation of the nasal skeleton.[11]

Signs and Symptoms

The signs and symptoms of traumatic anterior epistaxis are pain in the nose and dripping of blood from the nostrils.

Treatment

The treatment is mainly ice and compression at the anterior nose. Another approach is the use of

vasoconstrictors, such as topical oxymetazoline hydrochloride or phenylephrine. This may decrease the bleeding enough to allow cautery or compression to be utilized, avoiding the potential complications of packing. If this fails, visualization of the bleeding is required so that cautery at the bleeding site can be applied (silver nitrate or electrocautery pen). If cautery is not available or the bleeding site cannot be seen, anterior packing can be applied using gauze with petroleum jelly, expandable cellulose, intranasal tampons, or various pledgets that are commercially available. In patients that require packing, referral to an ear, nose, and throat specialist should be made within 24 hours and antibiotics should be initiated.[3,11]

Return to Play

Return to play with a traumatic anterior nosebleed is a judgment call,[8] as any force sufficient enough to cause bleeding is also capable of inducing a fracture. Care must be taken to rule out nasal fracture and dislocation (see above). If the bleeding is easily controlled and no evidence of fracture is seen, return to play can be granted with a warning about the risk of nasal function being impaired at a later date if reinjury occurs.

Posterior Epistaxis

Posterior epistaxis accounts for 5 to 10% of all nosebleeds and requires more aggressive intervention.[3] As with anterior epistaxis, the cause can be either traumatic and or nontraumatic. Posterior bleeding most likely occurs from the sphenopalatine arterial system in the lateral nasal wall.

Signs and Symptoms

The signs and symptoms are similar to anterior epistaxis, except that the bleeding drains to the posterior pharynx and care must be taken to protect the airway.

Examination

On examination, the bleeding site cannot be visualized and the athlete must be evaluated for other facial trauma, such as fractures of the nose and orbit. Bleeding that does not stop with ice and compressions is an indication of posterior origin. This type of bleeding is best treated on the field with topical decongestants. Hemostasis is usually achieved if the clot is removed, then a topical decongestant (vasoconstrictor) can be sniffed until the athlete can expectorate it from the posterior pharynx. This should be repeated until the bleeding stops.[3] If the bleeding is not controlled in this manner, a small Foley (16–18 Fr.) can be inserted though the nare into the nasopharynx and inflated (10–15 cc saline). Anterior traction is put in place to seat the balloon and tamponade the bleeding. All athletes with posterior epistaxis require evaluation by an ear, nose, and throat specialist within 24 hours and may require hospitalization.

Return to Play

As posterior epistaxis can be massive and has been associated with significant morbidity, the athlete is not allowed to return to play until clearance is obtained from an ear, nose, and throat specialist.

PREVENTION

Most nasal injuries can be prevented with the use of appropriate face masks (football) and other protective gear. Care should be taken to ensure proper bracing and masking in athletes that are returning from previous facial (nasal) injuries.

SUMMARY

Facial injury is common in sports, with the nose being the most frequently injured facial structure and the most commonly occurring fracture in sports. Most of these injuries are self-limited; however, the primary care physician must be able to assess blood loss, treat epistaxis, and provide guidance to allow a safe return to competition.

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41 INJURIES TO FACIAL BONES AND TEETH*

Joseph E. Allen

INTRODUCTION

Traumatic head injuries involving the face are primarily due to its large surface area and delicate construction. Facial fractures account for 11% of all sports-related injuries and are more prevalent in close contact sports (e.g., basketball, football, martial arts).[9] While most facial trauma is mild in nature, more significant injury frequently occurs to fragile facial bones. The nasal, ethmoid, and zygomatic bones quickly dissipate kinetic energy, thus sparing more vital underlying organs. High-impact forces are necessary to fracture the orbital margins and the anterior mandible; 50% of such trauma is associated with serious injury elsewhere. During evaluation of an acute facial injury, the sports medicine physician must first ensure adequate airway, breathing, and circulation and then carefully evaluate for potential cervical spine, ocular, or additional central nervous system (CNS) involvement.

FRACTURE OF THE FRONTAL SINUS

Injury to the frontal sinus may involve the anterior or posterior walls. Breech of the posterior wall may lacerate the dura, resulting in CSF leakage, CNS depression, and potential infection. Numbness may be felt over the supraorbital nerve distribution. Radiological evaluation involves computed tomography (CT) scan with 2-mm sinus sections. Fractures may be evident on PA /lateral and Water's view projections. If CNS involvement is suspected, an urgent neurosurgical evaluation should be initiated.

FRACTURE OF THE MANDIBLE

The mandible is the third most commonly fractured facial bone. Direct or indirect blunt or penetrating trauma to the mandible during contact/collision sports is the usual mechanism of injury. The majority of mandibular trauma is often associated with additional injuries, usually fractures.[3] The primary site of fracture depends on amount and direction of force, the most common sites being the condyle and mandibular angle. Symptoms in the injured athlete include pain, swelling, and inability to chew or completely close the mouth. The examiner may observe localized swelling and ecchymosis, step-off deformity, malocclusion, and deviation of the jaw to the fractured side. Point tenderness to palpation and complaints of paresthesias of the lower lip may be present. Bleeding may indicate an open fracture, and a thorough exam should identify potential dental alveolar involvement.

Preliminary radiographic studies to visualize suspected fractures and dislocations should include a mandibular series (PA and lateral obliques), panoramic and Towne's view. CT scan may be necessary to identify hairline and condylar

* In the second edition, this chapter was authored by Timothy Robinson and Richard B. Birrer.

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fractures. Initial treatment of a mandibular fracture involves temporary immobilization with an elastic bandage using Barton's or barrel methods. Further reduction and fixation (intermaxillary wiring or bone plates) may be required. Rehabilitation with intermaxillary jaw fixation allows for mild repetitive activities (lightweight training, cycling, or swimming) and a high protein/carbohydrate liquid diet for 4 to 6 weeks. A 5 to 10% weight loss commonly occurs. Resumption of full activity, including contact or collision sports, requires wearing special headgear and a customized mouthguard for an additional 1 to 2 months. Physical therapy is recommended during this time. The schedule is accelerated by 1 month if bone plates have been used or if the condyle was fractured. Possible complications include malocclusion, infection, or loss of dentition. Proper protective equipment and training technique are employed as further preventive measures.

DISLOCATION OF THE MANDIBLE

The mandibular condyles articulate with the mandibular fossae of the temporal bone to form the sliding hinge joint called the temporomandibular joint (TMJ). Indirect trauma to the mandible or direct trauma to the TMJ may result in mandibular dislocation. The mandible dislocates inferior and anterior. Symptoms of dislocation include pain, inability to chew, or presence of an open bite. Differential diagnoses associated with TMJ dislocation include hemarthrosis, fracture, capsulitis, and internal derangement of meniscus. On physical examination, the TMJ is tender, and palpable crepitus may be observed, along with malocclusion, swelling, restriction of jaw motion (opening <40 mm and lack of closure). The jaw will deviate to the normal side on opening. Radiographic studies should include panoramic and Towne's views.

An arthrogram, a CT, or magnetic resonance imaging (MRI) of the TMJ may be included to evaluate meniscus integrity. Initial treatment involves RICE (rest, ice, compression, elevation), temporary immobilization (Barton's or barrel elastic bandaging), and reduction (anterior or posterior [preferred]) utilizing postero-inferior-directed force with both thumbs of the operator firmly hooked inside the mouth on the third molars. Anesthetic (local or general) or muscle relaxants are helpful in difficult cases. Fixation or stabilization are also required if fracture is present.

Long-term management entails a soft diet, nonsteroidal anti-inflammatory drugs (NSAIDs) or analgesics for 1 to 2 weeks and progressive rehabilitation utilizing tongue blades. Graduated return to play including heavyweight training is permitted after the acute period (7 to 10 days) has subsided. Some restrictions apply following arthroscopy. If surgery has been performed, including arthroplasty, 1 to 2 months of rest before a 2- to 3-month period of progressive conditioning and training is recommended; the athlete should use a special headgear and mouthguard. Malocclusion, recurrent dislocation, and TMJ arthritis are possible long-term sequellae. As with mandibular fracture, proper protective equipment and training techniques are employed for preventative measure.

FRACTURE OF THE ZYGOMATIC COMPLEX

The zygoma forms the inferolateral aspect of the bony eye socket and extends laterally to articulate with the parietal bone to form the cheek. After the nasal bone, it is the second most commonly fractured facial bone in sporting events and frequently is a result of contact and collision sports (e.g., basketball, hockey, baseball, rugby.[9] Acute fracture of the zygomatic complex may present with cosmetic deformity, double vision (vertical diplopia with a blowout fracture), swelling, numbness of the cheek, and trismus or difficulty opening mouth. The face should be examined for abnormal extraocular movements, ecchymosis (periorbital and intraoral), and enophthalmos. Also observed may be obvious flattening of the cheek, step-off defect of the inferolateral orbit or zygomatic arch, and sensory deficit to the affected area, as well as periorbital edema and palpable emphysema. Ocular trauma should always be suspected and the eye carefully examined for visual deficit, subconjunctival hemmorhage, corneal abrasion, and hyphema.

Radiology evaluation should include a skull series, Water's view, and submental vertex. CT scan with 2-mm bone windows of the orbit may be warranted as well as an urgent ophthalmological consultation. Initial treatment involves protection of ocular structures, control of hemorrhage, and transport for comprehensive evaluation, surgical reduction, and stabilization. Healing is complete in 6 to 8 weeks, but special headgear should be worn for an additional 3 to 4 months. Potential complications due to zygomatic fracture include cosmetic deformity, diplopia, intraocular injury, and retrobulbar hemorrhage. Primary prevention requires proper protective gear and training techniques, as well as adequate supervision.

MAXILLARY FRACTURE

The maxilla forms the midface, and direct traumatic fracture to the area, though uncommon, has occurred in collision and contact sports. LeFort fractures of the midface are classified as types I, II, or III (Figure 41.1).[9] Every effort should be made to ensure patency of the airway and remove any potential obstruction (e.g., avulsed teeth). Symptoms of maxillary fracture include pain and swelling, deformity, or bleeding. The injured athlete may exhibit malocclusion, deformity (e.g., facial dishing or lengthening), swelling, ecchymosis of buccal vestibule, midfacial instability and mobility, and CSF rhinorrhea. It is essential to check for associated injuries (e.g., CNS, C-spine) and perform immediate assessment of the ABCs (airways, breathing, circulation), taking C-spine precautions if indicated. The conscious patient should be rapidly transported for further evaluation in the upright, forward-leaning position to allow for dependent external drainage of saliva and blood. Definitive treatment involves open reduction, internal fixation, and immobilization (primary and secondary). CT scan is the imaging modality of choice. Rehabilitation is similar to that utilized for mandibular fractures. Potential complications include malocclusion, airway compromise, cosmetic deformity, and infection. A customized face shield should be employed during the bone healing process for athletic participation in contact sports.

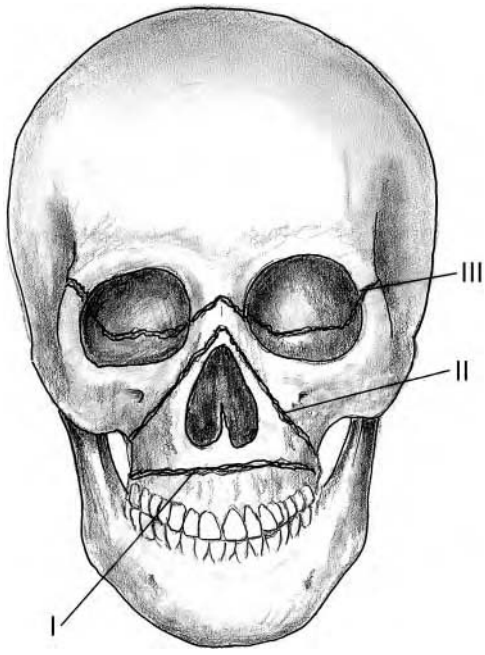

Figure 41.1 Le Fort classification of maxillary fractures.

FRACTURE OF THE ORBIT

The orbital ring consists of the supraorbital ridge, glabella, infraorbital, and lateral orbital rims. The walls, floor, and roof are paper thin, and the floor is most commonly fractured, as it is the weakest part.[1,4] Traumatic orbital fractures are unusual in sports and result predominantly from high-velocity racquet sports (e.g., tennis, squash, racquetball). Direct blunt trauma by a projectile (e.g., a ball) may often result in a rapid rise in intraorbital pressure. The acutely injured athlete may complain of pain, swelling, double vision, and facial numbness. The examiner may observe periorbital edema, ecchymosis, subconjunctival hemorrhage, enophthalmos, unilateral epistaxis, decreased extraocular motilities (EOMs), or hypesthesia over the affected infraorbital nerve distribution. Palpation of orbits and eyelids elicits pain and may detect subcutaneous emphysema. Careful examination of the eye is mandatory and urgent ophthalmological consultation should follow stabilization and transport. The area may be carefully iced without compression until definitive reduction and orbital reconstruction can be performed. Rehabilitation follows the zygomaticomaxillary complex (ZMC) protocol. Initial radiological evaluation with a Water's view may demonstrate herniation of orbital contents into the maxillary sinus (opacification) (Figure 41.2). CT scans with coronal and sagittal views (bone windows) with 2-mm thin cuts through the orbits are usually diagnostic. Complications include diplopia, retrobulbar hemorrhage, cosmetic deformity, and intraocular injury. Prevention requires proper protective equipment (see Chapter 13).

DENTOALVEOLAR INJURIES

The teeth consist of an enameled surface over the dentin that surrounds the vital pulp. The teeth are firmly anchored in the alveolar ridge of the mandible. Traumatic dental injuries are frequently associated with contact or collision sports.[2,8] Dentoalveolar injuries may be classified as a contusion or subluxation, avulsion, or fracture. Symptoms of dental trauma include pain, bleeding, malocclusion, and missing or fractured teeth. The examiner should first ensure a patent airway, then check the oral cavity for foreign bodies and associated lacerations. Every effort should be made to locate missing teeth, as they may be successfully reimplanted if intact, and also to ensure that they have not been aspirated. Dental films are

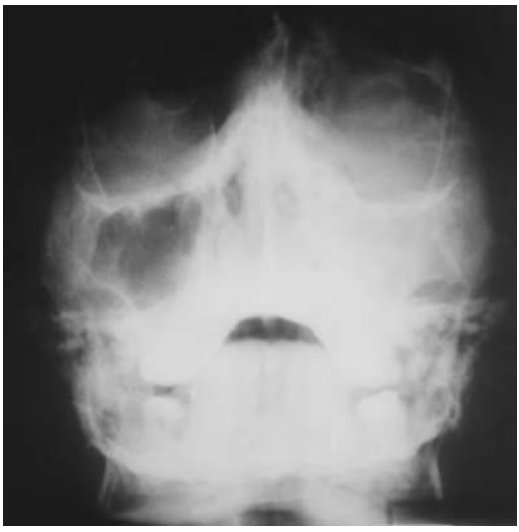

Figure 41.2 Water's view showing opacification of left maxillary sinus as a result of blood from an ipsilateral blowout fracture of the left infraorbital margin.

recommended for loosened teeth to rule out fracture below the gumline. Chest x-ray may be done to rule out aspiration.

Significant dentoalveolar injuries are best managed by an oral surgeon or dentist. Dental injuries are repaired prior to management of facial or lip soft tissue trauma. With the exception of desidual teeth in children, reimplantation of avulsed or extruded subluxed teeth should always be attempted within 30 minutes.[5] Hold the tooth by the crown and, if it is dirty, gently cleanse the tooth with tap water or saliva (do not rub). Replace into the tooth socket and have the patient bite down on gauze or clean soft tissue to keep the tooth in place.

If reimplantation is not possible, transport the tooth in cold saline, milk, or the patient's saliva (wrapped in gauze under the tongue or in cheek pouch). Patients with contaminated, avulsed teeth should be treated with oral antibiotics for 10 to 14 days. Tetanus prophylaxis should be updated if necessary. Activities should be restricted to light training when dental splints are in place; return to full play should be allowed after the splints have been removed, healing is deemed adequate (3 to 6 weeks), and appropriate face and mouthguards are in place. Complications include tooth loss and dentoalveolar ankylosis. Prevention requires proper training technique, appropriate protective gear (see Chapter 13), and adequate enforcement of regulations (e.g., mouthguards and chinstraps).

SUMMARY

Sports-related facial trauma is usually mild in degree (e.g., contusions). Fractures tend to involve thin facial bones, sparing important anterior underlying structures. Serious facial injuries follow high-velocity collision activities. Airway management is paramount, followed by a detailed and extensive trauma assessment and management. CT scan is the study of choice for the evaluation of suspected facial fractures. Outcome often depends on prompt evaluation and treatment of associated injuries. With the appropriate use of head and face protection, an estimated 100,000 sports-related injuries could be prevented annually.[8]

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PART III
SPORTS INJURIES: THE TRUNK

42 CHEST INJURIES

Christy Tharenos and Richard B. Birrer

INTRODUCTION

The large size and central location of the chest lead to frequent injury; however, the anatomic architecture of the thorax, particularly its bony support and muscular development, provides adequate protection against all but the most serious trauma. Contusions and strains, therefore, while common in collision and contact sports (e.g., cricket, football, rugby, hockey), are usually mild in nature and respond well to standard therapy. More significant injuries (e.g., breast contusions, third-degree strains) may be associated with a hematoma and should be surgically aspirated. Subcutaneous fat necrosis has been reported following breast hematomas. Third-degree strains of the pectoralis major muscle are associated with a "pop" or "snap" sound; sudden, sharp, upper-arm pain; ecchymosis; muscle belly bulging on resisted adduction; weakness of arm adduction, flexion, and internal rotation; a gap in the anterior axillary fold; and an absent pectoralis major shadow on a chest film. Surgical repair is recommended for young or professional athletes in order to restore full function and strength. Total recovery time is often 3 to 6 months.

BREAST PAIN AND JOGGER'S NIPPLE

I. Epidemiology

Common in running sports, jogger's nipple was originally described in males and has become more common due to the larger number of male runners. Breast pain is more common in females due to the size of the breast. Risk factors include size of breast, fibrocystic disease, pregnancy, premenstrual period, and absence of a supportive brassiere.

II. Mechanism of Injury

Repeated microtrauma or bouncing of the breast.

III. Anatomy

Frictional abrasion of the nipple, microcontusion of the breast organ, and spraining of Cooper's ligaments (probably not significant).

IV. Symptoms

Pain and bleeding from the nipple, breast soreness.

V. Signs

Erythema, denudation and hemorrhage of the nipple, nonfocal tenderness on palpation of the breast, or replication of symptoms on jumping or running.

VI. Laboratory/Radiology

None, unless a question of tumor arises.

VII. Special Studies

None.

VIII. Diagnosis

Jogger's/runner's nipple, breast pain.

IX. Differential Diagnosis

Mondor's disease, tumor, fibrocystic disease, hematoma, or Paget's disease.

X. Treatment/Rehabilitation

1. *Jogger's/runner's nipple:* Coat the nipples with talcum powder or petrolatum, cover them with a piece of tape (such as a Band-Aid®), or wear a silk shirt or shirt made of synthetic material (polyester).

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2. *Breast pain:* Use a minimally elastic support brassiere with at least 55% cotton for absorbency, with well-covered hooks and a minimum amount of seams. A 4-inch elastic wrap binding can also be used for additional support.

XI. Complications
Recurrence.

XII. Prevention
See Treatment/Rehabilitation.

SPRAINS (SUBLUXATIONS AND DISLOCATIONS)

I. Epidemiology
Sprains are common in contact and collision sports, such as wrestling, rugby, and football. Injury is usually unilateral and involves ribs number 8, 9, 10. Direct forces sufficient to dislocate the first three upper or floating ribs often cause significant injury to the underlying viscera.

II. Mechanism of Injury
Direct blow to the sternum or to the antero-lateral chest in association with twisting, lifting, or an arm pull causing a forward thrust of the ribs with subluxation and eventual dislocation.

III. Anatomy
- Grades 1 and 2 — partial or complete tearing of the anterior and posterior costochondral, costosternal, or costovertebral ligaments, sometimes accompanied by impingement on the overlying intercostal nerve.
- Grade 3 — subluxation dislocation (see chapter 45).

IV. Symptoms
Localized tenderness, swelling, and pain on respiration, bending, coughing, or straining with grades 1 and 2 sprains; popping, slipping out, or snapping sensation with grade 3 injuries.

V. Signs
Edema, crepitation, and localized tenderness in grade 1 and 2 injuries; step-off deformity and positive hooking maneuver (reproduction of pain when examiner hooks fingers underneath the costal margin and draws the rib cage forward) in grade 3 sprains.[1]

VI. Laboratory/Radiology
Rib, sternum, and spine films to rule out fracture.

VII. Special Studies
None.

VIII. Diagnosis
Sprain, grades 1 to 3 (slipping, clicking rib, rib tip syndrome).

IX. Differential Diagnosis
Fracture, costochondritis (Tietze's syndrome), strain, visceral trauma.

X. Treatment/Rehabilitation
A. Initially: Use RICE (rest, ice, compression, elevation) plus local injection of 1% lidocaine in the area of injury, or intercostal nerve block plus nonsteroidal anti-inflammatory drugs (NSAIDs).
B. Long-term: Use rib belt or circumferential strapping of the chest, range of motion (ROM) + progressive resistance exercise (PRE). Return to play can be allowed in 3 to 6 weeks; rarely, surgical resection of the rib is required.

Consultation: recommended for more serious dislocations (e.g., posterior costovertebral).

XI. Complications
Recurrence.

XII. Prevention
Wear properly fitted padding and bracing.

FRACTURES

I. Epidemiology
Fractures are common in contact and collision sports; less common in golf, pitching, batting, lifting, and racquet sport volleys due to indirect trauma from twists and turns. Fracture of the first four and last two ribs is often associated with significant visceral injury.[2]

II. Mechanism of Injury
Direct blow from a ball, body part, implement, or other device causes fractures of the sternum or ribs; indirect forces produce fracture of the posterior upper ribs, particularly the first rib; opposing traction forces from the serratus anterior and scalene muscles, particularly the anterior, due to shoulder protraction during weightlifting, rowing, surfing, throwing, or tennis serve snap the

first rib through from overpull; sudden vigorous contraction with different direction of pull produces avulsion fractures of the external oblique muscle attachments of the lower three floating ribs.[2]

III. Anatomy
Fracture of the ribs or sternum in association with sprains, strains, and contusions.

IV. Symptoms
Localized pain on inspiration resulting in rapid, shallow breathing; shock-like reaction in association with variable amounts of dyspnea following a sternal blow (failure of recovery after a short period should suggest more serious organ injury).

V. Signs
Trigger point tenderness plus crepitus or positive "bucket handling" — pain and crepitus at the fracture site produced by antero-posterior compression of the chest with one hand placed over the thoracic spine and the other over the sternum. A complete chest and abdominal exam should be performed to look for associated organ damage.

VI. Laboratory/Radiology
Antero-posterior, lateral, and oblique views of the sternum or ribs plus chest x-ray. Rib films may be negative due to the oblique course of the rib, overlying soft tissue, frequent lack of displacement, and depth of the thorax. Electrocardiogram (EKG) can be used for sternal injuries and may show ST-T wave abnormalities.

VII. Special Studies
None.

VIII. Diagnosis
Fracture.

IX. Differential Diagnosis
Contusion, sprain, strain, costochondral separation, pneumothorax, injuries to the heart and great vessels following sternal trauma, or internal mammary artery or intercostal nerve vessel damage.

X. Treatment/Rehabilitation
A. Initially: Use RICE plus NSAIDs plus localized 1% lidocaine injection.
B. Long-term: Use a rib belt or circumferential strapping of the involved side unless the injury is complicated by visceral injury or is in the setting of chronic cardiopulmonary disease, repeated intercostal nerve blocks, ROM plus PRE.[3] A sling can be used for fractures of the first ribs. Six to 12 weeks are required for healing and rehabilitation before allowing return to play in contact sports.

Consultation: flail or multiple fractures, fractures of the first four or last two ribs.

XI. Complications
None unless viscera also damaged.

XII. Prevention
Wear a properly fitted rubber chest protector/blocking vest (rubber air bladder covered with a plastic shield) or a similarly tailored contact pad.

SUMMARY
Because of its size and location, the chest is frequently subjected to sports-related trauma. Fortunately, most injuries are insignificant in nature. The primary care physician should, however, be alert for rare serious injuries (e.g., lung or heart) which often present with unassuming or subtle signs and symptoms.

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43 ABDOMINAL INJURIES

Richard B. Birrer

INTRODUCTION

A large surface area, absence of bony support, and a general lack of protective gear make the athlete's abdomen a prime target for potentially serious injury. Abdominal injuries may cause death because they are often subtle in presentation and clinically difficult to recognize.[1] A high index of suspicion is essential on the part of the sports physician to distinguish serious from lethal etiologies. The nature and localization of an athletic injury may not always be apparent due to the indirect accessibility of diagnostic maneuvers. Many abdominal injuries are occult in nature and may be delayed in presentation. The diagnostic assessment must be organized and thorough in order to identify the most serious injuries (Level of Evidence B, other evidence).[2] Finally, it is paramount that injury prioritization be kept foremost in the physician's mind. Thus, airway, breathing, and circulation should be guaranteed before further evaluation of possible internal abdominal injury is made.

VISCERAL INJURIES

I. Epidemiology

Sports-related trauma causes about 10% of all abdominal injuries;[3,4] serious injuries are rare.[5] Males are injured more commonly than females.[6] Consider pregnancy in the injured female athlete. Contact and collision sports are the most commonly involved, although endurance sports and the use of such vehicles as bicycles, motorcycles, ATVs, snowmobiles, dune buggies, etc. can result in significant abdominal injury due to spearing, acceleration, and deceleration mechanisms.[7] Less commonly encountered activities such as boogie boarding, SCUBA diving, martial arts, softball, snowboarding, weightlifting, and monkey bars or jungle gymnastics have been reported to cause serious or lethal injuries.[8–14] Overall, bicycling is the most common cause of abdominal injury.[7] Of intra-abdominal injuries, 30% are associated with significant trauma to other body areas (head, thorax, extremities). Infectious mononucleosis is a major risk factor for splenic injury.

II. Mechanism of Injury

The most common pathogenesis is blunt abdominal trauma (fist, ball, elbow, stick); others include penetration (e.g., spearing or impalement from a ski pole, handlebar, or skate point) and shear.[15,16] Specifically, the unguarded abdomen is at high risk from unexpected blows (e.g., being tackled after a pass reception, contacting the handle bars as the cyclist is thrown forward and down).

III. Anatomy

The spleen, particularly if enlarged and friable (infectious mononucleosis), is the most frequently injured organ and most common cause of death, followed by injury to the liver. Kidneys are less commonly injured, mostly from blunt abdominal blows and less frequently from flank trauma. The pancreas may be injured by stretch over the spine, whereas the duodenum and distal ileum may be subject to shear at the ligament of Treitz or mesentery, respectively. The stomach and bladder are rarely injured unless full.

IV. Symptoms

The following history items are essential:

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A. Type of object striking the abdomen (e.g., blunt or sharp, elbow, knee, helmet)
B. Position of body, muscle tone, and whether the bladder was full or empty at the time of impact
C. Injury location
D. Elapsed time since trauma
E. History of previous abdominal injury
F. Time of last meal and bowel movement
G. History of infectious mononucleosis or hepatosplenomegaly

The athlete may experience no pain or immediate pain, or discomfort in the area of contact may mimic a blow to the solar plexus ("wind being knocked out") or a "stitch in the side." Pain may then become dull and quickly disappear, only to recur as diffuse, incapacitating abdominal pain several hours to days later, depending on the nature and extent of the underlying injury. Aggravation of pain by body movements (e.g., walking, coughing, laughing, bouncing) suggests peritoneal irritation. Small bleeds often cause minimal symptoms; pain may be referred to the left shoulder (Kehr's sign) in splenic injuries or to the right shoulder with hepatic injuries. Gross hematuria may follow renal trauma. Rapid recovery does not indicate the absence of significant pathology.

V. Signs

The following examination items are essential:[2]

A. Vital signs. Initially, the abdomen may be scaphoid and localized tenderness may be noted. Later (hours, days), distension, obvious swelling or discoloration (Cullen's or Grey–Turner's signs), diffuse peritoneal signs (including referred and rebound tenderness), or shock may occur.
B. Inspection, palpation, percussion, auscultation of the abdomen.
C. Rectal and pelvic exams.
D. Chest exam.

Involuntary spasm is the most reliable physical sign indicating peritoneal irritation. Voluntary spasm, which is inconsistent, can be diagnosed by noting the decrease in muscular spasm under the examining hand as the supine patient is asked to breathe deeply with knees bent. Localized rigidity is suggestive of a walled-off lesion from bleeding or leakage. Confirmation is made by noting rebound tenderness to the primary site under consideration. Percussion has the lowest sensitivity but moderate specificity. Auscultation is not terribly useful, although the absence of bowel sounds is a frequent sign of early hemorrhage or significant abdominal injury. The patient may have no signs of small amounts of hemorrhage.

Serial exam including vital signs is the best method of diagnosing intra-abdominal injury. Examine the most tender area last. Retroperitoneal injuries may cause few, if any, intraperitoneal signs if the trauma is minor or in the early stages of injury.

VI. Laboratory/Radiology

Serial CBC, urinalysis, type, and crossmatch should be performed.[17] Normal urinalysis does not rule out renal injury. The amount of hematuria correlates poorly with the amount of injury. Diagnostic peritoneal lavage (DPL) is most sensitive and specific (95 to 98%) for intra-abdominal injury; it is less valuable in detecting retroperitoneal injuries. DPL is considered positive with the aspiration of non-clotting blood, the removal of 20,000 to 100,000 RBCs per m^3, 300 to 700 WBCs per m^3, elevated amylase, or the presence of bile, bacteria, or vegetable matter. Complications include puncture of viscera, vessels, or omentum. *In general, athletes with a positive history and physical exam indicating peritoneal involvement, particularly if they present in shock, do not need extensive diagnostic evaluations.*

For serial right decubitus and upright abdominal x-ray, allow the athlete to maintain position for at least 3 minutes before film is shot.[3] Free air is visible underneath the diaphragm leaflets following the rupture of a hollow viscus; obliteration of the splenic shadow of the colon, depression of the colonic flexure, elevation of the left hemidiaphragm, and perhaps prominence of the gastric rugae suggest splenic injury; loss of renal outline, associated rib or vertebral fractures, blurring of the psoas margin, elevation of the ipsilateral hemidiaphragm, and pulmonary atelectasis suggest renal injuries.

VII. Special Studies

Ultrasound is quick and inexpensive for the detection of fluid and identification of abnormal architecture; IVP/urethrogram reveals distortion of normal architecture or extravasation of dye following renal or bladder injury (Level of Evidence B, other evidence).[2,17] Retrourethrogram should be done first if a urethral tear is suspected, as further

damage can follow catheter insertion. Computed tomography (CT) scan with oral and intravenous contrast is probably the best imaging modality to investigate renal and duodenal injuries; it is also good for evaluation of pancreatic, liver, and splenic injuries. Radionuclide studies are useful for hepatic and splenic ruptures; endoscopy and barium swallow, for duodenal injuries. Angiography is indicated for continued bleeding.

VIII. Diagnosis

Visceral injury.

IX. Differential Diagnosis

Nonvisceral trauma (e.g., sprain, strain, fracture, contusion).

X. Treatment/Rehabilitation

A. Initially: Stabilize vital signs with two large-bore (14-gauge) intravenous lines and Ringer's lactate, perform immediate laparotomy if diagnoses are unclear and patient is not stable.

B. Long-term: Expectant observation should include bed rest and sequential serial exams.[18] Splenic, liver, and renal injuries (most intracapsular and 50% of extracapsular), in absence of shock, expanding hematoma, or free extravasation of the urine on intravenous contrast CT, usually do well nonsurgically. Renal pedicle, transcortical, or transcapsular lacerations associated with urine extravasation or massive hemorrhage require urological repair. Small retroperitoneal bladder ruptures may be treated with a Foley catheter and suprapubic cystostomy. Intraperitoneal and large retroperitoneal tears require operative closure. A minimum of 6 weeks is required for all individuals who are treated nonsurgically or surgically before they return to sports-related activities including those suffering hematuria. A 6-month minimum should be enforced for return to play in collision and contact sports.

Surgical consultation: indicated for all cases of abdominal trauma.

XI. Complications

Usually minimal with rapid and efficient field management, transport, diagnosis, and intervention. Prognosis worsens in cases of delayed recognition, massive trauma, or ineffectual treatment plan.

XII. Prevention

Use of protective equipment (pads, vests, etc.) in contact and collision sports, proper techniques (avoidance of body slams in wrestling, use of helmet or elbow blocks in football, being alert and on guard for a tackle during a pass or reception), improved supervision and enforcement of regulations (in cycling, horseback riding, downhill skiing, and sledding), and prohibition of contact and collision activities for patients with infectious mononucleosis.[18]

NONVISCERAL INJURIES

Non-organ injury is the most common type of abdominal trauma and includes abrasions, hematomas, contusions, and strains.[4,19] The majority of these injuries are not serious and can be managed according to the guidelines provided in Chapter 34. Rectus sheath hematomas can be distinguished from more serious intra-abdominal pathology by tensing the abdominal musculature with straight-leg raising. Muscle hematomas remain painful, palpable, and visible; intraperitoneal ones do not (positive Carnett's sign). Multiple, recurrent, or expanding hematomas should suggest a blood dyscrasia (leukemia, idiopathic thrombocytopenic purpura) or rupture of an epigastric vessel. Surgical exploration and vessel ligation are required in the latter case.

Muscular contusions may be difficult to distinguish from visceral injury and may even coexist. Tenderness is usually only over the site of the contusion and worsens with contraction (Carnett's sign), but improves after the muscle remains tense.[19] Contusions do not cause referred pain! Such injuries must be distinguished from strains of the abdominal muscles, common in gymnastics, pole vaulting, and high jumping, due to excessive twisting and torque forces. Grade II and III injuries may be associated with small nodular swellings (fiber rupture and hemorrhage).

Blows in the area of the solar plexus, particularly when an athlete is not on guard, can "knock the wind out of" the person. The epigastric contusion causes reflex paralysis of the diaphragm, cessation of respirations for several seconds, and a feeling of impending death. The airway should be checked for a foreign body (e.g., mouthpiece, turf, tongue). The athlete should be removed from the field; should be made comfortable by loosening the uniform, bending his or her knees, and applying a cold soak to the forehead; should be

reassured; and should be examined thoroughly before allowing further play.

A "side stitch" can occur in any untrained athlete who runs (Chapter 57). The pain is sharp, stabbing, and subdiaphragmatic, usually on the right side but sometimes on the left. If possible, the athlete may be able to "run through" the pain by deep or pursed-lip breathing. With persistent, severe pain stretching the abdominal area in flexion and extension may help. As fitness levels improve, stitches become less common.

As a reminder, the vigilant team physician must bear in mind that any abdominal wall injury can be associated with and should suggest the possibility of more serious internal injury. A unilateral rectus strain can mimic appendicitis, and splenic injuries have occurred following a solar plexus blow.

GENITAL CONTUSION INJURIES

I. Epidemiology
Contusions are a common, under-reported injury in contact and collision sports, particularly when protective equipment is not worn.

II. Mechanism of Injury
Direct blow from another player or a piece of equipment (e.g., straddle injury).

III. Anatomy
Hematoma or hematocele formation in the loose tissues of the scrotum and vulva; straddle injuries often are associated with rupture of the bulbous/membranous urethra, pelvic diastasis, or extravasation of urine into the perineum, genitals, and lower abdominal wall.

IV. Symptoms
Intense pain and discomfort in the lower abdomen, genitals, inner upper thighs, and perineum.

V. Signs
Swelling, tenderness, discoloration (ecchymosis), and a fluctuant mass that does not transilluminate.

VI. Laboratory/Radiology
None

VII. Special Studies
Ultrasound is sensitive for rupture; radionuclide scan is "cold" if torsion is suspected.

VIII. Diagnosis
Contusion.

IX. Differential Diagnosis
Torsion of the spermatic cord, testicular rupture, hematoma, hematocele, urethral rupture (bulbous or membranous).

X. Treatment/Rehabilitation
A. Initially: Dropping the patient from approximately 6 inches above the ground reduces cremasteric muscle spasm; use RICE (rest, ice, compression, elevation) and aspiration if necessary.
B. Long-term: Usually none is required.

XI. Consultations/Indications
Urologist if the extent of injury is unclear

XII. Prevention
Regular use of an athletic supporter in all sports and a cup in collision and contact sports.

MISCELLANEOUS INJURIES

The groin is involved in 2 to 5% of sports injuries.[20] Sports hernia or groin disruption is caused by posterior inguinal wall weakness, external oblique aponeurosis, or conjoined tendon tears and dehiscence of the inguinal and conjoined ligament, producing a direct or indirect hernia. The athlete complains of subtle, progressively worsening, diffuse, deep groin pain that is worsened by maneuvers that increase abdominal pressure. The pain may radiate along the perineum, rectus muscles, and inguinal ligament and 30% of the time testicular pain is noted. No clinically detectable inguinal sac is observed on physical exam, but resisted hip adduction may produce pain in 65% of cases. The differential diagnosis should include distal rectus strain/avulsion, back, hip joint, and genitourinary disease. While laboratory and imaging tests can rule out alternative diagnoses a conservative treatment trial should be tried for several weeks. A 90% success rate follows operative exploration and repair (Level of Evidence B, other evidence).

An atypical lower abdominal pain syndrome (refractory pain and paresthesias) has been described among elite hockey players. The conventional investigative workup is consistently negative. Tearing of the external oblique aponeurosis and muscle associated with ilioinguinal nerve entrapment are operative findings, which when repaired and followed by aggressive physiotherapy result in complete return to play.[21] "Athletic pubalgia" describes severe lower abdominal or inguinal pain in high-performance athletes,

particularly males.[22] It appears to be due to pubic symphyseal sprain from abdominal hyperextension and thigh hyperabduction. Surgical correction to strengthen the anterior pelvic floor is the procedure of choice.

"Skier's douche" occurs when high-speed water skiing results in irritation and rupture of the vagina or rectum. A water jet can enter the vagina, uterus, and fallopian tubes, leading to salpingitis, peritonitis, and abortion several days after the skiing event. Novices are particularly prone to the injury because they squat when trying to start skiing. The use of a neoprene wet suit while water skiing is preventive.

Repetitive microtrauma to the pudendal nerve (e.g., from cycling) can cause ischemic neuropathy of the penis or priapism. The paresthesias and priapism normally resolve after the race or tour. Prevention can be achieved by use of a furrowed saddle and by not squeezing the saddle when cycling uphill.

While torsion of the spermatic cord is not a sports injury *per se*, it may occur in association with recreational activity. The diagnosis must be kept in mind whenever a child or adolescent athlete has scrotal pain or swelling. The pain may be insidious or abrupt and excruciating and may be located in the abdomen, pelvis, or testes. The athlete may vomit and collapse. Examination may reveal local tenderness, edema, and hyperemia and adherence of the scrotal contents to the overlying skin. The cord and vas deferens will be inseparable. Elevation of the contents increases the pain with torsion but usually reduces it with epididymitis. Radionuclide scanning is "hot" during epididymitis but "cold" with torsion, due to hypoperfusion. If external manipulation cannot reduce the torsion, immediate bilateral orchiopexy is indicated.

SUMMARY

Like the chest, the abdominal area is subject to frequent injury. While most trauma is relatively mild, serious visceral injury to the liver and spleen can rarely occur in collision and contact sports. Careful vigilance on the part of the sports physician and a trauma prioritization plan based on advanced trauma life support (ATLS) protocols are paramount.

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44 SPINAL INJURIES*

Kenneth M. Bielak

SPORTS INJURIES OF THE SPINE

Spine complaints are a major cause of concern in both sports and industry. All age groups and body types sustain back injuries. Stars of some sports such as golf, tennis, and baseball have been incapacitated by low back pain. Other sports such as football, diving, and wrestling have their share of acute cervical and lumbar spine injuries.[1]

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High-velocity sports such as motor sports carry the highest risk for potentially life-threatening injuries to the spine.

Epidemiology

More than 80% of all Americans will have spine complaints in their lifetime.[1] Low back pain is the most common complaint and is seen typically in the third through sixth decades of life. It is the most common cause of disability in patients under 45 years of age. Cervical spine injuries become more common in the later adolescent years as speed and power play increasing roles in sports. Sports and recreational spinal-cord injuries have nearly doubled over the last several decades: shallow-water diving accounts for over one half of the injuries, followed by motor sports (9.8%) and hockey (5%).[2,3] In football, 90% of deaths are due to head and neck trauma, and this figure does not take into account the nonfatal morbidities involved with severe neck injury, which have been increasing.[4-7] Thoracic spine problems are more common in the mid-adolescent years; as the young athlete becomes more active, the immature spine is unable to tolerate the work demands on it. Backache is the second leading cause of work absenteeism after flu and colds, but it is unknown what percentage of this is truly discogenic back disease and how this really impacts on the sporting world. In a retrospective analysis of the 1996 portion of the Medical Expenditure Panel Survey, almost 9%, or 23 million, individuals experienced back pain, 4 million experienced work-related back pain, and approximately 2%, or 5 million, experienced missed work days due to back pain. This incidence of back pain resulted in total expenditures of $23 billion just for the medical care, which represents just a fraction of the total economic cost.[8]

Anatomy

The axiom "form follows function" is most evident in the spine. The spine acts as a suspension mechanism when it is parallel to the cyclist's roadway, it acts as a high-tension spring coil when it is twisted in wrestling competition, and it acts as a tower with multiple levers for various resistance loads in the weight lifter hoisting a heavy load. The normal curves (cervical lordosis, thoracic or dorsal kyphosis, and lumbar lordosis) help facilitate the motion and support needed for that region. The vertebral body and facets are designed for stacking, while the bony canal protects the spinal cord. The appendages (the spinous and transverse processes) make natural lever arms for the dynamic (muscles) and static (ligaments) stabilizers.

The disc is comprised of a central nucleus pulposus, which is circumferentially surrounded by the annulus fibrosus. Its water content is high (75%), and the connective tissue arrangement is designed to withstand repetitive loading. The annulus is arranged in concentric lamellae. Each layer has fibers oriented 120° to the adjacent layer, enhancing strength and durability. The interface of the disc and the vertebral body is tightly adherent tissue that dictates end points of motion such as torque and bending. The disc is located superior to the concomitant exiting nerve root. The vertebral body is composed of spongy trabecular bone which allows it to respond to different stresses. Vertical and oblique stress lines are evident in the vertebrae.

The body of the vertebra and the discs comprise the anterior joint. The paired facets of the spine comprise the posterior joint. The bilateral facets, together with the vertebral body and discs, comprise a three-joint complex. The facet joints are oriented in different planes, depending on the spine segments. Motion at any one level is dictated by the surface area and inclination of the facets. The facets are positioned to give an interlocking effect that enhances stability and dissipates stress. The individual facet joint is a diarthrodial joint having a meniscoid interface and which includes a meniscal attachment. As in other joints, it can attrite from overuse. The triple-joint complex can bear up to one half of the compressive loads delivered at each lumbar level.

The "hardware" of the spine is supported by the "software." Motion is balanced with stability by the layered arrangement of the muscles. An imbalance produces either instability, when motion predominates, or stiffness, when stability dominates. Flexion balances extension. The extrinsic (i.e., rectus) and intrinsic (i.e., psoas and iliacus) flexors contract unilaterally, causing ipsilateral side bending, or they can contract bilaterally, causing forward flexion. Motion at the hip is responsible for more forward flexion than are the intrinsic flexors. Lumbar lordosis is maintained and sometimes increased during flexion.

The lumbar extensors are divided into the superficial, intermediate, and deep groups. The superficial extensors are made up of the sacrospinalis or erector spinae muscle group — a large muscle mass in the sacroiliac area. Much like a suspension bridge cable system, the three columns of erector spinae muscles help keep the spine erect. The intermediate group (i.e., multifidus) is oriented obliquely so that

unilateral contraction will cause theoretical pure side bending, although in actuality side bending occurs with some rotation. Bilateral contraction causes backward extension. The deepest layer of extensors is made up of two groups of muscles: rotators and intertransversarii. As with the other layers, unilateral and bilateral contractions work in concert with the contralateral unit to cause side bending or extension, respectively. Even with flexion, the extensor groups work in an eccentric fashion to control the primary movement.

Spinal motion is described as flexion or forward bending, extension or backward extension, side bending or lateral flexion, and rotation or twisting. Usually rotation accompanies side bending as coupled motion. Motion is dictated by several variables: (1) orientation of the facets, (2) surface area of the facets, (3) disc resistance to motion, (4) disc thickness, and (5) local soft tissue attachments. In the normal lumbar spine, most flexion and extension occurs between L4–L5 and L5–S1.

The spine must balance several "moment arms" comprised of varying resistances or loads. The ligaments and muscles generate tension, and work must be done by the muscle to support the human chassis. The body's weight and any mass the body carries must be balanced by tensions generated by the ligament and muscle systems. The added mass of a gravid belly or the added mass of free weights must be balanced by increased work of the spine musculature. With overuse this counterbalance mechanism weakens and its components will attrite, degenerate, tighten, inflame, or become unstable. In addition to the extremes of physiologic motion, compression and angular force can also be a mechanism of injury.

Eight cervical nerves are associated with seven vertebral bodies. Nerves C1–C8 subserve dermatomal and specific motor distributions to the upper extremity. Nerve root compromise is usually secondary to herniation of the nucleus pulposus. The facets can be affected segmentally along with changes in the disc.

Thoracic and lumbosacral nerve roots exit inferiorly to the vertebral body and disc with which they are associated. The sciatic nerve trunk lies adjacent to the lower one third of the sacroiliac joint, separated only by the intervening capsule. Hypertrophic spurs and an inflamed sacroiliac joint can irritate the lumbosacral plexus enough to cause a radiculopathy similar to disc herniation. The conus medullaris is generally at the level of L1–L2 in the adult.

The vascular supply of the cervical spine originates from the aorta; the vertebral artery courses through the vertebral foramina and as a result is close to the facets. The carotid artery is located anterolaterally to the cervical spine. The posterior longitudinal ligament, posterior intervertebral disc, lateral and anterior aspects of the annulus fibrosus, posterior facet joints, and interspinous ligaments are also innervated.

Pathogenetic Mechanisms of Injury

The more serious injuries to the spine are the result of sudden acceleration or deceleration forces that impact the spine. Chronic, more subtle injury patterns result from overuse, muscular imbalances, or repetitive stresses that cause strain, sprains, or avulsion injuries to focal segments of the spine and its attachments. In the cervical spine, segmental motion is best appreciated at different levels depending on the particular motion: nodding, at occiput and C1; rotation, at C1–C2; flexion/extension, at C5–C6 and C6–C7; side bending, at the mid-cervical segments. Thus, rotational injury should prompt investigation for C1–C2 instability, whereas flexion/extension injury should suggest possible C5–C6 or C6–C7 trauma.[5,6] Most fracture or dislocation injuries to the cervical spine are due to vertical (axial) loading. Slight flexion removes the protective cervical lordosis, resulting in a segmented column that buckles with a compressive load.

History

In addition to the typical inquiry of what, how, and when, the history of spinal problems should be extracted in a methodical way, building a story of chronologic events rounded out by associated historical factors. Any of the available pain questionnaires and pain drawings will help qualitate the functional capacity of the person as well as changes in lifestyle — a lifestyle made to accommodate pain. Much overlap exists between mechanisms of injury and physical stresses. Vehicular vibration as seen in auto racing, aircraft, forklifts, and heavy machines can attrite the spine. Unusual mechanisms during the sport can cause injury, such as landing in an axial loading pattern while rebounding during a basketball game. Other apparent risk factors such as mild scoliosis, leg-length discrepancy, and increased lordosis are not associated with low back pain. Athletes with conditions such as trisomy 21 (Down syndrome), however, have a high incidence of cervical spine instabilities. Personality disorders such as adjustment reactions and depression are common findings in

TABLE 44.1
Commonly Tested Nerves and Nerve Roots

Disc Space	Nerve Root	Cutaneous nerve	Motor	Sensory	Reflex
C4–C5	C5	Axillary	Deltoid	Lateral shoulder	None
C5–C6	C6	Musculocutaneous	Biceps	Lateral forearm	Biceps
		Radial	Brachioradialis	Lateral forearm	Brachioradialis
C6–C7	C7	Radial	Triceps	Dorsal web between thumb and index finger	Triceps
T8–T10	T9–T11	—	Abdominal	T8–10 dermatome	Upper abdominals
T11–T12	T12, L1	—	Musculature	T11–12 dermatome	Lower abdominals
L1, L2	L2, L3	—	Cremaster	Anterior-lateral thigh	Cremasteric
			Anal muscle		Superficial anal
L2, L3, L4	L2, L3	Femoral	Quadriceps	Anterior-lateral thigh	Knee (patellar)
		Obturator	Adductors	Medial leg	Adductor
L4, L5, S1, S2	L5–S3	—	—	Leg and foot	Plantar
		Sciatic	Hamstrings	Posterior thigh	Hamstring
S1,2	S2	Tibial	Gastrocnemius	Heel, lateral foot	Ankle (Achilles)

athletes with chronic low back problems but are not prospective risk factors. The risk factors for low back pain in general are based on consensus opinion, as no formidable prospective analyses exist and systematic reviews can only implicate.[9]

Clinical Examination

Cervical Spine

Inspection of the neck requires full attention to anterior, posterior and lateral aspects, which are noted as the patient first enters the exam room. The important landmarks are identified, then palpated for signs of underlying pathology. The range of motion of the neck is tested through flexion, extension, lateral bending, and rotation, being mindful of limitations, asymmetry, and pain with motion. Muscle testing of the intrinsic muscles of the neck involves maximal resistance to the muscles that govern the typical neck motions. Nerve testing of the cervical roots is vitally important (Table 44.1). Deficits revealed by examination may be confirmed by additional neurodiagnostic and electrophysiologic testing.

Special Tests

- *Distraction* — Cephalad traction on the head may relieve pain caused by stenotic foramina, as this motion will widen the foramen and decrease the pressure on the facet's joint capsules.
- *Compression test (Spurling's maneuver)* — Caudad compressive force applied to the top of the cranium may elicit pain from the compressive forces of a narrowed foramen, muscle spasm, or increased pressure on facet joints.
- *Valsalva test* — Increased intrathecal pressure may increase pressure against any space-occupying lesion such as a herniated disk or tumor, resulting in symptoms (e.g., pain) or signs (e.g., weakness).
- *Swallowing test* — Difficulty with this maneuver may indicate spinal pathology in the proximity of the oropharynx, such as tumor, osteophyte formation, or bony protuberance of the anterior cervical spine.
- *Adson test* — This test is performed to elicit possible impingement of the subclavian artery. The examiner monitors the radial pulse for a deficit while the head is rotated to the ipsilateral side; additionally, the pulse is monitored while the arm is abducted, extended, and externally rotated. Any pulse deficit suggests compression of the subclavian artery by an extra cervical rib or by a contracted and hypertrophied scalenus muscle that surrounds the artery.

Lumbar Spine

The lumbar spine is inspected for signs of trauma such as ecchymosis and for stigmata such as café-au-lait spots or hairy patches that may herald underlying pathology. Exaggerated lumbar lordosis may result from weakened abdominal musculature. Loss of lordosis may be from paravertebral muscle spasm. Major body landmarks of the lumbosacral spine should be routinely identified and palpated, looking for underlying pathology. The lower extremities should be examined, as structural (anatomical short leg) and functional (pronated feet) problems are a frequent cause of back pain. Range of motion begins with observation of gait then testing back motion, including standing flexion, extension, lateral bending, and twisting (rotation), looking for limitations to motion or restrictions caused by pain and/or muscle spasm. Muscle testing allows the examiner to compare the relative strengths of the major muscle groups of the lower extremity, looking for weakness, atrophy, or asymmetry. The neurovascular exam is then completed by checking segmental areas for response to light touch, pinprick, temperature, and vibration and by checking the major pulses of the lower extremity (see Table 44.1). Finally, pelvic, rectal, and scrotal exams are an important part of the back evaluation.

Laboratory/Radiology Evaluation

With the exception of neck or back trauma, radiographs (antero-posterior, lateral, obliques) are often not necessary in the acute phase. Persistent and unremitting pain unresponsive to conservative treatment, back pain with persistent fever or unexplained weight loss, non-mechanical rest pain, significant trauma, history of cancer, or neurologic deficits are some indications for obtaining radiographs (Level of Evidence C, consensus opinion).[10–12] Radiographs may reveal fractures, infections, and tumors, as well as some pars defects but with quite limited sensitivity. Radiographs can demonstrate degenerative disc narrowing but are unreliable for evaluation of disc herniation.

Magnetic resonance imaging (MRI) is preferred in evaluation of most cervical, thoracic, and lumbar disorders, including disc disease, radiculopathies, myelopathies, infection, and tumor. MRI is the procedure of choice in the diagnosis of cord abnormalities including edema and hemorrhage. Advantages of MRI include lack of radiation, superior soft-tissue contrast, multiplanar imaging, and ability to image large segments of the spine. Disadvantages of MRI include susceptibility to motion and other artifacts and evaluation of calcifications and dense cortices, as well as expense. Important contraindications include pacemakers, cochlear implants, magnetically operated implants, metallic foreign bodies in the eye, and some intracerebral aneurysm clips. Imaging usually must be delayed 6 to 8 weeks after intravascular filter, stent, or coil placements.

When MRI is contraindicated, computed tomography (CT) or CT–myelogram is usually employed. CT excels in evaluation of calcific or osseous processes (especially osseous processes involving the cortices). Thus, CT is the first-line modality for acute fracture evaluation and exquisitely defines facet joint disease, osseous foraminal narrowing, and pars defects. CT or CT–myelography is also the modality of choice for most spinal disorders when MRI is contraindicated. Advantages of CT include high resolution, speed, and multiplanar imaging potential. Disadvantages of CT scanning include a substantial radiation dose and less soft-tissue contrast relative to MRI. Myelography is almost never indicated as an isolated procedure, but in combination with CT it remains very useful for evaluation of myelopathies and radiculopathies. This is an invasive procedure and is employed most often in settings where MRI is contraindicated or to further explore issues unanswered by MRI or CT.

The radionuclide three-phase bone scan is useful in the evaluation of suspected stress-related injuries, including lumbar pars interarticularis stress injuries. The bone scan typically becomes positive about the same time an overuse injury becomes symptomatic. Bone scans are highly sensitive in detection of stress fractures, infection, and most tumors but lack specificity. Anatomic localization is markedly improved if single-photon emission computed tomography (SPECT) is employed. Electromyography (EMG) and nerve conduction velocity (NCV) studies help localize herniated disc syndromes and root and peripheral nerve lesions; however, such testing requires 14 to 21 days for denervation changes to occur before these studies become positive.

HERNIATED NUCLEUS PULPOSUS (SLIPPED, HERNIATED, RUPTURED DISC)

Herniated nucleus pulposus (HNP), together with a whole spectrum of degenerative disc disease, is a well-recognized cause of low back pain.

I. Epidemiology

The incidence of intervertebral disc disorders has been reported to be 0.2 per 1000 persons under 18 years of age, 15.2 per 1000 persons ages 18 to 44 years, and 37.7 per 1000 persons ages 45 to 64 years. L5–S1 is the most common site followed by L4–L5 and L3–L4.[13] Low back pain and discogenic disease are common in gymnastics, swimming, football, weight lifting, wrestling, track, bowling, and racket sports.[14,15]

II. Mechanism of Injury

Acute low back pain can result from sudden jarring insults to the spine such as stepping into a pothole, lifting a heavy load, or twisting the torso against tension. During flexion and rotation, the disc material impacts against the weakest segment — the posterolateral annulus fibrosus. Repeated insults can result in herniation through this area. Other cases may be of slower onset, with increasing pain due to further activity or particular movements involving the back.

III. Anatomy

The disc is contained by intervertebral ligaments, the weakest of which is located posteriorly. Posterior herniations and extrusion of disc material into the spinal canal occur with degeneration of the posterior longitudinal ligaments. Desiccation of the disc will weaken its performance. The pain of the extruded disc is due to the mechanical action of pressure against a nearby nerve root, as well as to local inflammation due to chemical irritation from the substance of the nucleus. Thus, a laterally bulging disc is more likely to be symptomatic than a centrally bulging disc because of the proximity of the nerve root laterally.

IV. Symptoms

- Pain that radiates from the sacroiliac and buttock area to lateral thigh, extends to the lower leg onto the dorsum of the foot in the first and second web
- Occasional referred pain to hip and knee
- Paresthesia ("pins and needles") along the dermatome
- Radicular pain (electrical shock-like "zing"), usually unilateral but can be bilateral
- Provocation of symptoms when certain postures or activities are prolonged (such as riding in a car, standing, lifting, or physical exertion); increased pain with coughing, sneezing, or straining
- Relief with resting in certain positions (e.g., lying on back with hips and knees flexed, lying on the side or in fetal tuck position, or resting prone with belly supported)

All patients should be asked about any difficulty with control of bowel (rectal sphincter disturbance) or bladder functions; the presence of either is an ominous sign (e.g., cauda equina syndrome).

V. Signs

Localized tenderness, paraspinal muscle spasm, unilateral plantar flexion weakness (difficulty with toe walking), decreased ankle jerk reflex, diminished extension power of extensor hallucis longus (EHL, L5 nerve root).

Tests of Provocation

An increase in symptoms with the test is considered a positive sign. In general, true sciatic nerve compression or injury will elicit more than one of the following positive tests:

A. Tests to stretch the spinal nerve roots
   1. Straight-leg raise (SLR) (supine) — In the supine position, the extended leg is flexed passively at the hip to stretch the nerve root. A significant positive test is typically elicited from 10 to 60°. Under 30 years of age, this test is very sensitive but not very specific (many false positives, usually related to paravertebral muscle spasm). Over 30 years of age, this test is very specific but not very sensitive (usually due to degenerative changes of the spine).
   2. Straight-leg raise (SLR) (sitting) — Same as above, except performed sitting; discordant results may reveal secondary gain etiologies for back pain.
   3. Contralateral straight-leg raise — Very sensitive and specific for all ages when pain is localized.
   4. Lasegue's test — The hip and knee are both flexed to 90°; the knee is then further extended to the degree where pain occurs; additionally, the ankle can be dorsiflexed to determine whether symptoms increase (Spurling's maneuver).
   5. Bowstring test — During a SLR test, manual compression of the

popliteal fossa will exacerbate the pain of the stretched sciatic nerve.

B. Tests to increase intrathecal pressure
   1. Valsalva maneuver with terminal cough — Successful reproduction of pain is diagnostic with this maneuver. This can be augmented by manually, yet partially, occluding the external jugular veins, causing an increase in intrathecal pressure. Once the patient's face becomes plethoric, he or she is asked to perform the Valsalva maneuver and then cough at the climax of this maneuver to determine whether this reproduces the pain.

C. Tests to assess sacroiliac integrity
   1. Pelvic squeeze — The examiner's hands are placed over the anterior superior iliac spines (ASIS) of the pelvis and squeezed toward each other. This maneuver opens the sacroiliac (SI) joint posteriorly, provoking pain.
   2. Pelvic rock — The SI joint is stressed by placing a distraction or abduction force on the ASIS, which closes or pinches the SI joint.
   3. Direct sacral pressure — With patient lying prone, direct sacral pressure is applied in an anteroposterior direction, causing anterior displacement of the SI joint.
   4. Gaenslen's test — The supine patient flexes knees and hip, draws the legs up, and holds that position while the contralateral leg is allowed to actively hyperextend over the edge of the table. Rotation of the hemipelvis occurs, increasing the contralateral sacroiliac torsion and SI joint pain (a positive test).
   5. Patrick (FABERE) test — The hip is flexed, abducted, externally rotated, and extended, with the heel allowed to rest over the opposite knee. The examiner then presses the ipsilateral knee downward while securing the contralateral hip with the opposite hand. A positive test suggests hip or SI joint pathology.

D. Tests of segmental innervation are based on the fact that the anterior abdominal musculature is innervated by the same roots that supply the segmental areas of the erector spinae group. Weakness of the abdominal musculature may be a cause or a component of the overall low back pain picture, and can give clues to the dysfunctional spinal level. Abdominal muscle strength is tested by asking the patient to raise both heels off the table to a height of 6 inches. Strong abdominal muscles should be able to maintain this for at least 30 seconds. Umbilical winking with light cutaneous stimulation can signal weakness of the musculature of the contralateral side, giving clues to test the paraspinal musculature of the weak side. Palpation of the paraspinal areas for signs of inflammation such as hyperemia, warmth, hyperesthesia, edema, tenderness, and skin rolling can also be done. The affected dermatome sometimes provides additional subtle clues to the examiner.

Referred pain should be distinguished from radicular pain (Table 44.1). As previously mentioned, pelvic, rectal, and scrotal exams also should be part of the back evaluation.

VI. Laboratory/Radiology

For recurrent low back pain, complete blood count, erythrocyte sedimentation rate, liver and renal biochemistries, and blood sugar and serologic tests help rule out infections, kidney disease, diabetes, calcium dysphysiologies, and metabolic bone disease. The second tier of diagnostic tests includes the rheumatoid factor, antinuclear antibody, serum protein electrophoresis for multiple myeloma, acid phosphatase, prostatic-specific antigen (PSA) prior to digital manipulation of the prostate, and thyroid function tests. Urinalysis with urine protein electrophoresis can be helpful to look for urinary tract infections or multiple myeloma. As a general rule, radiologic and other imaging studies should be done on patients over 50 and on younger patients whose low back pain lasts more than 6 weeks. Points to remember when interpreting lumbar radiographs include:

- Discs should normally be as wide or wider as one progresses distally in the lumbar spine. L5–S1 is the exception, often being narrower than L4–L5 as a normal variant in the absence of disc disease.

- Facet joints should not be narrowed and sclerotic as in osteoarthritis.
- The pars interarticularis should be intact.
- No horizontal translation of one vertebral body on another should be evident, as in spondylolisthesis.
- No scoliosis or extremes of lordosis should be evident. Reversal of the normal lordotic curve may be a normal postural finding or due to muscle spasm.
- The interpedicular distance should not be focally widened, as can occur with fracture or canal tumor.
- No lytic compromise of cortices or blastic lesions should be present.
- No excessive demineralization should be present.
- No evidence of calcified abdominal aortic aneurysm anterior to the lumbar spine should be seen.
- The sacroiliac joints should be examined for gross abnormalities on the antero-posterior view.

VII. Special Studies:

Electromyography and nerve conduction velocities show 90% sensitivity, with 38% specificity. They are best for documenting motor lesions and poor for sensory disturbances; also, poor results are obtained with acute problems.

VIII. Diagnosis

Herniated nucleus pulposus level _____.

IX. Differential Diagnosis

Local infection, fracture, tumors (such as multiple myeloma, sarcoma, lymphoma, chordoma, and metastasis from breast, lung, thyroid, kidney, and prostate), benign bone tumors (e.g., osteochondromas, osteoid osteomas, aneurysmal bone cyst, osteoblastoma, and hemangioma), inflammatory causes (e.g., rheumatoid arthritis, ankylosing spondylitis, Reiter's syndrome, psoriatic arthritis), degenerative causes (e.g., disc degeneration without herniation, spinal stenosis, and osteoarthritis), osteoporosis, strains and sprain, and extraspondylitic diseases (e.g., piriformis syndrome, osteoarthritis of the hip, torsional deformities of the lower limb, peptic ulcer disease, cholecystitis, colitis and polyneuritis, aortic aneurysm, uterine myoma, and prostatitis).

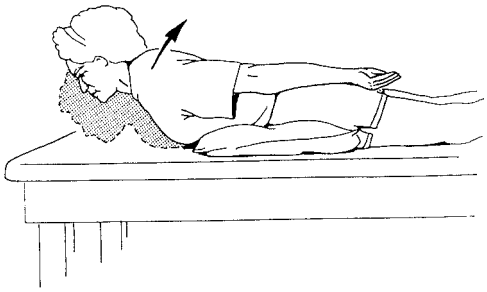
Pearl: Muscle strains radiate to the thigh, usually stopping at the knee. Radicular pain is electric-like and extends into the leg and foot.

X. Treatment

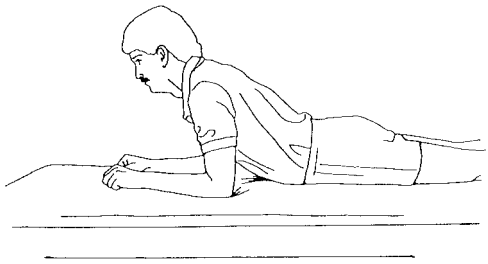
In treating all low back pain, it is worthwhile to keep in mind that, no matter what is done, 90% of all cases will probably improve in 6 weeks. The mainstays of treatment include:

A. Relative bedrest — Unload the spine for 3 or 4 days.
B. Pain management — Minimize narcotics, maximize nonsteroidal agents and physical modalities such as ice and massage.
C. Spasm management — Use ice, postural changes, manipulation, and proprioceptive neuromuscular facilitation (PNF).[16]
D. Flexion exercises can help maintain suppleness, but these aggravate disc disease so extension exercises are usually recommended (Figure 44.1).[16]
E. Hyperextension maneuvers can reduce symptoms.
F. Behavioral modification is helpful in reassessing expectations and to help control drug-seeking behavior.
G. Immediate referrals include the diagnosis of cauda equina syndrome (bilateral neurological deficits associated with bowel or bladder function impairment), progressive motor symptoms or paralysis, evidence of infection, abscess or epidural hematoma, or patients with a history of malignancy and new evidence of nerve entrapment.
H. Epidural steroid injection (ESI) is generally reserved for patients demonstrating limited response to conservative measures of oral medication, physical therapy, and other non-invasive measures (Level of Evidence C, consensus opinion).[17]

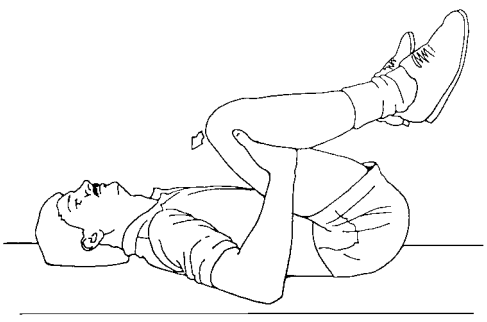
Consultation: Refractory cases may require surgical intervention (laminotomy, laminectomy, discectomy). Standard discectomy produces increased self-reported improvement at 1 year, but not at 4 and 10 years when compared to conservative treatment. No significant differences have been observed in clinical outcomes with standard discectomy vs. microdiscectomy. Adverse effects are similar with both procedures.[18] Intensive multidisciplinary bio-psycho-social rehabilitation with a functional restoration approach improves pain and function.[19]



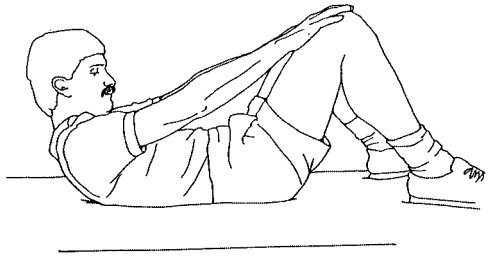
(A) *Upper back:* Place pillow under waist. Gently raise arms off table 4 to 6 inches, pinch shoulder blades together, and lift shoulders and head off table. Keep head forward, looking at floor. Do not hold breath. Hold 3 to 5 counts and relax. Perform ____ repetitions, ____ times daily. Progress to ____ repetitions, ____ times daily.



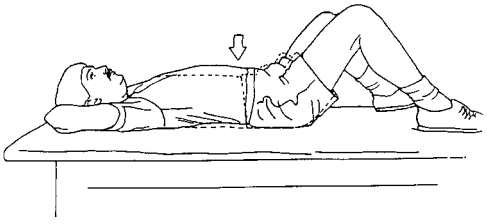
(B) *Prone on elbows exercise:* Lie on stomach with arms bent over head. Raise head and upper back by pulling elbows in so you are propped up on your forearms. Keep low back relaxed. Hold for ____ seconds. Lower. Repeat. Perform ____ times, ____ times daily.



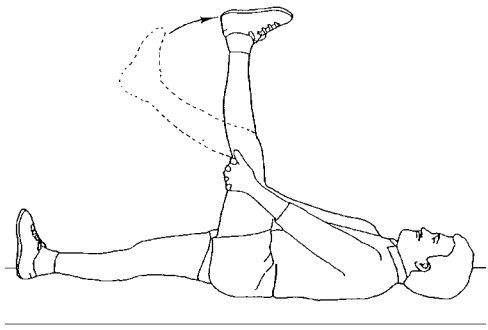
(C) *Double knee to chest:* Lie on back with knees bent. Clasp one hand underneath each knee. Gently pull both knees to chest. Hold for ____ seconds. Relax, but do not release knees. Repeat. Perform ____ times, ____ times daily. Increase to ____ times, ____ times daily.



(D) *Abdominal curls:* Lie on back with knees bent. Perform a pelvic tilt, then reach with both hands toward knees. Hold for ____ seconds. Remember to exhale as you sit up. Lower slowly. Relax. Repeat. Perform ____ times, ____ times daily. Increase to ____ times, ____ times daily.



(E) *Pelvic tilt:* Lie on back with knees bent. Tighten stomach muscles and tilt pelvis so lower back becomes flat against the floor. Hold for ____ seconds. Relax. Repeat. Perform ____ times, ____ times daily. Increase to ____ times, ____ times daily.



(F) *Supine hamstring stretch:* Lie on back with one leg straight. Clasp both hands under the other knee with hip bent 90°. Slowly straighten knee to feel a gentle stretch in back of thigh. Keep toes pulled back toward you. Hold for ____ seconds. Relax, but do not release leg. Repeat as indicated with both legs. Perform ____ times, ____ times daily.

Figure 44.1 Common flexion and extension exercises for the back.

XI. Complications
Further extrusion of the disc material with increased symptoms and progressive and permanent nerve damage resulting in loss of bladder and bowel control and paralysis.

XII. Prevention
A. Strengthen the abdominal musculature.
B. Increase hamstring flexibility.
C. Reduce body fat content below the 20 to 25% range.
D. Increase aerobic power with prolonged exercises such as walking, running, swimming, cycling, and Nordic skiing.
E. The "hot disc" group of exercises can be used on an as-needed basis.
F. Preseason screening and preparticipation sports placement may be helpful.

SPONDYLOLYSIS AND SPONDYLOLISTHESIS

I. Epidemiology
Spondylolysis is generally due to a hyperextension stress fracture from extension activities prior to skeletal maturity. Spondylolysis is the most common cause of spondylolisthesis. The incidence of spondylosis is 63% in diving; 32 to 36% in gymnastics, wrestling, and weightlifting; 23% in track and field; and 5% in the general population.[20,21]

II. Mechanism of Injury
Repetitive microtrauma from shear, longitudinal loading, and hyperextension of the vertebral column (e.g., gymnastics, swimming, rugby, American football, weightlifting, rucksacking, overhead lifting and overhead occupations such as painting, carpentry, and electrical work) (Figure 44.2).

III. Anatomy
Spondylolysis is a bony defect in the pars interarticularis, once thought to be congenital but now taken to be an acquired defect or stress fracture to this inherently weak portion of the vertebra.[20,21] It may be multilevel and bilateral. Spondylolisthesis is the sliding of one vertebra over the top of another due to loss of the posterior locking mechanism. Pathologically, spondylolisthesis may be isthmic (spondylolytic), dysplastic (congenital deficiency of the inferior fifth lumbar or superior sacral facets or both), degenerative, traumatic, or pathologic. Isthmic subtypes include acute fractures and repeated microfractures (most common) of the pars interarticularis. Spondylolisthesis usually involves L5 sliding over the sacral promontory.

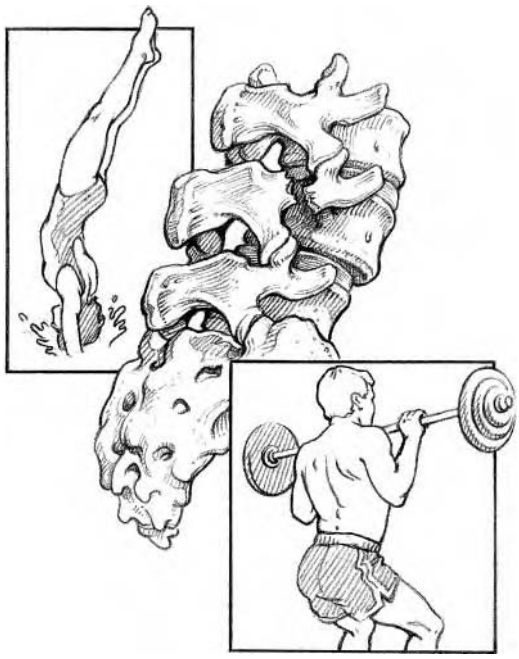


Figure 44.2 Hyperextension in diving, weightlifting, and similar activities can fracture the pars interarticularis.

IV. Symptoms
The most common complaint is that of a recurrent dull ache, usually localized, which can be provoked by extension activities such as an arabesque maneuver and is accompanied by stiffness afterwards. The stiffness is usually relieved by rest but may be worsened. An outstanding feature of spinal stenosis from slippage (spondylolisthesis) is neurogenic claudication (pseudoclaudication); pain is provoked by walking but relieved with sitting.

V. Signs
A. Scoliosis and tenderness due to segmental muscle spasm are commonly seen.
B. Limited range of motion is noted, especially in backward extension and side bending to the affected side.
C. The pain is usually decreased by flattening the lumbosacral lordotic curve with a knee-to-chest maneuver.

D. The pain can be provoked by an arabesque, with very acute localization to the injured level.
E. Hyperesthesia over the affected level can also be found on examination of the skin; the neurovascular exam is usually normal.
F. A stepwise deformity between L5 and S1 indicates spondylolisthesis.

VI. Laboratory/Radiology

At a minimum, a lateral radiograph should be taken in the upright position. The collar on the "scottie dog" (best seen in the oblique view) indicates spondylolysis; however, oblique lumbar views impart easily the largest radiation dose of any radiograph and are not recommended on routine basis. Anterior translation of the vertebral body on the lateral view indicates spondylolisthesis: grade 1, 0 to 25% of the AP diameter of the vertebra; grade 2, 25 to 50%; grade 3, 50 to 75%; and grade 4, 75 to 100%.

VII. Special Studies

A. The radionuclide bone scan is highly sensitive for acute or subacute lumbar spondylolysis. When SPECT tomographic imaging is employed, the abnormal activity can be localized to the pars region, improving specificity.
B. Thin-slice CT imaging with sagittal and coronal reformations is sensitive and specific for diagnosis of pars defects. CT or bone scan is usually performed first when radiographs are inconclusive and symptomatic spondylolysis is suspected.
C. Magnetic resonance imaging may potentially detect early developing cancellous microfractures in the pars region before they are visible on CT and radiographs. MRI also excels at diagnosis of alternative causes for back pain; however, MRI is expensive, and bone scan or CT is usually ordered first to diagnose pars defects. MRI may be helpful in the setting of severe spondylolisthesis to define cord and nerve relationships.

VIII. Diagnosis

Spondylolysis level _____, spondylolisthesis level _____.

IX. Differential Diagnosis

Paraspinal muscle spasm, facet syndrome, herniated nucleus pulposus, and an intertransverse or interspinous ligament sprain.

X. Treatment

Spondylolysis

A. Acute injury (positive scan, negative radiograph): Rest, including avoidance of all activities placing loading forces on the spine for 8 to 12 weeks; use of a modified Boston brace may be required for up to 3 to 6 months and require comanagement with an orthopedic spine surgeon.
B. Semi-acute injury (positive scan, positive radiograph): Rest, unloading, and immobilization with ambulatory antilordotic bracing (i.e., Boston) or body cast (e.g., one thigh pantaloon) for 12 weeks. Brace advantages include continued participation to some degree in sport activity while under treatment and aesthetic consideration. Gradual (over 3 months), progressive return to activity follows, beginning with flexibility and strengthening exercises and concentrating on lumbosacral stabilization program. Return to play is allowed only after a symptom-free rehabilitation program is completed.
C. Chronic injury (negative scan, positive radiograph): Established nonunion unresponsive to immobilization; operative fusion of involved segments for symptomatic athlete who does not wish to forego aggravating activity. Return to play 1 year following successful fusion. Permanent avoidance of upright weight training (e.g., dead lifts, squats, snatch and jerk). Surgical decompression or fusion appears to be no more effective than placebo or conservative treatment (Level of Evidence C, consensus opinion).[22]

Spondylolisthesis

A. Flexibility (hamstring) and strengthening exercises for grades 1 and 2
B. Surgical fusion for poorly controlled symptoms or symptomatic spinal stenosis due to high degrees of slippage (≥grade 3)

XI. Complications
Fibrous union, malunion, nonunion progression to spondylolisthesis, occasionally spinal stenosis with neurogenic claudication.

XII. Prevention
A. Back retraining and abdominal muscle training (antilordotic exercises)
B. Preseason physical screening
C. Job and sport profiling to avoid repetitive lumbar extension

PARASPINAL MUSCLE SPASM AND STRAIN

I. Epidemiology
Muscle spasm and strain accounts for the vast majority of "acute backs" or "mechanical back syndromes."[16] Poor posture and improper lifting mechanics can be found in most histories. Lifting with the back instead of the legs is the usual cause. Unreasonable loads usually are not the culprit, but repetitions of near-maximal loads are dangerous. Recurrent episodes and whole families of back patients can be elicited.

II. Mechanism of Injury
A. Direct blunt trauma, indirect macrotrauma with sudden eccentric load
B. Overuse microtrauma

III. Anatomy
The muscles of the back that are the most commonly injured include the deep and superficial muscle groups. The injury is classified according to Chapter 34 guidelines.

IV. Symptoms
A constant, dull, vague ache poorly localized in the low back area, sometimes centrally located and sometimes on a particular side; usually does not radiate like a radiculopathy but can include posterior thigh pain.[23,24] The spasm can be brought on by improper posture even for just a moment, such as trying to push a refrigerator around to clean behind it, or by maintaining a seemingly innocuous posture, such as sitting in a car for a long trip. Patients generally feel better with rest, warmth, and aspirin. Usually the patient has a history of a prior occurrence.

V. Signs
A. Widened stance and hobbling gait (antalgia); patients are typically observed sitting on a haunch, tripodding while sitting on the table, or standing and leaning against the exam table.
B. Markedly limited active range of motion in all planes and large areas of tender, doughy muscle with some indiscrete areas of spasm are found.
C. Scoliosis and flattening of the lumbar lordosis due to muscle spasm. Limited segmental passive range of motion is observed in side-bending with rotation.
D. No signs of neurovascular embarrassment are observed.
E. Dysesthesia, hypesthesias, and inflammatory skin changes can be found overlying the affected muscle groups.

VI. Laboratory/Radiology
Not indicated with acute strains; consider if no improvement occurs within 2 to 4 weeks.

A. Flattening of the normal lumbar lordosis and functional scoliosis, although an accentuated lumbosacral angle can be a normal variant or due to muscle spasm as well.
B. The bowel gas pattern may show reflex paralytic adynamic ileus in response to the somatic changes.

VII. Special Studies
Not indicated unless there is a question of underlying pathology.

VIII. Diagnosis
Paraspinal strain (biomechanical back syndrome/lumbosacral strain).

IX. Differential Diagnosis
Back pain due to visceral disease (inflammatory bowel disease, diseases of the great vessels and pelvic viscera), herniated nucleus pulposus.

X. Treatment
A. Initially: Pain and spasm management
   1. Bed rest, judicious use of muscle relaxants (Level of Evidence C, consensus opinion)
   2. Analgesics such as NSAIDs, which are effective for short-term symptomatic relief in patients with low back pain (Level of Evidence B, systematic review)[25]

B. Long-term:
   1. Unloading (e.g., walking in a swimming pool, inversion boots)
   2. Physical modalities such as ice, heat, galvanic stimulation, ultrasound, TENS, microwave applications, and cold low-power laser[13]
   3. Rehabilitation
      a. Back schools have better short-term effects than other treatments for chronic low back pain. In an occupational setting, they are more effective compared to placebo or waiting list controls, but little is known about their cost effectiveness (Level of Evidence B, systematic review).[26]
      b. Modify the exercise prescription and activity profile; promote a hardening program (attendance at a back school) and back exercises (e.g., Williams, McKenzie) (see Figure 44.1). Emphasis is on achieving ideal weight, increasing aerobic power, strengthening abdominal muscles, and increasing hamstring flexibility. Physical conditioning programs that include cognitive-behavioral programs can be effective in reducing the number of sick days of workers with back pain, when compared to usual care, but no evidence of their efficacy for acute back pain has been reported.[27]

Consultation: chronic dysfunction

XI. Complications
Recurrent pain/chronic disability.

XII. Prevention
A. Weight reduction if overweight
B. Aerobic power, hamstring flexibility, and abdominal muscle strength improvement
C. Proper sitting, lifting, and lying mechanics

FACET SYNDROME

I. Epidemiology
Facet syndrome usually occurs in people who use poor biomechanics or in people whose posture is held in one position for an inordinate period of time. Usually this occurs with some amount of muscle spasm. Facet syndrome is very common in sedentary people, especially among desk jockeys, phone jockeys, truckers, all sorts of drivers, and athletes involved in marksmanship, bowling, and golf.

II. Mechanism of Injury
A. Direct blunt trauma
B. Overuse microtrauma, especially with axial loading and rotation (e.g., prolonged abnormal position such as hunched over a desk)

III. Anatomy
Inflammation and immobility of one or more facet joints.

IV. Symptoms
A. Loss of range of motion is worse than the pain; can be almost painless if the patient is satisfied with staying in a fixed position.
B. Pain can be a tolerable, dull ache that intensifies to a knife-like stab when the extremes of motion are made.
C. Pain may be ill defined (sclerotomal); with lumbar etiology, it may radiate to the posterior thigh and occasionally to below the knee, mimicking radicular pain, or, with cervical etiology, it may radiate to the shoulder and arm.

V. Signs
A. Bent-over stance with a defensive gait (lumbar facet)
B. Hyperesthesia and hyperemia overlying the dysfunctional segment, with doughy, edematous spinal muscles
C. Marked limited range of motion, specific for the facet involved
D. Usually normal neurovascular exam

VI. Laboratory/Radiology
See paraspinal spasm.

VII. Special Studies
None indicated.

VIII. Diagnosis
Facet syndrome level _____.

IX. Differential Diagnosis
Pars interarticularis defect, intervertebral ligament sprain, paraspinal muscle strain, or any mechanical back etiology.

X. Treatment
A. Initially:
   1. Ice massage, analgesics, and NSAIDs
   2. Bed rest in the prone position with a small pillow under the umbilicus (lumbar facet); cervical collar (cervical facet).

B. Long-Term: Focus on the surrounding soft tissue to increase the flexibility and vascular supply, and usually the facet will fall into its easy normal or neutral position. This is an oversimplified explanation of this very complex method of care:
   1. The soft tissue is prepared and then, by effecting a passive range of motion, the joint usually falls into place.
   2. Alternatively, high-velocity, low-amplitude maneuvers in a direction opposite to the fixed position can be effective. That is, if the facet is stuck in flexion, the movement should be toward extension.
   3. Proprioceptive neuromuscular facilitation includes reciprocal innervation to relax tight supporting structures and allow normal motion.
   4. Progressive resistance exercises (PRE) can be used.

Consultation: chronic dysfunction

XI. Complications
Chronic pain/disability.

XII. Prevention
A. Re-education in the proper biomechanics
B. Maintaining proper body weight
C. Strengthening and flexibility exercises

SCOLIOSIS

I. Epidemiology
Scoliosis is more common in females than males (1.25:1), and the ratio increases markedly with degree of curve (7:1 for curves greater than 20°).[19] It is usually found during screening at the middle-school level, ages 11 to 15 years. The incidence of idiopathic scoliosis is 8 to 10%; for curves greater than 10°, the incidence is 2%. Progression of the curve is seen in 15.4% of females with curves greater than 11°.[28]

II. Mechanism of Injury
The two types (both may be aggravated by overuse, trauma, or loading imbalance) are:
A. Nonstructural or flexible due to leg length discrepancy or vertebral osteoid osteoma
B. Structural or nonflexible, which tends to be more severe and not correctable by side bending.
   1. Idiopathic (85 %)
   2. Congenital (10%)
   3. Neuromuscular (5%)

III. Anatomy
Lateral S-shape curve of the spine in its coronal plane; usually two curves are involved with transitional vertebrae at the transition of the curves. The upper limit of normal thoracic kyphosis is 50° and the lower limit is 20°.

IV. Symptoms
A. Many times the condition is asymptomatic but can present with just a vague, dull ache of the back.
B. The patient has a history of scoliosis in the family.

V. Signs
A. Shoulder-level asymmetry as seen from behind with patient standing.
B. Asymmetry of the hemithoraces as seen by inspection of the spine from the rear with forward flexion (Adam's test). At times, the scoliotic curve can be seen when using the level of the iliac crest and the ears as reference points. Leg length discrepancies and pelvic tilt can be identified.
C. Note area of the curve (e.g., left lumbar, right thoracic), magnitude of the curve, amount of deviation from C7 plumb line, degree of shoulder elevation (measured by scoliometer).

VI. Laboratory/Radiology
Usually no laboratory markers or serum markers are present. Radiographs are performed differently

from routine spine radiographs. Long-cassette radiographs should be performed in the upright position. The PA technique minimizes breast radiation dose. Primary and secondary curves can be identified on radiographs. Cobb's angle describes the region of the primary curve side of the convexity (greatest curve angle and not the concavity) that is formed by the intersection of the two lines drawn from the perpendicular of the endplate of the vertebrae in question (e.g., 65° thoracic curve convex to right with the apex at T-7).[29] Also, the presence or absence of pelvic tilt should be noted. Radiographs should of course be examined for other coexistent pathologic spinal processes.

VII. Special Studies
Occasionally bone scan may be helpful to rule out an occult stress fracture or other comorbid conditions in a patient with acute back pain and scoliosis.

VIII. Diagnosis
Scoliosis: idiopathic

IX. Differential Diagnosis
Spondylolysis, secondary scoliosis (spinal cord tumor, syringomyelia, Friedreich's ataxia, leg length discrepancy)

X. Treatment
Treatment depends on degree of curvature.

A. Mild scoliosis (<20°): No treatment is required as long as curve is nonprogressive; reassess every 3 to 4 months until skeletal maturity is achieved.
B. Moderate (20 to 45°): Bracing (e.g., Milwaukee or Boston) should be worn 18 to 23 hours per day for progressive curves in skeletally immature athletes.[30] Bracing for 8 or 16 hours per day is significantly less effective than bracing for 23 hours per day.[31] Individualized regimens may permit daily sport activity out of the brace. A comprehensive program to strengthen the abdominal musculature as well as maintain an ideal body weight is prescribed. In general, almost all adolescents with scoliosis should be allowed relatively unrestricted physical activity, including competitive athletics. While swimming and walking are preferred activities, no evidence has been reported that collision or contact sports can increase progression or complications; however, patients who have had spinal fusion procedures or who have abnormalities of the cervical or cervicothoracic spine should be advised against contact, collision, or strenuous noncontact activities.
C. Severe (>45°): Possible surgical fusion with instrumentation.

Consultation: progressive curvatures >20°

XI. Complications
Progression of curve, respiratory compromise with early deaths from respiratory failure with curves greater than 60° if left untreated; lumbar curves greater than 50° have high risk for degenerative disc disease and pain.

XII. Prevention
Early diagnosis and regular follow-up.

RHIZOPATHY (CERVICAL BRACHIALGIA, BRACHIAL PLEXOPATHY)

I. Epidemiology
Seen in motor sports and aircraft enthusiasts.

II. Mechanism of Injury
Hyperextension microtrauma from rapid rotation side bending, and vibratory stress.

III. Anatomy
Osteophytic impingement of the peripheral nerves as they exit the neuroforamina in the cervical spine.

IV. Symptoms
A. Decreased sensation to area of extremity corresponding to cervical peripheral nerve impingement
B. Weakness
C. Common combination of decreased grip strength with paresthesia and dysesthesias in the palm of the hand

V. Signs
A. Decreased deep tendon reflexes, flaccid paralysis/paresis without signs of long tract diseases or cerebellar diseases
B. Muscle atrophy (e.g., decrease in the circumference of the distal limb)

C. Positive Spurling test, where symptoms are reproduced by placing the cervical spine in a side bent position and then applying axial pressure

VI. Laboratory/Radiology
Osteophytes in the neuroforamina are best observed in oblique projections.

VII. Special Studies
Magnetic resonance imaging is the imaging study of choice for cervical radiculopathy, even though it tends to over-estimate foraminal stenosis. The next best non-invasive study is CT with IV-contrast. Cervical CT–myelogram is an excellent choice in this setting but is invasive, thus is reserved for problem cases. When the symptoms have been long-lasting, an EMG and NCV can confirm clinical suspicions.

VIII. Diagnosis
Cervical brachialgia (rhizopathy).

IX. Differential Diagnosis
Double crush syndrome, paracervical muscle spasm, and herniated cervical disc.

X. Treatment
A. Initially:
   1. Mild cervical traction
   2. NSAIDs and analgesics p.r.n.
   3. Physical modalities
B. Long-term: Range of motion exercises, gentle axial traction to distract the vertebrae, as well as paracervical muscle-strengthening exercise; a systematic review of the use of epidural steroids showed no significant difference at 6 weeks or 6 months in pain relief scores when compared to placebo.[32]

Consultation: surgery for progressive disease and pain

XI. Complications
Progressive distal weakness and intractable pain.

XII. Prevention
Not applicable.

TORTICOLLIS (WRY NECK)

I. Epidemiology
Torticollis is prevalent in all age groups, although the actual rate is unknown. It is seen in sports with sudden neck rotation (e.g., racket sports) or in activities requiring prolonged positioning of the neck (e.g., marksmanship sports).

II. Mechanism of Injury
A. Usually post-traumatic muscle spasm after a sudden unaccustomed rotation or side bending
B. Physical noxious stimulus, such as exposure to cold
C. Anterior cervical adenopathy, possibly due to viral illness

III. Anatomy
Spasm of one side of the neck muscles producing a characteristic side bending and rotation of the cervical spine.

IV. Symptoms
Insidious onset of stiffness and spasm, although many patients awaken from a night's sleep in the offending position.

V. Signs
A. An obvious prominence of the sternocleidomastoid and trapezius muscles on the affected side, while the cervical spine is side bent to the ipsilateral side but rotated away from that side. Sometimes with continued spasm of the sternocleidomastoid, the face is drawn into an upward gaze.
B. No motor or sensory deficits are observed.
C. Deep tendon reflexes are normal.

A search for adenopathy should be made, as well as supraclavicular fossa and axillary examinations to look for masses.

VI. Laboratory/Radiology
No serologic markers are characteristic of torticollis, but support for other concomitant diseases can be made through serum testing. Radiographs show lateral tilt and rotation of the cervical spine, indistinguishable actually from normal positional neck rotation/tilt. History is thus critical.

VII. Special Studies
In the setting of significant trauma to the neck and a torticollis-like presentation, CT of the upper C-spine may be appropriate to exclude C1–C2 fixed rotatory dislocation.

VIII. Diagnosis
Torticollis.

IX. Differential Diagnosis
Underlying infections, tumors, or trauma.

X. Treatment
Generally this is a self-limited problem.

A. Initially: NSAIDs and analgesics p.r.n. plus local heat and a soft collar
B. Long-term: Reciprocal innervation and proprioceptive facilitative techniques to relax the musculature; physical modalities (e.g., hydrocollator packs, neuromuscular stimulation using galvanic stimulation)

XI. Complications
Recurrences.

XII. Prevention
Not applicable.

STINGER/BURNER (CERVICAL ROOT COMPRESSION SYNDROME)
See Chapter 36.

CERVICAL DISC SYNDROME (HNP/SLIPPED DISC/RUPTURED OR HERNIATED DISC)

I. Epidemiology
Increased incidence of cervical disc syndrome occurs with lifting, driving, and smoking; 51% of adults have arm and/or neck pain at some point in their lives.

II. Mechanism of Injury
A. Repetitive overload
B. Trauma from axial loading and hyperflexion (e.g., wrestlers, divers, linebackers)

III. Anatomy
Most common at C5–C6, which is the level of the most motion as it lies anterior to the plumb line of the neck and forward bending is accompanied by increased force of extensors at that level. The next most common sites of herniation are C6–C7, C4–C5, C3–C4, and C7–T1.

IV. Symptoms
History of previous trauma, complaints of pain along dermatomal distribution patterns, and sharp neck pain worsened by neck movement and Valsalva maneuvers.

V. Signs[22]
Tender myofascial structures of the neck, with spasm of the paravertebral musculature and decreased range of motion. Neurovascular exam should check for subtle motor, sensory, and reflex changes (see Table 44.1). Severe cases may have positive Babinski reflex and loss of bladder, bowel, and sexual function.

VI. Laboratory/Radiology
Plain films are often normal.

VII. Special Studies
Magnetic resonance imaging is the initial test of choice. MRI effectively diagnoses spinal stenosis and lateral stenosis. MRI also screens well for unexpected traumatic disc herniations, cord injuries, fractures, or epidural hematomas. CT (routine or CT-myelogram) is also effective and is exceptional at defining bone abnormalities and osseous lateral stenosis. Electrodiagnostic studies are helpful with multilevel findings on imaging studies.

VIII. Diagnosis
Cervical disc herniation

IX. Differential Diagnosis
Cervical fracture, shoulder pathology, peripheral nerve entrapment, vascular disorders (impingement), and underlying medical problems (arthritis, tumor).

X. Treatment
A. Initially: Immobilization with cervical collar, NSAIDs, and analgesics on a fixed schedule.
B. Long-term: Myofascial work for soft tissue shortening, tightness; segmental mobilization for stiff spinal segment; manual traction for radicular or facet symptoms; stabilization through strengthening, posture, and body mechanics.

Consultation: recalcitrant progressive radicular and myelopathic signs (consider anterior cervical fusion); no reliable evidence of surgery for cervical

spondylotic radiculopathy or myelopathy has been reported (Level of Evidence A, randomized controlled trials).[33]

XI. Complications
Sensory and motor deficits.

XII. Prevention
Not applicable.

UPPER CERVICAL INSTABILITY

I. Epidemiology
Contact/collision sport athletes, while off guard or unprotected, may suffer injury from a blow to the forehead (forced hyperextension) or occiput (hyperflexion); "special athletes" such as Down syndrome/trisomy 21 and rheumatoid arthritis patients are at risk for atlantodens subluxation/dislocation.

II. Mechanism of Injury
Trauma from compressive axial loading during hyperflexion or extension.

III. Anatomy
Disruption of the ligaments supporting the vertebral bodies (e.g., anterior and posterior longitudinal interspinous ligaments) (for grades, see Chapter 34). Connective tissue laxity and congenital bony anomalies of C1 and C2 in special athletes are additional risk factors. Associated cervical fractures may or may not be present.

IV. Symptoms
See Cervical Spine Fractures section, below.

V. Signs
See Cervical Spine Fractures section, below.

VI. Laboratory/Radiology
All special athletes with Down syndrome, connective tissue disorders, or other conditions predisposing to atlantoaxial instability (Morquios syndrome, or glycogen storage diseases) should have baseline radiographs. Passive flexion–extension may reveal instability not appreciable on static radiographs. CT scans have emerged as the best procedure to detail bony anomalies of the upper spine. Radiographs may reveal abnormalities, including a hypoplastic or absent dens or a widened atlantoaxial interval. An atlantoaxial interval of greater than 5 mm in children (3mm in adults) suggests laxity. Evidence of instability disqualifies athletes from any competition involving contact, collision, or axial neck loading. Serologic markers for rheumatoid diseases and other connective tissue problems can be sought but often are absent, especially in the ANA-negative connective tissue diseases and soft-tissue rheumatism. It must be noted that recent studies have concluded that lateral plain radiographs are of potential but unproven value in detecting patients at risk for developing spinal cord injury during sports participation. Current information suggests that the presence of neurologic symptoms and signs of cord compression may be more predictive of potential progression of injury than are abnormalities of radiographs in the asymptomatic patient (Level of Evidence B, systematic review).[34]

VII. Special Studies
None indicated.

VIII. Diagnosis
Atlantoaxial instability (dislocation/subluxation)

IX. Differential Diagnosis
Fracture (Jefferson, hangman), sprain, strain.

X. Treatment
A. Initially: ABCDE and stabilization (see Chapter 35); immediate transport to hospital for further management. Although still somewhat controversial, high-dose methylprednisolone steroid therapy is the only pharmacological therapy shown to have modest efficacy when it can be administered within 8 hours of injury (Level of Evidence A, randomized controlled trials).[35]
B. Long-term:
   1. Strengthening programs for the cervical musculature if atlantoaxial interval is <1 cm.
   2. Surgical procedure designed to enhance the stability, especially if the interval is ≥1 cm.
   3. Protective bracing.

XI. Complications
Frank dislocation, cervical cord severance.

XII. Prevention
Maintain high index of suspicion for special athletes.

SUPRASPINOUS LIGAMENT TEAR

I. Epidemiology
Collision and contact sports, particularly in athletes with long, thin necks.

II. Mechanism of Injury
Falling with rapid extension of the neck or sudden forceful upward jerk (e.g., clay shovelers).

III. Anatomy
The supraspinous ligament acts as an extension apparatus, scalloping from one spinous process to another (C1–L5). Tears of the ligament are classified according to Chapter 34. The most common site of sprain is C7–T1.

IV. Symptoms
Sudden onset of local pain in the midline of the posterior neck.

V. Signs
A. Palpable defect along the posterior neck, especially directly above or below the vertebra prominens at C7, T1.
B. Range of motion is decreased, with extension < flexion.

VI. Laboratory/Radiology
Radiograph findings are normal, especially the lateral view of the cervical spine showing even spacing between the spinous processes. Radiographs should be adequate to visualize spinous processes of lower cervical vertebrae to rule out clay-shovelers fracture. MRI may be helpful in identifying ligamentous disruption.[36]

VII. Special Studies
None indicated

VIII. Diagnosis
Supraspinous ligament sprain grade _____.

IX. Differential Diagnosis
Facet syndrome, paracervical muscle spasm, fracture of the spinous process, fibromyositis, arthritis.

X. Treatment
A. Initially: Rest, ice, immobilization with a soft collar, NSAIDs, and analgesics p.r.n.
B. Long-term: PRE using small incremental loads

Consultation: not indicated

XI. Complications
Progressive instability.

XII. Prevention
A. Specific strengthening and flexibility exercises
B. Cervical collar/towel roll in collision or contact sports for athletes with thin, long necks.

CERVICAL FACET SYNDROME
See Facet Syndrome section, above.

CERVICAL SPINE FRACTURES

I. Epidemiology
With rule changes in the mid 1970s essentially outlawing the spear tackle, the incidence of cervical spine injuries has decreased in the football-playing athlete.[1] Nonetheless, American football, swimming, and motor vehicle accidents account for almost all of the scenarios where cervical spine fractures are found.[2,3] Trampoline, gymnastics, cheerleading, wrestling, rugby, and hockey accidents account for the remaining sports-related fractures. The National Football Head and Neck Injury Registry found an incidence of 0.92 cervical spine injuries per 100,000 players from 1971 through 1985.[4–7] Serious disability from cervical spine fractures has been a current focus of both the sports medicine and medical jurisprudence communities. Any athlete who sustains an injury sufficient to cause any degree of mental status change, ranging from unresponsiveness to a dazed state that quickly returns to lucidness, has probably also placed the cervical spine at risk for fracture.

II. Mechanism of Injury
Axial loading of C-spine (e.g., spear, head, or butt blocking) in flexion (more common) or extension (Figure 44.3).

III. Anatomy
The common fracture patterns found include Hangman's fracture, Jefferson's fracture, and burst fracture. Fractures of the posterior joints tend to be more unstable.

IV. Symptoms
Range from none through vague aches to progressive loss of motor functions (e.g., weakness, paralysis) and sensory functions (e.g., paresthesias, numbness) to complete paralysis.

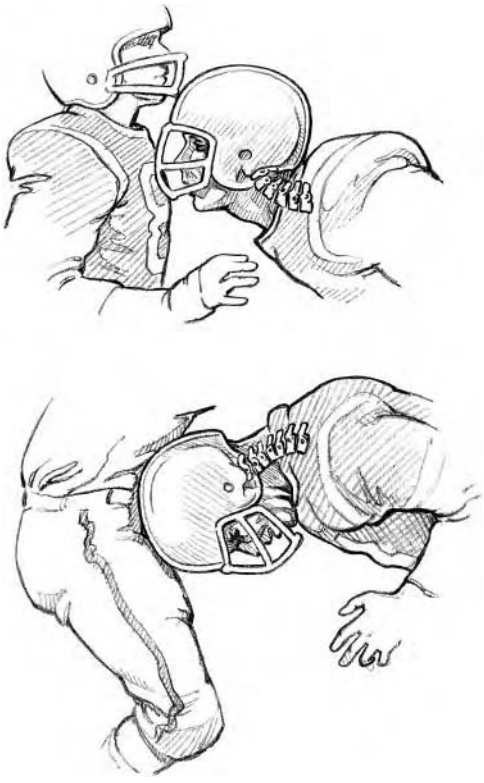


Figure 44.3 Illegal face blocking leads to hyperextension injury. Illegal head or butt blocking (spearing) causes hyperflexion damage.

V. Signs

A. Range from none through decreased ROM, posturing, paraspinal spasm, and marked motor deficits (e.g., paresis, paralysis) and sensory deficits (e.g., numbness)
B. Posterior point tenderness with occasional step-off deformity of spinous process
C. Reflex changes (see Table 44.2)

VI. Laboratory/Radiology

Most trauma centers recommend a routine trauma C-spine series to include lateral, open-mouth, and AP views. A swimmer's view is added if the lower C-spine or T1 is not diagnostically visualized. CT is the initial modality of choice when radiographs are negative and in the presence of significant clinical risk or suspicion of fracture. Negative radiographs and CT do not rule out ligamentous injury. Flexion–extension views are indicated to screen for ligamentous injury when pain persists despite absence of fractures on images. Flexion–extension views must be done *actively*; that is, the patient slowly and gently flexes and extends neck without any manipulation by professional staff. MRI is indicated in incompetent patients with suspected ligamentous injury who cannot safely undergo flexion–extension radiographs and is also the procedure of choice to evaluate cord injury.

VII. Special Studies

None indicated.

VIII. Diagnosis

Cervical fracture level _____. Stable/nonstable.

IX. Differential Diagnosis

Sprain, strain, facet syndrome.

X. Treatment

A. Initially: ABCDE and stabilization (Chapter 35); immediate transport to hospital for further management.
B. Long-term: Cervical tongs with traction or open reduction and internal fixation with or without external fixators involving neck bracing and skull screws; comprehensive rehabilitation program including ROM and progressive resistance exercises. Return to the injury-producing sport is a tenuous and often anxiety-fraught decision that should be made in concert with the athlete, parents, coach, trainer, and even the legal counsel of the school.[5]

XI. Complications

Spinal cord injury (paraplegia, quadriplegia, death).

XII. Prevention

A. Do strengthening and flexibility exercises.
B. Avoid situations that load the axial cervical spine.
C. Properly use cervical pads or an A-frame type pad that aids in fixing the neck to the upper chest.

SOFT-TISSUE INJURY OF THE NECK

I. Epidemiology

An infrequent but serious problem that occurs in sports with missiles (baseball, football), sticks (hockey, lacrosse), and exposed necks. Ice

TABLE 44.2
Radicular versus Referred Pain

Tests	Referred Pain	Radicular Pain
Symptoms	Deep, boring nonspecific, poorly localized	Sharp, localized, electric-like
Radiation	To posterolateral thigh	Follows sciatic nerve distribution
Sensory	Rare changes	Follows dermatomes
Motor	Weakness; atrophy is rare	Frequent objective finding of weakness
Reflex	Rarely diminished	Frequent deficit
Tension	SLR negative	SLR positive
Provocative	None	Relief with injection of anesthetic; isolates level

hockey accounts for 75% of injuries to the anterior neck in intercollegiate sports.[37–39]

II. Mechanism of Injury

Direct trauma to exposed anterior or lateral neck by sport missiles (pucks, sticks, balls, boxing glove, or even human appendages (e.g., clothesline-type tackles); indirect trauma (e.g., stretching, tearing, compression of the carotid artery following neck rotation from a blow).

III. Anatomy

Soft-tissue areas of the neck are protected by the cervical spine posteriorly but are relatively exposed anteriorly and laterally. The chin affords some degree of protection anteriorly, but an extended neck increases the exposure dramatically. Injuries include crush fracture of the thyroid, cricoid, and tracheal cartilage; edema and hemorrhage surrounding the glottic structures; laryngospasm; disarticulation of the vocal cords; and carotid artery thrombosis, rupture, dissection, or aneurysm. The areas most susceptible to injury are the glottis, subglottis, and upper cervical trachea.

IV. Symptoms

A. Hoarseness, difficulty breathing
B. Coughing, pain with swelling, nausea, and dizziness

V. Signs

A. Aphonia, hoarseness
B. Stridorous respirations, respiratory distress
C. Coughing, hemoptysis
D. Subcutaneous emphysema
E. Palpable crepitus
F. Pain at impact site, loss of normal contour secondary to edema and hemorrhage

VI. Laboratory/Neck and Chest Radiographs

Radiographs are useful to detect foreign bodies and confirm air in soft tissues with airway injury; however, soft tissue air is a non-specific finding with many possible causes. Gross airway distortion may be evident on radiographs.

VII. Special Studies

Indirect and direct laryngoscopy are the primary tools to diagnose airway injuries. Imaging plays a secondary role in airway injury but a primary role in vascular injury. CT is the initial imaging modality of choice with significant acute injury to neck soft tissues. Fractures of the hyoid bone, tracheal or cricoid cartilage, or other airway segments are sometimes visible, especially in older patients with greater mineralization of the airway structures. CT–angiography (CTA) using rapid-contrast injection and thin-slice techniques is extremely valuable for diagnosis of neck vascular injuries. CTA (or MRI angiography) are fast replacing angiography at institutions with adequate facilities to do these studies. Conventional angiography may be necessary when bullets or other metallic foreign bodies would cause artifacts on CT or MR imaging. MRI may allow diagnosis of airway injuries when CT is inconclusive, especially in poorly mineralized airways.

VIII. Diagnosis

Specific soft-tissue injury, location _____.

IX. Differential Diagnosis

Contusion, sprain, strain, carotid sinus barotrauma, carotid artery injury (dissection, occlusion, stenosis, rupture, or aneurysm), fracture (thyroid, cricoid, hyoid, epiglottis, tracheal ring, C-spine), laryngeal injury (tear, fracture, spasm, edema).

X. Treatment

A. Initially: ABCDE and stabilization (Chapter 35), with immediate transport to a hospital for further management.
B. Long-term: Consultation with ear, nose, and throat and vascular specialties.

XI. Complications

Late obstruction or compromised airway from expanding hematoma; laryngeal injury resulting in delayed or chronic hoarseness; neurologic deficits

XII. Prevention

A. Protective gear for the neck
B. Strict adherence to rules preventing dangerous situations (such as high sticking in ice hockey or "clothes lining" in tackle football)

SPINAL CORD CONCUSSION

I. Epidemiology

Transient disturbance of the spinal cord as the result of trauma that manifests itself by demonstrable neurologic deficits of the limbs with complete resolution within 24 to 48 hours. Its incidence in football is 1 to 2 per 10,000 athletes.[40]

II. Mechanism of Injury

High-velocity trauma injuries to the vertebral column in association with hyperflexion, hyperextension, or axial loading; violent falls onto the back such as those sustained in football tackling. The magnitude and direction of the force ultimately determine the subsequent pattern of neurologic deficit. Oftentimes, a pre-existing vertebral abnormality results in narrowing of the canal (i.e., congenital cervical stenosis) or increased hypermobility, reducing the effective size of the protective cerebrospinal fluid cushion and allowing more direct transmission of the kinetic energy to the spinal cord.

III. Anatomy

The cervical cord is most commonly affected because of its greater mobility, but concussion to the spinal cord can occur at any level (typically, the site of injury). Generally, no demonstrable gross or microscopic pathologic changes to the spinal cord or nerve roots occur.

IV. Symptoms

A. Decreased sensation, paresthesia such as burning or tingling, and/or inability to move arms or legs
B. Loss of consciousness (atypical)

V. Signs

Variable, depending on location and extent of trauma: dysesthesias, anesthesia (pain and light touch), weakness, paresis, paraplegia or quadriplegia, hyperreflexia.

VI. Laboratory/Radiology

Radiographs (and/or CT) can be performed quickly and are useful, but MRI is the definitive study to rule out cord compression, hemorrhage, and edema.

VII. Special Studies

None indicated.

VIII. Diagnosis

Spinal cord concussion (spinal cord neuropraxia).

IX. Differential Diagnosis

Fracture, subluxation/dislocation, compression; brachial plexus injury (stinger); lumbosacral spine injury; Brown–Sequard hemicord syndrome.

X. Treatment

A. Initially: ABCDE plus stabilization; immediate transport to hospital.
B. Long-term: usually unnecessary since concussion of the spinal cord will resolve within 24–48 h; failure to do so requires further workup and management.

Consultation: neurosurgery for atypical cases

XI. Complications

Cord compression, persistent weakness.

XII. Prevention

A. Maintain rules of sport to avoid spearing tackles.
B. Do strengthening/flexibility exercises.
C. Athletes with congenital spinal anomalies (e.g., stenosis) should be advised to discontinue participation in collision/contact sports.
D. Use seat belts in motor sports.

MISCELLANEOUS CAUSES OF BACK PAIN

The stuck rib syndrome can be found in athletes who have been sedentary or who are acutely injured from a deceleration fall. Oftentimes, the mechanism of injury is simply intercostal muscle spasm, or occasionally the disorder is secondary to splinting from visceral diseases such as pneumonia or pleuritis. Typically, the patient complains of limited side bending motion secondary to pain or inability to fully inspire without pain. Excursion on the affected side is decreased as the dorsal trunk is palpated. Mobilization of the rib can be achieved by hooking the examiner's fingers around the edge of the rib and moving the rib in a bucket handle motion in concert with the adjacent ribs. Differential diagnosis includes facet syndromes or paraspinal muscle spasms.

Sacroiliac (SI) sprain syndrome is a commonly overlooked source of unilateral low back pain that may produce sciatic nerve irritation due to proximity of the nerve to the lower SI joint.[41] Many of the SI joint tests are positive, although the neurovascular exam is usually normal. Anti-inflammatories, rehabilitative exercises that include manipulation, and stereoscopic corticosteroid injections of the SI joint are important treatment modalities. Sacral torsion is a pathologic posture of the sacrum about an axis around which the sacrum revolves. It is seen more commonly in females than in males (4:1) in the second and third decades and responds well to physical manipulation techniques. The differential diagnosis of sacroiliac pain should include Reiter's syndrome, anklyosing spondylitis, and sacroileitis.

Scheuermann's disease is an epiphysitis of the vertebral spine endplates that causes a rounded appearance of the shoulders and thoracic spine.[20,21,42] It is seen more commonly in young males, typically at ages of greatest growth spurt (7 to 9 and 13 to 17 years). The usual symptoms include a dull, generalized aching of the dorsal spine with provocation of pain during sitting and after activities, with amelioration of symptoms during activities. Exam reveals hyperkyphosis in the region of T7 with normal motion and strength. Unlike postural roundback, the kyphosis cannot be voluntarily corrected. Radiographs show wedging of at least three successive vertebral bodies and disc space narrowing, endplate irregularities, and multiple Schmorl's nodules (herniations of the disc into the vertebral end plates). These radiographic findings are essentially diagnostic of the entity when noted in young individuals. Treatment is aimed at the epiphysis, with training exercises to increase flexibility and strength of agonist and antagonist muscle groups. A Milwaukee brace has been found to be beneficial for those with kyphosis with or without pain.

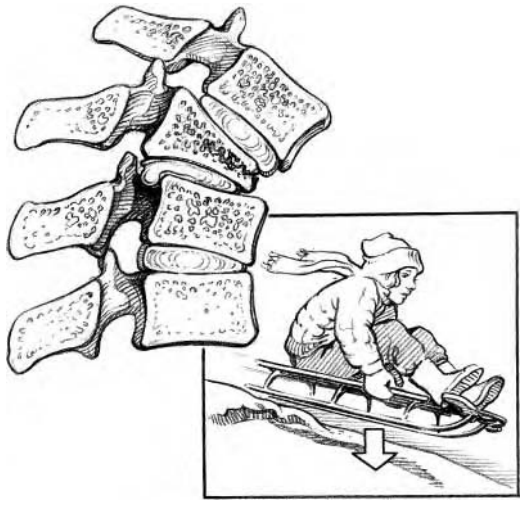


Figure 44.4 The hyperflexed spine is vulnerable to compression injury during sledding or tobogganing.

The preflexed spine (e.g., sitting on a sled or toboggan) can lead to compression fracture of the lower thoracic and upper lumbar vertebral bodies, particularly if a bump is hit (Figure 44.4).[42,43] The athlete may complain of vague pain or more serious motor/sensory deficits. Prompt, thorough evaluation followed by bed rest and traction are essential to reduce associated morbidity.

SUMMARY

With increasing levels of recreational activity and sports participation, the spine has joined the elbow, ankle, knee, shoulders, and other anatomic areas as a site for athletic-related trauma. Such injuries, while infrequent, are often severe, resulting in permanent disability or death. Spinal anatomy and biomechanics as well as a variety of pathologic conditions specific to the spine must be understood by the competent sports physician. High levels of fitness, young age, and lack of compensation considerations should not dissuade the wary physician from a thorough search for significant injury, underlying disease, training errors, and environmental factors. Finally, a comprehensive rehabilitative program emphasizing normal range of motion and strength together with a preventive strategy including protective

equipment are essential before return to full play should be prescribed.

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PART IV
SPORTS INJURIES: THE UPPER EXTREMITY

45 SHOULDER INJURIES

Brian C. Halpern and Osric S. King

INTRODUCTION

The diagnosis and management of shoulder injuries is often challenging to the examiner. Complaints are typically vague and nonspecific. In addition to isolated musculoskeletal pathology, the etiologies of shoulder symptoms can come from a variety of neurological, inflammatory, and cardiovascular conditions. Determining the cause and treatment relies equally on the history, mechanism of injury, and physical examination. Imaging studies that depend strongly on technique and quality can confirm the diagnosis and help guide management.

GENERAL EPIDEMIOLOGY

Most shoulder injuries involve the soft tissues (e.g., cartilage, muscle–tendon unit) and occur in

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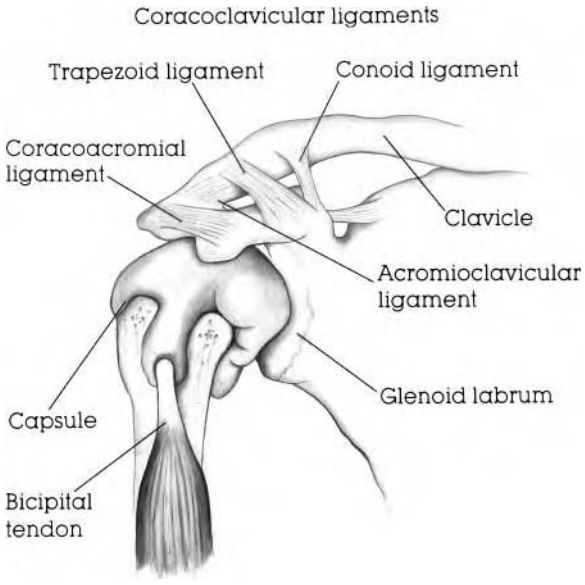


Figure 45.1 Ligamentous anatomy of the shoulder.

the sedentary as well as athletic individual. Many shoulder injuries result directly from repetition as in throwing and racquet sports. Similar damage can result from nonathletic, overhead activities such as painting or ladder climbing. In throwing and racquet sports, shoulder problems can account for more than 50% of injuries (Level of Evidence B, nonquantitative systematic review).[1] Anterior shoulder problems in these athletes are usually secondary to biomechanical and fatigue related instability. Unless direct trauma is the etiology, most athletic shoulder problems involve the dominant extremity. In sedentary individuals, symptoms in the nondominant or inactive shoulder are not uncommon. In an older population nonmusculoskeletal sources of symptoms can include cardiac, neurologic, primary neoplastic, and metastatic disease, as well as degenerative changes in the glenohumeral joint and rotator cuff. Age-related changes, poor conditioning, over-training, and trauma have common injury features. Studies have shown that rotator cuff and deltoid muscle weakness can cause superior migration of the humerus. This impingement of the humeral head against the subacromial arch is the hallmark event that contributes to bursitis and deterioration of the rotator cuff and biceps tendons. Investigations have repeatedly demonstrated subacromial impingement, whether seen in sports or related to poor conditioning, as one of the most common mechanisms of pain (Level of Evidence B, nonquantitative systematic review).[2]

ANATOMY

The shoulder girdle comprises the sternoclavicular joint, acromioclavicular joint, glenohumeral joint, subacromial space, and scapulothoracic space. Movement about all of these articulations allows for the complexity of the throwing motion. Motion exceeds 180° in three planes, with approximately two thirds of the full elevation occurring at the glenohumeral joint (Level of Evidence B, nonquantitative systematic review).[3] The sternoclavicular joint supports the anteromedial clavicle, and the articulation is between the proximal clavicle and superolateral portion of the manubrium. The posterior capsule of the joint is much stronger than the anterior capsule, thus allowing more anterior dislocations. The acromion is the anterior extension of the scapula. It has been described as having three shapes. Type 3 acromion is hooked and has been found to cause or aggravate rotator cuff impingement (Level of Evidence B, nonquantitative systematic review).[4] The acromioclavicular joint is between the lateral end of the clavicle and medial surface of the acromion and is associated with an intraarticular meniscus that may be incomplete. The acromioclavicular ligament provides superior support, but the major stabilizing structures are the coracoclavicular ligaments (the conoid and trapezoid) (Figure 45.1).

The glenohumeral joint is a synovial ball-and-socket joint in which one third of a spherical humeral head sits in the shallow glenoid process. To improve the containment of the humeral head,

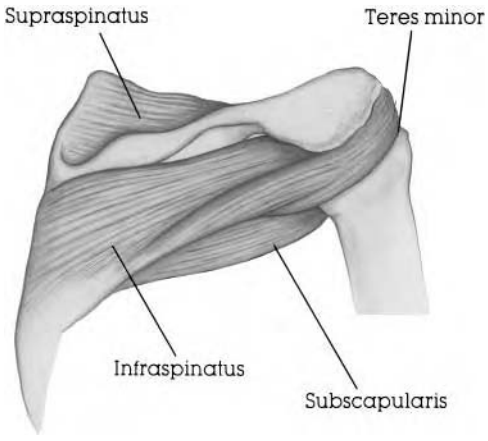


Figure 45.2 Rotator cuff muscles.

the glenoid labrum attaches peripherally around the margin of the glenoid. Thickening of the capsule forms the glenohumeral ligaments: superior, middle, and inferior. They are folds in capsule unlike the distinct ligaments found elsewhere in the musculoskeletal system. The inferior glenohumeral ligament has the most important role in stabilizing the shoulder joint in abduction and external rotation. The four major muscles of the shoulder girdle constitute the rotator cuff: the subscapularis anteriorly, the supraspinatus, the infraspinatus, and the teres minor posteriorly (Figure 45.2). The posterior rotator cuff muscles are major contributors to abduction and external rotation of the humerus.

The subacromial space (rotator cuff interval) lies between the subacromial arch and the rotator cuff. The supraspinatus and biceps tendon lie adjacent to each other in this area. This space allows free movement between the rotator cuff below and the coracoacromial ligament, acromion, and deltoid above, providing the wide functional mobility of the shoulder girdle. The critical area of clearance is between the coracoacromial arch and the greater tuberosity.[2] In addition to this anatomic factor, a suboptimal healing environment worsens the pathology present in this space. Vascular studies have demonstrated that the blood supply to the supraspinatus tendon, with the arm at the side in the abducted position, has an area of avascularity 1 cm in length. It extends proximally from the musculotendinous junction directly to the point of insertion of the tendon onto the greater tuberosity. Likewise, the intracapsular portion of the biceps tendon has a similar avascular zone as it passes over the head of the humerus (Figure 45.3).

HISTORY AND PHYSICAL EXAMINATION

A description of symptoms should include the quality of the pain, the location, where it radiates, and whether or not it is associated with any swelling or tenderness. Are the symptoms present or worsened with a particular motion? Is there any associated numbness and tingling, which could indicate a brachial plexus injury or an associated cervical spine problem? Can the patient

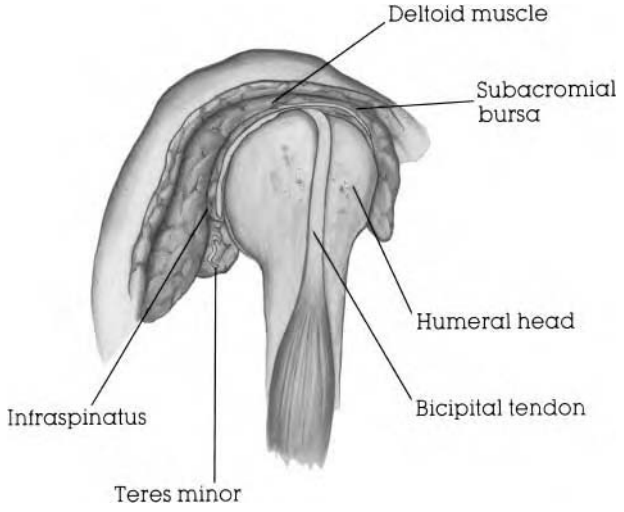


Figure 45.3 Lateral view of the shoulder; the bicipital (biceps) tendon runs through the bicipital groove in the head of the humerus.

recall a traumatic event that preceded the pain? A dull aching discomfort, often felt at night, corresponds with rotator cuff tears, whereas a stabbing, burning pain is more typical of a bursitis or a tendinosis. The location of the pain can be diagnostic. Pain over the acromioclavicular joint might suggest degenerative disease and, if associated with trauma, might indicate an acromioclavicular joint sprain. Pain deep in the shoulder may come from rotator cuff involvement, capsular inflammation, arthritic changes, or a glenoid labrum tear.[3]

A popping or catching noise inside the joint can indicate a glenoid labrum tear or biceps subluxation. A history of shoulder instability (described as giving way, slipping, or popping out) suggests subluxation or dislocation of the glenohumeral joint. Dislocation is more commonly caused by a contact injury, whereas either contact or non-contact injuries in addition to congenital ligamentous laxity, anteriorly or posteriorly, can cause subluxation. If the stability of the shoulder is affected by either trauma or fatigue, such intraarticular structures as the labrum can be torn by traction from the biceps tendon or impingement between the humeral head and the glenoid cavity. These types of injuries occur in baseball and tennis and often result from a combination of acceleration–deceleration forces and internal–external rotational velocities of the humerus; therefore, it is essential to question the patient regarding what phase of the motion the symptoms occur. Complete the review with a good past medical history inquiring about gout, chondrocalcinosis, arthritis, diabetes, neurovascular and metabolic disorders, and neoplastic disease.

The diagnosis is usually confirmed by the physical exam. Begin with an anterior inspection of both clavicles and acromioclavicular joints. The uninjured shoulder should always be examined as a "normal" comparison. Look for asymmetry, ecchymosis, swelling, and atrophy. Palpate the injured area last. Check the acromioclavicular joint for pain, crepitus, and motion. Test for the stability of the clavicle by pushing down on its distal third. Dislocation of this joint can occur in the anterior, posterior, or superior direction. Perform the cross-over test by placing the hand of the injured shoulder on the uninjured shoulder (Figure 45.4). If there is pain with this maneuver, an acromioclavicular joint injury probably exists. Next, inspect and palpate the posterior rotator cuff muscles. Also check the trapezius and latissimus dorsi muscles, which accentuate rotator cuff movement. A prominent scapular spine suggests

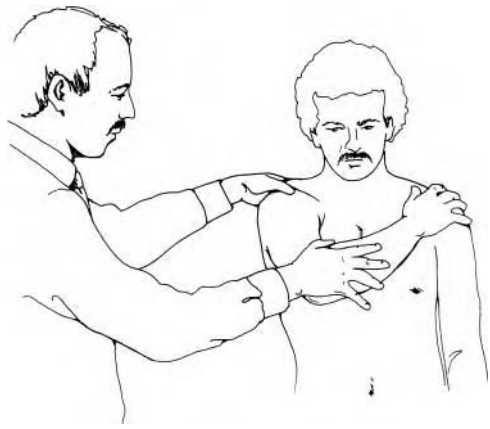


Figure 45.4 Cross-over test; testing for a sprain of the acromioclavicular joint.

posterior rotator cuff atrophy from a complete tear or suprascapular nerve injury. Have the patient push against a wall and note any winging of the scapula, signifying injury to the long thoracic nerve or serratus anterior muscle.

Assess the patient's range of motion, beginning with abduction, which occurs with glenohumeral and scapulothoracic movement in a 2:1 ratio. For every 3 degrees of shoulder abduction, 2 degrees occur at the glenohumeral joint and 1 at the scapulothoracic joint.[3] Check internal and external rotation in the sitting and supine positions with varying degrees of abduction. Rotator cuff tears, loose bodies, cervical radiculopathy, adhesive capsulitis, and osteoarthritis tend to decrease the glenohumeral motion segment and enhance the scapulothoracic segment during active shoulder elevation. The patient appears to shrug the injured shoulder into abduction. Testing active and passive range of motion is important because it helps to distinguish a rotator cuff tear from adhesive capsulitis. Remember that conditioned overhand throwers consistently demonstrate increased external rotation of the dominant extremity.

Injuries to the supraspinatus muscle are best assessed by instructing the patient to slowly abduct and smoothly lower the arm and by applying downward force to the forearm with the shoulder in 90° of abduction, 30° of forward flexion, and full internal rotation (drop arm test, supraspinatus stress test, empty can test) (Figure 45.5). Weakness, pain, or a limited range of motion can indicate injury to the supraspinatus muscle or suprascapular nerve.[3] To test for strength of the subscapularis muscle, have the patient internally rotate against resistance with

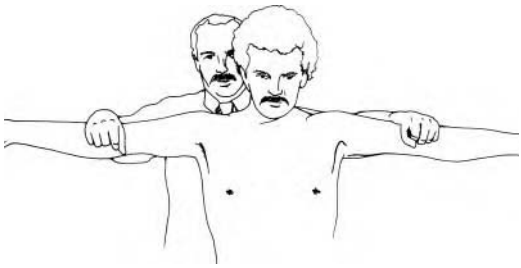


Figure 45.5 Supraspinatus test; testing for strength of the supraspinatus and deltoid muscles.

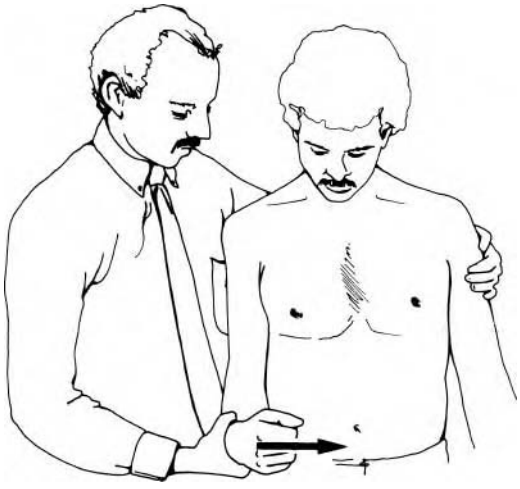


Figure 45.6 Testing for strength of the subscapularis muscle.

maximum adduction of the arm and elbow flexion to 90° (Figure 45.6). External rotation against resistance with maximum adduction of the arm and the elbow flexed 90° tests the infraspinatus and teres minor muscles (Figure 45.7).[3] Impingement maneuvers tend to drive the greater tuberosity under the coracoacromial arch. Examples include the Hawkins test (Figure 45.8), which elicits pain by internal rotation of the humerus in the forward flexed position; the cross-over test; and the Neer impingement test, which utilizes extreme forward shoulder flexion with forearm pronation. As impingement progresses, refractory tendinosis, wearing of the supraspinatus and biceps tendon, and partial or complete thickness rotator cuff tears can occur.[2] A 10-cc injection of 1% lidocaine beneath the anterior acromion may demonstrate pain relief with repeated impingement maneuvers. The test helps to rule out cervical radicular etiologies but the maneuver does

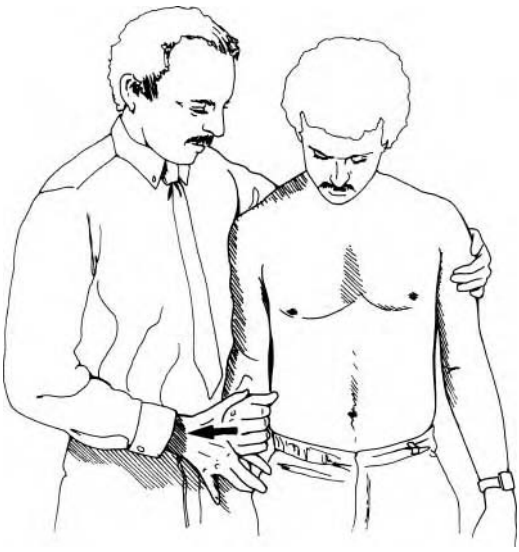


Figure 45.7 Testing for strength of the infraspinatus and teres minor muscles.

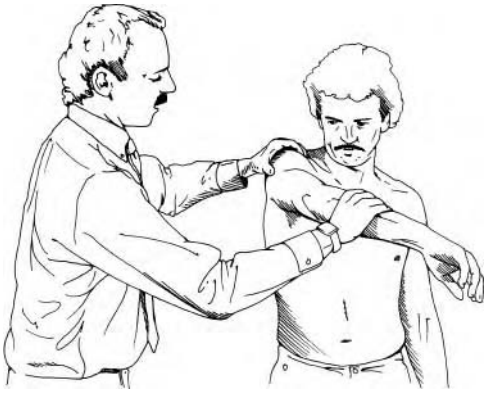


Figure 45.8 Impingement test; testing for impingement against the coracoacromial arch.

not distinguish among impingement, anterior humeral head subluxation, or other rotator cuff pathology as the source of pain.

Instability testing is done with the patient standing, sitting, or supine. Slowly abduct the patient's shoulder with the elbow flexed to 90°. Place your hand on the glenohumeral joint with the fingers palpating the humeral head posteriorly and the thumb anteriorly. With your other hand, support the patient's arm. Apply anterior-directed stress to the humeral head, levering it anteriorly (Figure 45.9). Repeat this maneuver at varying degrees of abduction, feeling for anterior subluxation. Place your thumb on the patient's humeral

Figure 45.9 Testing for anterior subluxation.

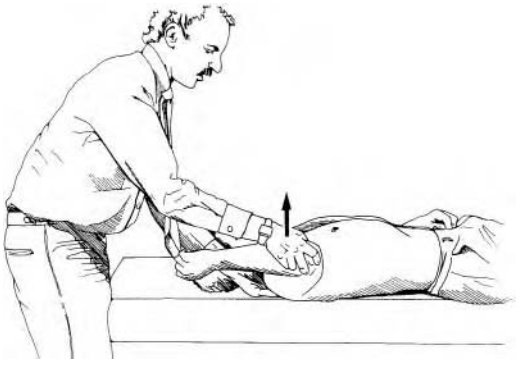


Figure 45.10 Testing for posterior subluxation.

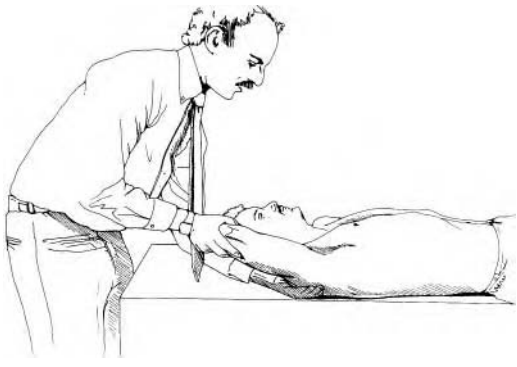


Figure 45.11 Clunk test; testing for an anterior labral tear.

head, flex the arm forward, and direct a posterior stress (Figure 45.10). Feel for any posterior subluxation with your posteriorly placed fingers. Posterior laxity (up to 50%) is often a normal finding in a throwing athlete's shoulder. Multidirectional instability presents an often-overlooked source of shoulder pain, so the examiner should test for inferior instability as well. Apply traction downward on the arm in 0° of elbow flexion and shoulder adduction. The appearance of a sulcus between the humeral head and lateral acromion is noted as a positive "sulcus sign" and is significant for inferior instability.[3] Apprehension or pain (positive apprehension, instability, or quadrant test) suggests prior subluxation or dislocation, although the tests may be positive with a tear of the rotator cuff or glenoid labrum.

A lesion of the glenoid labrum can be diagnosed by the clunk test or labral grind test. Place your hand posterior to the humeral head while the other hand rotates the humerus. Bring the arm into full overhead abduction with 90° of elbow flexion while providing an anterior force to the humeral head. The clunk test is positive when a clunk, pop, or grind is felt in the shoulder as the humerus comes into contact with the labral tear (Figure 45.11). The presence of a labral tear can also be tested with the active compression (O'Brien's) test. The maneuver consists of having the patient's arm forward flexed to 90°, adducted to 15°, and maximally internally rotated. The patient is instructed to resist as the examiner applies a uniform downward force. The patient then maximally supinates the arm and the maneuver is repeated. The test is positive if pain is felt deep within the shoulder and improved when the forearm is in the maximally supinated position. (Figure 45.12). The long head of the biceps can be palpated in the bicipital groove while the patient is in the supine position. With the examiner's finger still in the bicipital groove, the subscapularis tendon can be palpated for tenderness by externally rotating the humerus. The supraspinatus tendon can be palpated for tenderness by internally rotating the humerus. The integrity of the biceps tendon can be further assessed with the following maneuvers:

- Speed's, straight-arm flexion, or bowling test — Resisted forward flexion of the humerus with the forearm supinated and the elbow extended produces bicipital groove pain.
- Yergason's or supination test — Supination and external rotation of the forearm

against resistance with the elbow flexed at 90° produces bicipital groove pain.
- Ludington's or subluxation test — Apprehension and pain follow passive shoulder abduction with bicipital groove pressure, alternating resisted rotation, and biceps contraction.
- Gilcrest's test — Bicipital groove pain and possible subluxation follow external rotation of the shoulder and forearm supination with a light weight at 90° of shoulder abduction.

All examinations must include a neurovascular assessment of the upper extremities. Brachial, radial, and ulnar pulses should be checked. The neurologic exam should include motor and sensory exams, deep tendon reflexes (biceps, C5; brachioradialis, C6; and triceps, C7), and a Spurling test. The latter is performed by lateral rotation of the cervical spine toward the painful shoulder and then applying axial compression.

DIAGNOSTIC PROCEDURES

X-rays of the shoulder should include an anteroposterior (AP) view in internal and external rotation (true AP view). An axillary or west point axillary view is included in cases of less severe shoulder trauma, as these projections more accurately evaluate the relationship of the glenoid and humeral head and bony anatomy. For more severe trauma, a true lateral or Y view is important (Level of Evidence B, nonquantitative systematic review).[5] Ultrasonography and magnetic resonance imaging (MRI) are highly accurate for rotator cuff tears. Computed tomography (CT) demonstrates subtle fractures of the glenoid rim and reactive bone changes about a subluxing or recurrently dislocating shoulder. CT arthrotomography visualizes the internal shoulder structures and is particularly useful in delineating labral lesions. An MRI can confirm rotator cuff and labral tears as well as provide evidence of shoulder subluxation and dislocation.

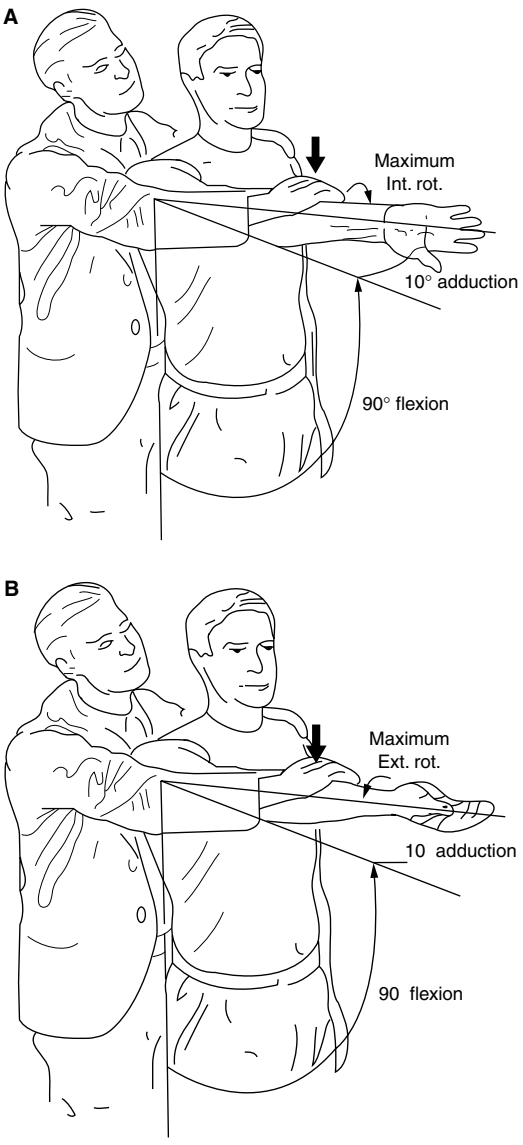


Figure 45.12 Active compression test.

STERNOCLAVICULAR SPRAIN/SUBLUXATION/ DISLOCATION

I. Epidemiology

The sternoclavicular joint is the only articulation between the upper extremity and axial skeleton yet has the least amount of bony stability of any joint in the body. Sternoclavicular injuries are rare and associated with trauma to the chest or shoulder. They are classified as anterior or posterior, determined by the anatomic position of the medial head of the clavicle in relation to the sternum (Level of Evidence C, nonquantitative systematic review).[6] Anterior dislocation is more common than posterior, and many dislocations are actually fractures through the physeal plate because the epiphysis at the medial end of the clavicle closes at approximately 23 to 25 years of age.

II. Mechanism

The mechanism of injury is an indirect force applied from the anterolateral or posterolateral aspect of the opposite or uninjured shoulder. This

usually occurs when the athlete falls on the injured shoulder with additional forces applied through the opposite shoulder. Injury can also occur with a direct force to the anteromedial aspect of the clavicle, pushing the clavicle posteriorly behind the sternum into the mediastinum. Disruption of the pulmonary and vascular systems can occur.

III. Anatomy

Tearing of the capsule, intraarticular disc, and costoclavicular ligaments:

- Grade I — All ligaments are intact and joint is stable.
- Grade II — Partial disruption of the sternoclavicular and costoclavicular ligaments leads to subluxation of the sternoclavicular joint.
- Grade III — Complete disruption of the sternoclavicular joints leads to anterior or posterior dislocation.

IV. and V. Symptoms and Signs

- Grade I — slight swelling and tenderness at the joint; no instability; mild to moderate pain, especially with arm movement
- Grade II (subluxation) — increased pain and swelling; palpable anterior or posterior subluxation
- Grade III (dislocation) — severe pain with range of motion (ROM)

In Grade III, the patient supports the injured arm across the trunk with the normal arm. In the supine position, pain increases and the involved shoulder does not lie flat on the table.

Note: In anterior dislocation, the medial end of the clavicle is visibly prominent anterior to the sternum. In a posterior dislocation, the medial end of the clavicle is palpable and displaced posteriorly. Venus congestion may be present in the neck or upper extremity. Breathing difficulties, shortness of breath, swallowing difficulties, subcutaneous emphysema, and ipsilateral pulselessness, swelling, and discoloration can occur.

VI. Diagnostic Imaging

Anteroposterior (AP) or posteroanterior (PA) x-ray views of the chest or sternoclavicular joint may suggest an abnormality as one clavicle appears displaced compared to the normal side. A 40° cephalic tilt view (Rockwood view) is recommended. CT scan or MRI can be used to evaluate anterior or posterior dislocation, fractures, great vessels, and trachea.

VII. Special Studies

Not applicable.

VIII. Diagnosis

Sternoclavicular.

IX. Differential Diagnosis

Fracture.

X. Treatment

(See Wirth and Rockwood;[7] Level of Evidence B, nonquantitative systematic review.)

- Initially: Use RICE (rest, ice, compression, elevation) plus analgesics (as needed) plus nonsteroidal anti-inflammatory drugs (NSAIDs) plus sling immobilization.
- Long-term:
  Grade I — Wean patient from immobilization at 5 to 10 days with a gradual return to use of the arm.
  Grade II (subluxation) — Reduction may be required; draw the shoulders back and hold them with a clavicle strap. Protection with a sling and/or clavicle strap for a period of 4 to 6 weeks is recommended.
  Grade III (dislocation) — *Anterior:* Have the patient supine on the edge of the table with a sandbag between the shoulders. With the upper extremity at 90° of abduction and extension, apply traction in line with the clavicle. Use a post-reduction figure-eight dressing for 6 weeks. Operative repair is debatable. *Posterior:* Have the patient supine with a sandbag between the shoulders. Apply lateral traction to the abducted arm which is then gradually brought back into extension. Use a post-reduction figure-eight dressing for 6 weeks. Because of potential damage to the posterior structures, some physicians prefer operative repair. Either way, reduction should be performed with extreme care for the posterior vasculature.

XI. Complications

Injury to the pulmonary vessels, trachea, superior vena cava, esophagus, and other mediastinal structures, especially with posterior dislocations. Risk of traumatic arthritis and chronic joint instability.

XII. Prevention
None

ACROMIOCLAVICULAR SPRAIN/SUBLUXATION/ DISLOCATION

I. Epidemiology
The acromioclavicular joint is commonly injured in contact sports and falls off bicycles.

II. Mechanism
The mechanism of injury is a direct fall or blow to the point of the shoulder at the lateral edge of the acromion. Indirect force from a fall on an outstretched arm or blow to the upper back can also cause injury to the joint.[3]

III. Anatomy
Acromioclavicular injuries are classified as grades I thru VI:

- Grade I — acromioclavicular ligament and capsular stretching
- Grade II — acromioclavicular ligament disruption with slight upward migration of the clavicle and tearing of the coracoclavicular ligaments
- Grade III — acromioclavicular and coracoclavicular ligaments and intraarticular meniscus disruption; acromioclavicular joint dislocation with the clavicle displaced upward relative to the acromion (Figure 45.13)

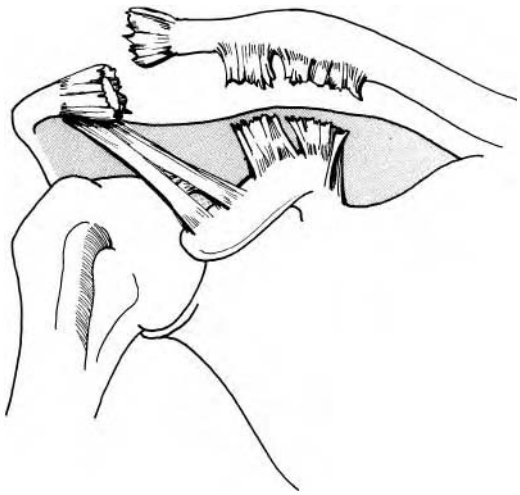


Figure 45.13 Grade III acromioclavicular dislocation.

- Grade IV — a grade III injury with the clavicle anatomically displaced upward and posterior into or through the trapezius
- Grade V — Severe upward dislocation of the distal clavicle relative to the acromion with complete destruction of the acromioclavicular and coracoclavicular ligaments and disruption of the deltoid and trapezius muscle attachments to the clavicle; clavicle may pierce the muscle and even the skin in some cases
- Grade VI — Inferior dislocation of the clavicle underneath the coracoid; injury to the underlying neurovascular structures likely

IV. and V. Symptoms and Signs

- Grade I — Swelling and tenderness over the acromioclavicular ligament with no instability of the distal clavicle. Patient performs the cross-over test with minimal or no pain.
- Grade II — Snapping of the acromioclavicular (AC) joint on shoulder motion with swelling and tenderness over the acromioclavicular ligament and some lack of symmetry when compared to the normal side. Tenderness and slight instability of the distal clavicle with downward pressure are observed. The cross-over test is painful, yet the patient can resist pressure when the examiner pushes the elbow downward.
- Grades III to VI — Swelling and marked tenderness over the acromioclavicular and coracoclavicular ligaments and marked asymmetry with high-riding clavicle. Marked instability of the distal clavicle is observed. The patient is unable to perform the cross-over test.

VI. Diagnostic Imaging
Anteroposterior and axial x-ray views can demonstrate widening of the AC joint and high-riding clavicle, and Alexander view demonstrates posterior clavicular dislocation:

- Grade I — normal
- Grade II — 1.3 cm coracoacromial separation
- Grades III to IV — greater than 1.3-cm separation or >50% increase in the distance when compared to the uninjured side

VII. Special Studies
Not applicable.

VIII. Diagnosis
Acromioclavicular.

IX. Differential Diagnosis
Contusion, fracture, synovitis, osteoarthritis.

X. Treatment
(See Phillips et al.,[8] Bethis et al.,[9] and Larsen et al.;[10] Level of Evidence A, quantitative systematic review.)

- Initially: Use RICE plus analgesics as needed plus NSAIDs plus sling or shoulder immobilizer.
- Long-term:
  Grades I and II — Wean out of immobilization as tolerated; use early physical therapy (ROM + PREs) for several weeks.
  Grade III — Same as above followed by physical therapy for 6 to 8 weeks; surgery is occasionally indicated for the throwing arm of an athlete; however, in non-athletes, significant benefit from surgery is questionable.
  Grade IV — The clavicle must be dislodged from the trapezius, usually with closed reduction, but occasionally operative intervention is required.

XI. Complications
Traumatic arthritis, persistent decreased range of motion and strength in addition to pain.

XII. Prevention
None.

GLENOHUMERAL INSTABILITY: SPRAIN/SUBLUXATION/ DISLOCATION

I. Epidemiology
Instability and laxity are terms often applied to the shoulder. Laxity is the asymptomatic translation of the humeral head on the glenoid. It may be a normal variant and represent a necessary feature of the soft tissue about the shoulder required for glenohumeral rotation. Instability is the excessive translation of the humeral head on the glenoid occurring during active shoulder rotation in association with symptoms. It represents varying degrees of injuries to dynamic and static structures that function to contain the humeral head in the glenoid. Throwing and racquet sports can also produce anterior glenohumeral instability from repetitive stretching (Level of Evidence C, nonrandomized clinical trial).[11] A sprain occurs when there is sequential tearing of the glenohumeral ligaments and capsule with pain but no obvious displacement of the humeral head. Subluxation is the symptomatic increased humeral translation beyond that permitted by normal tissue laxity, but without complete separation of the articular surfaces. Dislocation is the complete separation of the articular surfaces of the glenoid and humeral head; 85% of dislocations detach the glenoid labrum (Bankart lesion). Without the protection of the labrum, recurrent subluxations and dislocations can potentially lead to ectopic bone formation evident on radiographic images. In dislocations, 95% are anterior and inferior. Posterior injuries are uncommon and pure superior and inferior injuries are very rare.

II. Mechanism

A. Acute — *Anterior:* Most cases of dislocation are anterior. Major trauma is usually involved but the shoulder does not often dislocate as a result of a direct blow (Level of Evidence C, nonquantitative systematic review).[12] A combination of forces stresses the abducted extended and externally rotated arm, which applies leverage to the anterior capsule, glenohumeral ligaments, and rotator cuff. The humeral head is forced anteriorly and out of the glenoid fossa. The most common type of anterior dislocation is the subcoracoid dislocation in which the humeral head is anterior to the glenoid and inferior to the coracoid process. The head of the humerus is anterior and below the glenoid fossa in subglenoid dislocation. The head of the humerus lies medial to the coracoid process at the inferior border of the clavicle in subclavicular dislocation. Significant trauma is associated with an intrathoracic dislocation where the head of the humerus lies between the ribs and thoracic cage (Level of Evidence C, historical uncontrolled study).[13] *Posterior:* Can be caused by direct force such as a direct blow to the anterior aspect of the glenohumeral joint. A posterior dislocation can occur as a result of an indirect force such as a fall on an outstretched

arm with the shoulder in internal rotation, adduction, and flexion. When blocking, as in football, a direct axial load applied to a flexed arm, adducted, and internally rotated may cause a posterior subluxation or dislocation of the humeral head. Seizure or electric shock can produce a muscular contraction forceful enough to cause posterior displacement of the humeral head (Level of Evidence C, historical uncontrolled study).[14]

B. Chronic — Glenohumeral translation is less than that detectable on physical examination as clinical subluxation but is sufficient enough to cause excessive edge loading or shearing stress to the labrum (Level of Evidence B, nonrandomized clinical trial).[15] The result is the failure of the humeral head containment during motions such as throwing.

III. Anatomy

A. Sprain — sequential tearing of the glenohumeral ligaments and capsule
B. Subluxation — joint laxity causing more than 50% of the humeral head to passively translate over the glenoid rim without dislocation or causing the humeral head to actively translate more than 4 mm from the center of the glenoid
C. Dislocation — joint instability with the humeral head losing contact with the glenoid and lodging beside the joint; of anterior dislocations, 85% detach the glenoid labrum (Bankart lesion), potentially leading to ectopic bone formation with recurrent dislocations
B. Chronic injury[9] — less intense and less localized pain; sensation of instability or apprehension with overhead activities; history of previous dislocations and progressively less trauma needed to sublux or dislocate shoulder

IV. and V. Symptoms and Signs

A. Sprain — Capsule is tender to palpation; no instability; positive apprehension and relocation test (see Figure 45.14).
B. Subluxation — Sensation of the shoulder "popping" out and back into place; the pain present may be associated with paresthesias down the arm or a sensation of the arm "going dead";[9] patient resists placing shoulder in abduction and external rotation and extension. Capsule is tender with mild swelling; positive apprehension/relocation test. Brachial plexopathy commonly involves the axillary, musculocutaneous or suprascapular nerves.[13]

Figure 45.14 Apprehension test; testing for anterior stability.

C. Dislocation — *Anterior:* Acute severe pain with loss of ROM and possible numbness or weakness present before or after reduction (Level of Evidence C, historical uncontrolled study).[16] Patient presents with arm slightly abducted and externally rotated and held firmly by the other arm; prominent acromion process and coracoid process may not be identified because of swelling. Shoulder assumes squared-off appearance with anterior shoulder fullness. Movement of the arm is painful and limited, especially with attempted adduction or internal rotation. *Posterior:* Arm is fixed in the adducted position and internally rotated; coracoid process is more obvious. On the dislocated side, the anterior aspect of the shoulder is flat; the posterior aspect of the shoulder is rounded and more pronounced than the normal shoulder. External rotation of the shoulder is blocked; severely limited abduction.

VI. Diagnostic Imaging

Pre-reduction (if possible) and post-reduction films reveal displacement of the humerus and associated avulsion fractures. Routine AP views in internal and external rotation in the chest plane, AP views in the scapular plane, and axillary lateral views are recommended for posterior dislocations. True lateral view of scapula and modified axillary lateral (west point) view are good for chip fractures of the anterior–inferior glenoid rim and labral tears (Bankart lesion). The Hill–Sachs view identifies posterior humeral head defects caused by compression of the head against the glenoid during subluxation. The Stryker notch view is a posterior view of the humeral head that helps check for a Hill–Sachs lesion. Magnetic resonance imaging is recommended for detailed assessment of the entire shoulder but is highly equipment and technique dependent; if optimum resolution is available, it can be used to identify bone bruising, muscle atrophy, neurovascular structures, and the extent of rotator cuff and biceps tears. It can also evaluate intraarticular pathology, such as the presence of ganglion cysts, glenoid labrum tears, and articular cartilage damage (Level of Evidence C, nonquantitative systematic review).[17] Computed tomography is useful for evaluating bony details especially with complicated fractures.

VII. Special Studies

Not applicable.

VIII. Diagnosis

Glenohumeral.

IX. Differential Diagnosis

Fracture (avulsion, humerus), acromioclavicular joint sprain, glenoid labral tear or fracture, rotator cuff injury, atraumatic osteolysis of distal clavicle.

X. Treatment

(See Bottoni et al.;[18] Level of Evidence B, lower quality randomized controlled trial.)

- Initially: Use reduction, immobilization with a sling and swathe or shoulder immobilizer, RICE, NSAIDs, and analgesics as needed. For a *sprain*, wean patient from immobilization in 5 to 10 days; use of arm should return gradually. For *subluxation*, use a sling and swathe or shoulder immobilizer for 2 to 4 weeks, then initiate aggressive rehabilitation. For a *dislocation*, perform anterior reduction by gentle external rotation using the following methods (Figure 45.15):
  - Kocher — The patient lies supine with the elbow externally rotated and flexed to 90°. Progressive traction is applied to the humerus followed by gentle adduction and internal rotation of the arm after the humeral head slips into the glenoid.
  - Stimson — With the patient lying prone on a table, attach a 5-pound weight with two half hitches to the wrist; gravity and the weight together tend to relocate the humerus. Allow 20 to 30 minutes; muscle relaxants and compression of the humeral head may be necessary.
  - Milch — The arm is externally rotated and abducted overhead while the humeral head is pushed back into place.
  - Hippocratic, double sheet, or Rockwood method — With the athlete supine, gentle steady traction is applied to the affected arm in 30 to 45° of abduction, and counter-traction is applied in the opposite direction by means of a sheet or swathe wrapped around the upper thorax. The foot should not be placed on the chest wall for counter-traction as it may slip into the axilla and damage the brachial plexus. The affected arm should be flexed in muscular individuals to relax the biceps tendon and a wrist lock technique used for maximum traction.
  - Scapular rotation maneuver — With the patient prone, longitudinal traction is placed on the flexed affected arm; the inferior angle of the ipsilateral scapula is rotated toward the spine so that the glenoid and humerus are aligned; a pillow under the athlete's chest facilitates the relocation.
- For *posterior reduction*, with the patient supine, apply traction to the adducted, internally rotated, flexed arm in the line of deformity, along with a gentle lifting and internal rotation of the humeral head back into the glenoid fossa; open reduction is indicated following 1 to 2 unsuccessful trials.
- Long-term: Immobilization for 3 to 6 weeks, depending on age and activity level, is recommended. The shoulder

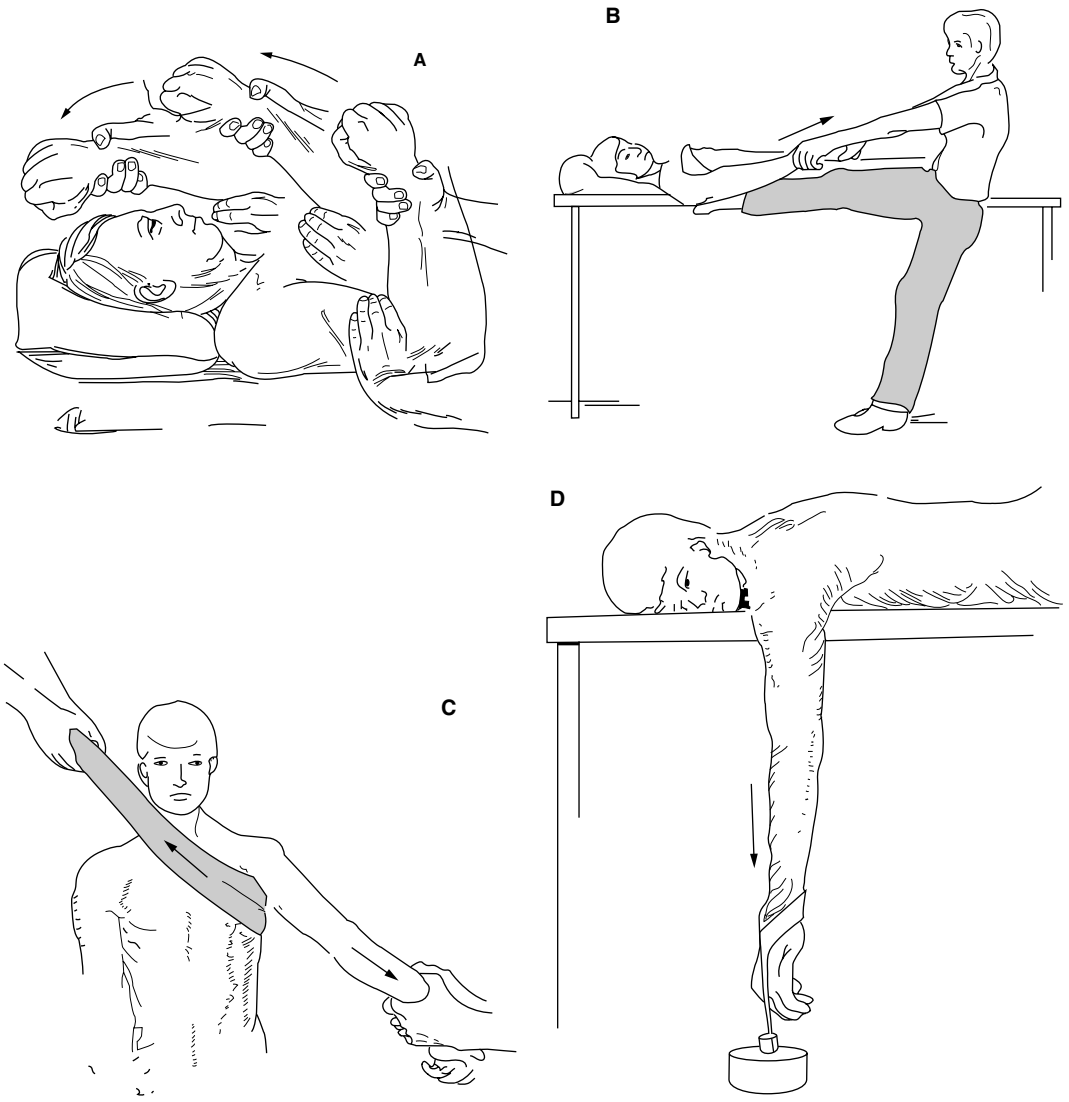


Figure 45.15 Shoulder reduction techniques.

spica in the neutral position is preferred by some for posterior dislocations. Range of motion and isometric strengthening should be started early, followed by a resistive strengthening program. An exercise program should emphasize both internal and external rotators as well as large scapular muscles. Surgery is indicated for persistent instability.

XI. Complications

The incidence of fractures rises with age; complications include compression fracture of the humeral head (Hill–Sachs lesion), fracture of the glenoid lip (anterior or posterior), fracture of the greater or lesser tuberosity, fracture of the acromion or coracoid, rotator cuff tear, nerve injury (axillary, brachial plexus, musculocutaneous), vascular damage.

XII. Prevention

Flexibility and strengthening exercises of internal rotator muscles will improve anterior instability, and a shoulder harness (e.g., Denison–Duke Wyre shoulder vest, Simply Stable shoulder stabilizer) can be helpful.

GLENOID LABRUM TEARS

I. Epidemiology

Glenoid labrum tears may occur from repetitive shoulder motion or acute trauma. In the throwing athlete with repeated anterior shoulder subluxation, tears of the middle and inferior portion of the labrum may occur, leading to instability. Glenoid labrum tears may also result from anterior instability during the release phase of throwing, secondary to the long head of the biceps tendon pulling on the anterior labrum. Weightlifters may also develop glenoid labrum tears from repetitive bench pressing and overhead pressing. Most patients present with nonspecific shoulder pain associated with activity. Complicating the presentation is that the majority of lesions reported in the literature are associated with other shoulder disorders such as rotator cuff tears, acromioclavicular joint disorders, and instability (Level of Evidence C, nonquantitative systematic review).[20] In the largest review in the literature, out of 140 lesions, only 28% were isolated. The majority of glenoid labral tears occur from traction and compression injuries.[18] Sudden contraction of the biceps can occur with overhead athletes during the release phase of throwing. Compression injuries occur with forceful subluxation or dislocation of the humeral head over the fibrocartilaginous labrum. Tears of the glenoid labrum may also occur from acute trauma such as falling on an outstretched arm. Also, horizontal adduction and internal rotation during the acceleration phase of throwing can damage the labrum by applying a sheering stress across the labrum.[12]

II. Mechanism

Multidirectional instability (MDI) occurs on a background of generalized shoulder-capsule laxity which may be genetic (35%) (e.g., Ehlers–Danlos syndrome) or environmental (previous trauma, 30%; overuse, 35%) (Level of Evidence C, nonquantitative systematic review).[19] The instability may be voluntary (patient consciously subluxates/dislocates joint) or involuntary.

III. Anatomy

The labrum is a triangular structure located around the periphery of the glenoid. It is composed of dense fibrous tissue rather than cartilage (Level of Evidence C, historical uncontrolled study)[21] and functions as a static stabilizer of the glenohumeral joint (Level of Evidence B and C, nonquantitative systematic review).[22,23] The average depth of the articular surface of the glenoid in the transverse plane is 2.5 mm. The labrum serves to deepen the glenoid by an additional 2.5 mm both anteriorly and posteriorly, adding to the static stability of the joint (Level of Evidence C, nonquantitative systematic review).[24] The glenoid labrum and biceps tendon are closely associated. The biceps tendon is attached to the superior portion of the labrum and often included in the classification of superior labrum tears. (Figure 45.16). Superior labral anterior-to-posterior (SLAP) lesions describe anatomic lesions of the superior glenoid labrum and biceps anchor. Glenoid labrum tears that occur in close proximity to the biceps, in the superior third of the labrum, can demonstrate symptoms similar to those of biceps tendon subluxation.

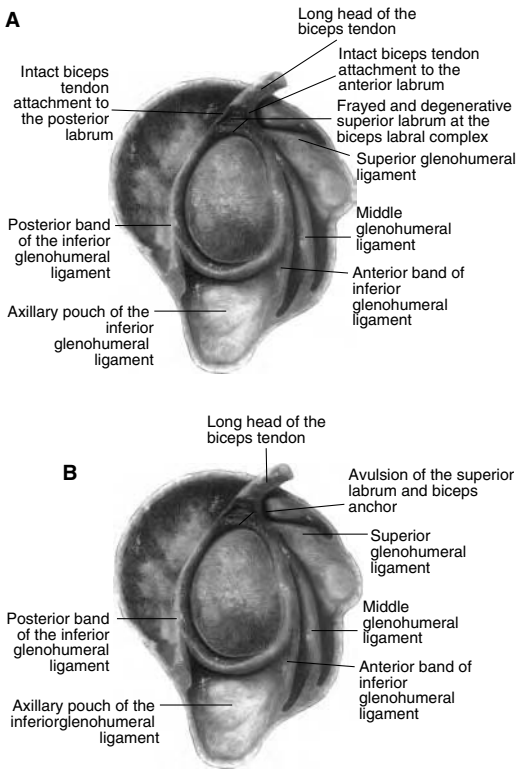


Figure 45.16 Labrum injury types I and II. (From Stoller, D.W., *MRI, Arthroscopy, and Surgical Anatomy of the Joints*, Lippincott-Raven, Philadelphia, 1998. With permission.)

IV. and V. Symptoms and Signs

An audible or palpable clunk occurs as the tear flips in and out of the joint. In throwing athletes, more pain is sensed with release because of the deceleration effect of the biceps pulling on the torn labrum. With other tears, a sense of popping, clicking, or snapping in the joint is experienced during abduction and external rotation (e.g.,

cocking phase). Anterior humeral joint tenderness and pain with abduction and external rotation are observed. The labral clunk test is positive, the active compression (O'Brien) test is positive, and the biceps load test is positive (Figure 45.17).

Pain and instability are often insidious and bilateral. Apprehension tests are present in all directions, the sulcus sign is positive, and increased humeral joint play is present.

VI. Diagnostic Imaging

Standard x-rays often demonstrate Hill–Sachs and Bankart lesions. An injury involving the labrum only will appear normal; if the glenoid is involved, a fracture (bony Bankart lesion) may be present. Magnetic resonance imaging provides excellent evaluation of labral injuries (Level of Evidence B, lower quality randomized controlled trial).[25] Especially with a dedicated extremity coil, a good correlation with surgical findings has been found. Ultrasonography also has a promising role in the evaluation of the glenoid labrum, particularly in excluding tears when the labrum appears normal on sonography (Level of Evidence B, lower quality randomized controlled trial).[26]

VII. Special Studies

None indicated.

VIII. Diagnosis

Glenoid lateral tear.

IX. Differential Diagnosis

Instability, rotator cuff tear, impingement.

X. Treatment

Of patients with MDI secondary to ligamentous laxity, 50 to 70% respond to prolonged conservative rehabilitation (6 to 12 months). MDI secondary to trauma often requires surgical correction. Voluntary MDI secondary to psychological problems is often resistant to all forms of intervention.

- Initially: Use conservative management with RICE and NSAIDs with sling immobilization.
- Long-term: Recommend a program of flexibility and muscle strengthening exercises, specifically for the posterior rotator cuff muscles (as demonstrated in Figure 45.18). If symptoms do not resolve, these injuries require definitive treatment through arthroscopic surgery.

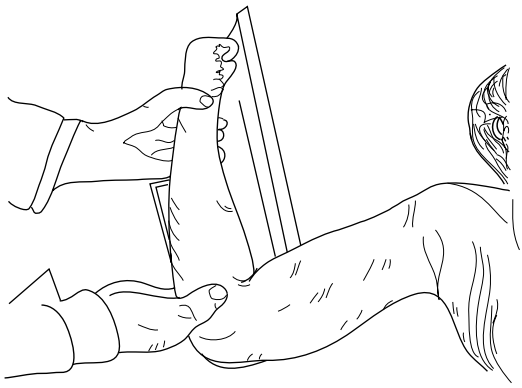


Figure 45.17 Biceps load test.

XI. Complications

Disability secondary to arthritis.

XII. Prevention

Proper throwing mechanics; progressive training and conditioning.

IMPINGEMENT SYNDROME (BURSITIS, TENDINOSIS, SUPRASPINATUS SYNDROME)

I. Epidemiology

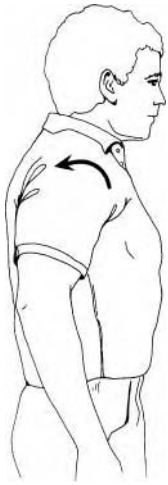
Impingement syndrome is the most common soft-tissue injury of the shoulder due to repetitive use of the arms above the horizontal plane, such as in throwing and racquet sports, swimming (40 to 60% of swimmer's shoulder), weightlifting, and javelin (Level of Evidence C, nonquantitative systematic review).[20,27–29] Inflexibility, fatigue, and mechanical and technique errors are risk factors.

II. Mechanism

With repetitive microtrauma from throwing, stroking, and serving, a dynamic imbalance occurs between the external (rotator cuff muscles) and internal (pectoralis and latissimus dorsi muscles) rotators. This allows the humeral head and its rotator cuff attachments to migrate proximally and impinge the undersurface of the acromion and the coracoacromial ligament. Symptoms can also be caused by direct trauma to the acromion and or chronic irritation under the coracoacromial arch due to a hooked (type 3) acromion or subacromial osteophyte.

III. Anatomy

The rotator muscle group consists of the supraspinatus, infraspinatus, subscapularis, and teres minor muscles. Recurrent inflammation of

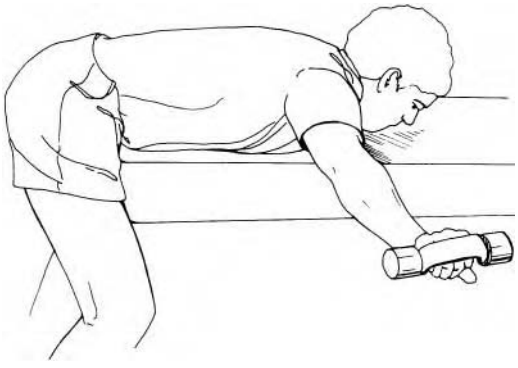


(A) Shoulder shrug with scapular abduction.

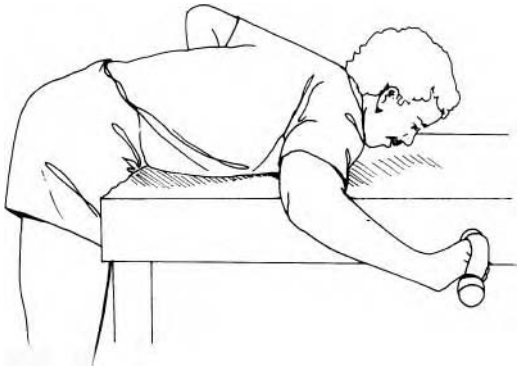
(B) Strengthening the anterior portion of the deltoid muscle by forward-flexion exercises.

(C) Strengthening the middle portion of the deltoid muscle by abduction exercises.

(D) Strengthening the supraspinatus muscle by internally rotating and abducting the humerus.



(E) Shoulder position for strengthening the external rotators.

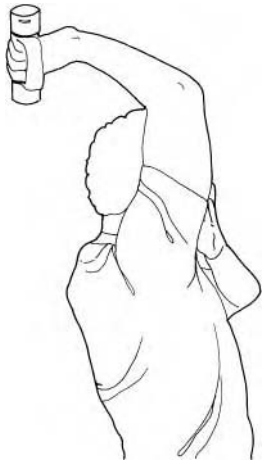


(F) Strengthening exercise for the posterior portion of the deltoid muscle and the rotator cuff.

Figure 45.18 Muscle strengthening exercises for the shoulder.

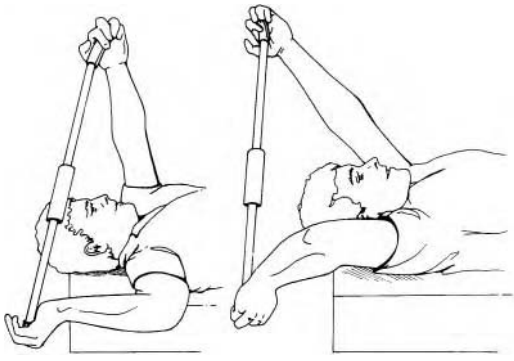
(G) Strengthening the shoulder depressor, horizontal adductor, and internal rotator muscles.

(H) Modified push-up.

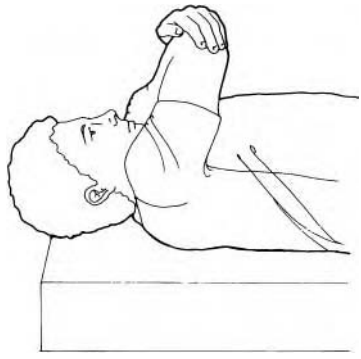


(I) French curl exercise for strengthening the triceps muscle.

(J) Biceps or elbow curl for strengthening the biceps muscle.



(K) External rotation flexibility exercises performed with the shoulder abducted from 90° to full abduction.



(L) Stretching exercise for the posterior shoulder structures.

FIGURE 45.18 (Continued)

the rotator cuff, especially the avascular area of the supraspinatus tendon, the long biceps tendon, and the subacromial bursa, leads to impingement.[25]

IV. and V. Symptoms and Signs

Initially, discomfort may be minimal and present deep within the shoulder during activity with no obvious loss of strength. Symptoms progress to significant pain that is frequently worse at night and with overhead movements. Pain-free range of motion is restricted, with a painful arc of abduction 70 to 120° (i.e., positive impingement sign). In the worse stage, the patient may experience constant pain with any motion. Also observed are point tenderness over the greater tuberosity and anterior acromion (coracoacromial ligament), tenderness over the biceps tendon proximally in the bicipital groove, pain with supraspinatus testing, positive straight-arm raising test, resisted forward flexion of the humerus with the forearm supinated and the elbow extended (i.e., Speed's test), positive resisted supination forearm test (i.e., Yergason's sign), and weakness of rotator cuff and biceps. Atrophy may be present over the supraspinatus.

VI. Diagnostic Imaging

X-rays tend to be supportive but not diagnostic; they may reveal sclerosis and osteophyte formation on the anterior–inferior acromion, an enlarged or hooked (type 3) acromion, and diminished distance (≥5 mm) between the acromion and proximal humeral head.[2,14] Magnetic resonance imaging demonstrates excellent visualization of partial and full cuff tears, inflammation of the subacromial bursa and supraspinatus tendon, and tears of the capsule and subscapularis.[15] Ultrasound is good for cuff tears and abnormal cuff mechanics. Arthrogram may reveal dye leakage superiorly through the defect and outside the confines of the rotator cuff, usually adjacent to the undersurface of the acromion. Sensitivity decreases with partial tears and complete tears that have sealed.

VII. Special Studies

Local anesthetic injection of subacromial space improves shoulder pain and range of motion.

VIII. Diagnosis

Impingement syndrome.

IX. Differential Diagnosis

Acute traumatic bursitis, subluxating shoulder, arthritis of AC or glenohumeral joint, cervical disc, adhesive capsulitis, suprascapular nerve injury, glenoid labrum tear, thoracic outlet syndrome, atraumatic osteolysis of the distal clavicle.

X. Treatment

- Initially: Use RICE, NSAIDs, and analgesics as needed. Total rest may be necessary if the patient experiences pain that is sufficiently disabling to affect performance during and after activities.
- Long-term: Implement injection therapy with steroid and local anesthesia into subacromial bursa (not recommended for young athletes) (Level of Evidence B, lower quality randomized controlled trial; Level of Evidence A, randomized controlled trial)[30,31] Physical therapy improves range of motion as well as rotator cuff and scapular stabilizer muscle strength. Modalities such as electric stimulation and ultrasound may help with symptoms but do not necessarily improve healing (Level of Evidence A, lower quality randomized controlled trial).[32] Surgical decompression (open vs. arthroscopy) of subacromial space, acromioplasty, and coracoacromial ligament resection should be considered for patients who fail conservative management (Level of Evidence C, nonquantitative review; Level of Evidence A, randomized controlled trial).[33,34]

XI. Complications

Rotator cuff arthropathy or tears, adhesive capsulitis, decreased arm/shoulder function and strength.

XII. Prevention

- Flexibility and strength training of the rotator group (Level of Evidence A, randomized controlled trial)[35]
- Improved biomechanics for swimming (avoidance of hand paddles and sprints, increased body roll, and alternation of breathing sides), weightlifting (avoidance of overhead training such as bench and military presses), pitching (slower "opening up" — turning body toward home plate well ahead of the throwing shoulder)

ROTATOR CUFF STRAIN

I. Epidemiology

Acute tears are rare in the young. Chronic overuse and tears (e.g., skiing fall, body surfing, throwing, equestrian activities) occur in the "over-45 weekend warrior."

II. Mechanism

Indirect force to the abducted arm and repetitive microtrauma are the usual mechanisms of injury.

III. Anatomy

Acute or chronic tearing of the rotator cuff muscle–tendon unit (see impingement syndrome). Most common site (critical zone) is the relatively avascular supraspinatus tendon just proximal to the greater tuberosity of the humerus.

IV. and V. Symptoms and Signs

- Acute: significant pain, inability to abduct arm against minimal resistance
- Chronic: gradual onset of pain (initially nocturnal, later persistent) and weakness, especially with 70 to 120° of arm abduction and external rotation; palpable crepitus during supraspinatus abduction; positive impingement sign; abduction not restored by local anesthetic injection into subacromial space; variable loss of strength and atrophy of cuff muscles

VI. Diagnostic Imaging

X-rays are usually normal but may show proximal migration of humeral head, subacromial spurring, and calcific deposits in chronic tears. Magnetic resonance imaging is sensitive and specific for complete tears and may reveal evidence of chronic rotator cuff wear in absence of tears. Ultrasound is sensitive and specific in 85 to 95% of complete tears; possible increased hyperemia in absence of tears.

VII. Special Studies

Not applicable.

VIII. Diagnosis

Rotator cuff tear.

IX. Differential Diagnosis

Impingement syndrome, rotator cuff tear, cervical radiculopathy.

X. Treatment[32]

- Initially: Use RICE, NSAIDs, analgesics, and, as needed, a sling.
- Long-term: Prolonged physical therapy (ROM + PRE) should emphasize abduction and external rotation and strengthening scapular stabilizer muscles. Arthroscopic or open surgery should be followed by physical therapy when conservative (non-operative) management fails.

XI. Complications

Complete tear, recurrence.

XII. Prevention

See Impingement Syndrome section above.

CALCIFIC TENDINITIS

I. Epidemiology

A different entity from impingement, calcific tendinitis occurs in up to 8% of the population over the age of 30 with shoulder pain. Of asymptomatic patients, 30% demonstrate rotator cuff calcification, of which 35 to 45% will eventually become symptomatic, possibly suddenly without any obvious trauma or overuse.

II. Mechanism

A history of repetitive microtrauma, possibly in combination with hypoxic changes in the tendon, can lead to calcium deposition. The two types of deposits are amorphous deposits, which are characteristic of resorptive phase of healing but associated with exquisite pain, and well-circumscribed, sand-like deposits consistent with the asymptomatic, chronic phase.

III. Anatomy

Calcification (calcium apatite) in the rotator cuff, particularly the supraspinatus, 1 to 2 cm from the tendon insertion in the greater tuberosity.

IV. and V. Symptoms and Signs

The arm is held across the chest in the protected position. Onset of severe shoulder pain is sudden; symptoms are present with activity and at rest. Patient experiences difficulty in sleeping, and any shoulder motion is extremely painful. Point tenderness over the area is noted.

VI. Diagnostic Imaging
Routine shoulder views reveal deposits, commonly seen in the area of the supraspinatus tendon at the point of insertion into the greater tuberosity, which are best visualized with the humerus externally rotated. The lateral scapula view is good for determining the precise location. No correlation exists between the size of the deposit and symptoms. Magnetic resonance imaging demonstrates soft tissue calcification. Ultrasound demonstrates soft tissue calcification and allows for simultaneous therapeutic intervention (e.g., injection).

VII. Special Studies
Not applicable.

VIII. Diagnosis
Calcific tendinitis.

IX. Differential Diagnosis
Rotator cuff impingement, tendinosis or tear, biceps tendinosis, subacromial bursitis, adhesive capsulitis, glenohumeral arthritis.

X. Treatment

A. Initially: Use RICE, NSAIDs, and analgesics as needed plus sling immobilization.
B. Long-term: Injection therapy is helpful, particularly with type 1 deposits.

XI. Complications
Rotator cuff tear, chronic disability.

XII. Prevention
Modify training program to improve biomechanics.

ADHESIVE CAPSULITIS (FROZEN SHOULDER)

I. Epidemiology
Adhesive capsulitis (frozen shoulder) is a poorly defined syndrome in which both active and passive shoulder motion is lost because of soft tissue contracture. An insidious onset of symptoms commonly occurs among individuals ages 40 to 60 years old. Females are affected more than males. It is believed to be a benign self-limited disorder that tends to resolve over 1 to 2 years, although some patients are often left with some residual loss of motion. The etiology is unknown but it is more common in patients with diabetes mellitus, for which it is frequently bilateral and resistant to treatment. The pathologic process is characterized by range of motion limited initially by pain secondary to inflammation (capsulitis), then mechanically from adhesions. Three stages have been defined. Stage 1 has pain with motion but no mechanical limitation in range. Stage 2 has pain with some functional limitations in range of motion. Stage 3 is less painful but the range of motion is extremely limited mechanically by adhesions. It may be secondary to other pathologies such as impingement, fractures, cervical radiculopathy, coronary artery disease, reflex sympathetic dystrophy, and dislocations. It has been suggested that adhesive capsulitis is a fibrosing condition, rather than an inflammatory one, because inflammatory cells are absent or scanty on histologic studies (Level of Evidence B, basic scientific research).[36]

II. Mechanism

III. Anatomy
Adhesive fibrosis and scarring between the capsule, rotator cuff, subacromial bursa, and deltoid.

IV. and V. Symptoms and Signs
Onset of poorly localized pain is insidious, frequently extending down the arm; nocturnal pain is common. Shoulder stiffness and decreased active and passive motion are noted, particularly in internal and external rotation. Palpable mechanical block to motion is with or without a tender endpoint.

VI. Diagnostic Imaging
X-Rays are usually negative. Magnetic resonance imaging may not reveal much evidence of the adhesions if resolution is poor and synovium thickness is less than 4 mm (Level of Evidence C, nonrandomized controlled study).[37] Ultrasound is more sensitive to revealing adhesions. CT/arthrogram demonstrates a decrease in joint volume and may improve condition through forced joint distention.

VII. Special Studies
Not applicable.

VIII. Diagnosis
Adhesive capsulitis.

IX. Differential Diagnosis
Rotator cuff tendinosis, subacromial bursitis (impingement syndrome), rotator cuff tear, cervical

radiculopathy, any fracture or dislocation of the shoulder, arthritis, thoracic outlet syndrome.

X. Treatment

(See Arslan and Celiker[38] and Kivimaki and Pohjolainen;[39] Level of Evidence B, randomized controlled study.)

A. Initially: Use NSAIDs with aggressive physical therapy.
B. Long-term: Manipulate the shoulder glenohumeral joint; apply steroid injection therapy into the shoulder joint, followed immediately by aggressive physical therapy. Manipulation under anesthesia can restore range of motion and should be followed by steroid injection and physical therapy. Arthroscopic surgery is another option.

XI. Complications

Chronic disability.

XII. Prevention

Recommend early progressive physical therapy after shoulder injuries and have patient avoid prolonged immobilization.

SCAPULOTHORACIC PROBLEMS (BURSITIS, WINGING)

I. Epidemiology

Bursitis is common in sports requiring repetitive shoulder motion (e.g., swimming, throwing, racquet sports). Winging is spontaneous following strenuous activity, such as cycling, swimming, golf, backpacking, or weightlifting, or in collision/contact sports and may be due to long thoracic nerve injury.

II. Mechanism

Usually caused by high-velocity repetitive microtrauma (shotputting, back-scratch position during tennis serve) on a background of excessive shoulder motion; bursitis and winging can also follow direct (e.g., a forceful blow) or indirect (e.g., excessive traction) trauma.

III. Anatomy

Architectural malalignment along the inferior–medial scapular border leads to bursal inflammation. Paralysis of the serratus anterior can follow direct trauma of the long thoracic nerve.

IV. and V. Symptoms and Signs

Pain, grating, and snapping at the inferior angle of the scapula are noted with shoulder elevation. Dull ache or pain can be noted in the shoulder girdle usually is asymptomatic. Crepitus observed with arm elevation, and the scapula rolls over the posterior chest wall; palpable mass at the inferior–medial angle is best felt with 60° abduction and 30° flexion of the shoulder. Winging occurs when the arm is brought into the elevated position and is emphasized when the patient pushes against a wall with arms extended. Increased pain with contralateral head tilting or ipsilateral arm elevation is noted.

VI. Diagnostic Imaging

Anteroposterior and lateral views of the proximal humerus in the scapular plane are usually negative. Computed tomography and MRI may provide information.

VII. Special Studies

Electromyograms and nerve conduction studies demonstrate conduction delays and denervation; findings may be minimal in the first 6 weeks.

VIII. Diagnosis

Scapulothoracic.

IX. Differential Diagnosis

Strain fibromyositis.

X. Treatment

Bursitis

A. Initially: NSAIDs
B. Long-term: injection therapy and physical therapy (ROM + PREs)

Winging

Rest and gentle physical therapy (ROM + PREs). While recovery may require 2 years, resolution is rarely complete. Winging *per se* should not be a criterion for return to play.

Consultation: if conservative treatment fails after 6 to 12 months

XI. Complications

Chronic bursitis

XII. Prevention
Improve sport biomechanics (e.g., reduced shoulder movement during cocking position of tennis serve) and use protective equipment.

BICEPS TENDON PROBLEMS (TENDINITIS, SUBLUXATION/DISLOCATION, RUPTURE)

I. Epidemiology
Tendinosis is common in a wide range of throwing, swimming, and racquet sports. In particular, male gymnasts are at risk for biceps tendon injury. Subluxation, dislocation, and rupture often occur on a background of tendinosis, repeated corticosteroid injections, or traumatic injury. Distal ruptures occur predominantly in athletes over 30 years of age. All three pathologies may be associated with an impingement syndrome, cuff disease, or glenohumeral instability.

II. Mechanism
The mechanism of injury is usually repetitive microtrauma (tendinosis). Acute injury commonly (rupture, subluxation/dislocation) occurs following sudden forceful contraction against resistance or direct blow (e.g., checked baseball swing, fast-pitched softball, arm tackle on a quarterback's passing hand).

III. Anatomy
The biceps tendon originates at the superior border of the labrum (long head) and coracoid process (short head), travels intracapsularly under the transverse humeral ligament covering the intertubercular groove, and inserts into the bicipital tuberosity of the radius. It has a relatively avascular area intracapsularly. A tight, narrow groove; rough supratubercular area; or steep medial wall due to a prominent lesser tuberosity can irritate and inflame the tendon. Chronic inflammation can lead to degenerative rupture. Tears of the transverse humeral ligament in association with a shallow groove or low medial wall can produce subluxation or dislocation. Most subluxations and dislocations are medial, but lateral cases have been reported. Ruptures occur in the muscle–tendon junction in the very young and at the top of the groove in the middle-aged or older athlete; 97% are proximal, 3% are distal.[3]

IV. and V. Symptoms and Signs
Patient usually has a significant history of rotator cuff disease or impingement syndrome. Tenderness and crepitus over the bicipital groove are noted, as are vague pain and snapping in the region of the proximal humerus and tendon. A snapping or popping sensation with external rotation indicates subluxation/dislocation; the findings move laterally with external arm rotation or medially with internal rotation. Speed, Yergason, Ludington, and Gilcrest (subluxation/dislocation) tests are positive. Complete rupture may show ecchymosis and a palpable visible gap in muscle belly (complete rupture) with supination and flexion weakness (40 to 50% decrease in distal ruptures, 10 to 20% in proximal ruptures). A "popeye" deformity is seen in proximal long-head ruptures (distal movement of muscle mass); antecubital fossa tenderness (distal rupture) is noted.

VI. Diagnostic Imaging
(See Tuckman[40] and Farin;[41] Level of Evidence B, historical uncontrolled study.) Tunnel views may show a narrow, shallow groove with osteophytes, tendon calcification, or a prominent lesser tuberosity with tendinosis and subluxation/dislocation. Avulsion fracture may be present in a rupture. X-rays can rule out fracture of the lesser tuberosity. Ultrasound is diagnostic for ruptures, associated effusions, and hyperechoic foci (calcifications and loose bodies) seen chronically in tendinosis. Magnetic resonance imaging is diagnostic for rupture. Arthrography has good specificity but low sensitivity. Arthroscopy is definitive for intraarticular ruptures and negative arthrogram.

VII. Special Studies
Not applicable.

VIII. Diagnosis
Bicipital.

IX. Differential Diagnosis
Proximal: above etiologies plus impingement syndrome, rotator cuff disease, glenohumeral instability, partial rupture, osteochondral fracture; distal: anterior capsulitis, annular ligament sprain.

X. Treatment
(See Patton and McCluskey[42] and Curtis and Snyder;[43] Level of Evidence C, nonquantitative review.) For conservative management, initially use RICE, NSAIDs, and analgesics p.r.n. For tendinosis, use counter-force bracing proximal to biceps belly. Injection therapy of biceps sheath or subacromial space may be warranted for very severe symptoms or in recalcitrant cases of pure impingement; tenodesis is recommended if

patient does not respond to conservative management. For subluxation/dislocation, surgical repair of the transverse humeral ligament is recommended. For rupture, surgical repairs for all distal and proximal ruptures in younger, more athletic patients are recommended.

XI. Complications
Rupture, impingement.

XII. Prevention
Athlete should warm up properly, avoid shoulder-motion extremes, initiate a flexibility and strengthening program (especially of the internal shoulder rotators), and modify technique.

THORACIC OUTLET SYNDROME

I. Epidemiology
The condition may be seen in racquet, throwing, and aquatic sports and such occupations as paper hanging, painting, and carpentry (Level of Evidence C, nonquantitative review).[44,45] Occurs at a ratio of 6 females to 1 male, with an average age of occurrence of 36 years.

II. Mechanism
Thirty percent of patients have a history of repetitive activity involving holding the arm over the head. Symptoms are caused by compression of the subclavian artery, vein (effort thrombosis), or lower ramus of the brachial plexus in the interscalene, costoclavicular, or subcoracoid region from trauma, repetitive overhand/overhead movements, or poor posture.

III. Anatomy
Presence of cervical or anomalous first rib, clavicular callus or malunion, shoulder droop, large transverse process (C7), tumor.

IV. and V. Symptoms and Signs
Generally vague, nonspecific ipsilateral upper extremity and neck pain, swelling, and paresthesias (C8 dermatome) are noted with particular positions and activities; referred pain to ipsilateral breast or chest may also be noted. Paresis, pallor, and cold in arm and hand with subclavian artery compression are observed, as are weakness of hypothenar and interossei muscles, hypalgesia, and hypesthesia in the region of the ulnar nerve (C8, T1). Ask the patient to spread the fingers apart against resistance or grip a test card between extended fingers. A positive Adson test is indicated by marked decrease of the radial pulse with abduction, extension, external rotation of the ipsilateral arm in association with a deep breath, and head turning to the same side; the maneuver must be interpreted with caution as it can be falsely positive in the normal patient. A positive Allen test is the same as the Adson test but with head turned toward the opposite side. The elevated arm test (Roos maneuver) is positive when pronounced arm weakness and pain are noted within 2 minutes when the arms are elevated to 90° in abduction, externally rotated with the shoulders and elbows braced back (i.e., military posture), and hands are opened and closed with moderate speed during a 3-minute period;[19] this is the most reliable test. The axial compression test is negative. Rarely, a palpable mass or bruit is found in the supraclavicular area.

VI. Diagnostic Imaging
Anteroposterior, lateral obliques of cervical and upper thoracic spine and chest may demonstrate pathology.

VII. Special Studies
Doppler flow studies, apical lordotic views, angiography, myelogram, electromyography, nerve conduction studies, CT/MRI.

VIII. Diagnosis
Thoracic outlet syndrome.

IX. Differential Diagnosis
Shoulder capsulitis, tendinosis, strain or sprain, neuropathy, herniated cervical disc, carpal tunnel syndrome, radiculopathy, cervical osteoarthritis, coronary ischemia, multiple sclerosis, reflex sympathetic dystrophy, cervical spondylosis, hysteria.

X. Treatment
(See Baker and Liu;[46] Level of Evidence C, nonquantitative review.) Patient should avoid positions or activities that precipitate the symptoms. Initiate physical therapy (ROM + PREs) to increase shoulder girdle and neck muscular tone for patients with hypotonia, posture exercises. Advise patients to avoid sleeping with arms in an elevated position or folded under the pillow.

XI. Complications
Neurovascular compromise.

XII. Prevention
Avoid overhead activity, maintain proper posture, do strengthening/flexibility exercises for shoulder girdle muscles.

CLAVICULAR FRACTURES

I. Epidemiology
One of every 20 fractures involves the clavicle. The majority of these injuries occur in children or in collision/contact sports.

II. Mechanism
Fractures of the distal portion of the clavicle result from a downward blow from above, striking on the point of the shoulder. Fractures of the middle and inner clavicle usually result from direct trauma (less common).

III. Anatomy/Classification
Classification: mid-third (most commonly fractured), distal or interligamentous, and inner third. Fractures of the outer clavicle are subdivided again into three categories:

1. Nondisplaced fractures of the distal clavicle (Type I)
2. Displaced fractures (Type II)
3. Articular fractures (Type III)

IV. and V. Symptoms and Signs
When the fracture is displaced, the shoulder slumps downward and inward and the patient holds the arm against the chest to protect against shoulder movements. Pain may radiate to the trapezius. Direct and indirect tenderness, ecchymosis, and often a visible palpable deformity and crepitus at the fracture site are noted. Deformity may be absent in preadolescent greenstick fractures. Crepitus outside the fracture site suggests subcutaneous emphysema. Distal clavicular fractures may simulate AC separations because of a tendency to ride up and displace the medial part of the fracture; however, no edema or pain of the AC joint is noted. For medial or inner clavicular fractures, pain, tenderness, and swelling at the area of the sternoclavicular joint are noted, as well as pain on hyperabduction of the shoulder. Check lung fields for intact breath sounds.

VI. Diagnostic Imaging
Clavicular, AC, and Alexander views are useful for distal clavicular fractures, sternoclavicular (SC) views for medial fractures. Check for associated scapula fracture, subcutaneous emphysema, and pneumothorax. Weight-bearing views are useful for distal clavicular fractures. Magnetic resonance imaging and CT are useful for evaluating the SC joint.

VII. Special Studies
Not applicable.

VIII. Diagnosis
Clavicular.

IX. Differential Diagnosis
Acromioclavicular separation, SC dislocation/subluxation or epiphyseal injury, contusion.

X. Treatment
(See Jensen et al.;[47] Level of Evidence B, randomized controlled trial.)

Outer Clavicular Fractures

- Type I fractures — Sling is appropriate.
- Type II fractures — Kenny Howard sling should be used due to instability (beware of skin breakdown); figure-eight not useful. Occasionally, a sling alone is acceptable. Internal fixation may be appropriate.
- Type III fractures — Sling is appropriate but condition may eventually require a Mumford procedure.

Mid-Clavicle Fractures
Fractures of the mid-clavicle heal well and rarely require open reduction; a simple sling may be preferable to a figure-eight immobilizer for comfort and fewer complications.

Inner Clavicle Fractures
Fractures of the inner clavicle are treated with a supporting sling unless displaced or associated with a fracture dislocation of the sternoclavicular joint (see SC dislocation). Physical therapy (ROM + PREs) is initiated after 4 to 5 weeks of healing.

XI. Complications
Traumatic arthritis, non-union, pneumothorax, subcutaneous emphysema.

XII. Prevention
None.

OTHER FRACTURES
Fracture of the proximal humerus, acromion glenoid fossa, coracoid, and scapular neck usually

results from any violent force to the shoulder and should always be considered part of the differential diagnosis of acute painful shoulder injury. The trauma may follow a direct blow (e.g., fall on the upper arm) or indirect forces (e.g., fall on outstretched hand with an extended elbow or sudden contraction of arm flexors or rotators, such as occurs in throwing or wrestling). Fractures may be associated or confused with glenohumeral instability, acromioclavicular separation, or a simple contusion. A limited range of motion is usually noted, as well as significant disability, swelling and paresthesias around the shoulder, and, in the cases of coracoid and acromion fractures, point tenderness over these structures. Routine shoulder films will usually confirm the diagnosis. Be aware of an unfused acromial epiphysis masquerading as a fracture in a prepubertal patient.

Treatment of stable coracoid, acromion, glenoid fossa, and scapular neck fractures is conservative. Open reduction is reserved for unstable fractures. Minimally displaced fractures of the proximal humerus (80% of cases) usually respond well to 3 to 4 weeks of a shoulder immobilizer or sling. Two-part fractures (10% of cases) are usually amenable to closed reduction and an immobilizer. Greater tuberosity fractures (>1 cm displacement) and some shaft displacements require open reduction and internal fixation, as do three-part (30% of cases) and four-part (40% of cases) fractures, as well as articular surface fractures of more than 20%.

Humeral stress fracture has been reported in over-30 amateur baseball players. Treatment consists of a shoulder immobilizer or sling for 1 to 2 weeks. A 3-month conditioning program is preventive.

Severe trauma (e.g., stock car racing, equestrian events, pile-up in rugby or football) is required to fracture the body of the scapula. Associated injuries include fractures of the vertebrae, sternum, and ribs; pneumothorax; brachial plexus injuries; and subcutaneous emphysema. Localized swelling and pain of the scapula and avoidance of all arm motion are noted. Conservative therapy including RICE, NSAIDs, analgesics, and a shoulder immobilizer usually suffices. Early progressive rehabilitation emphasizing shoulder motion is essential for shoulder fractures.

MISCELLANEOUS SHOULDER INJURIES

Vascular

Repetitive overhand activity (e.g., pitching) and violent abduction of the shoulder during repeated falls (e.g., wind surfing) have been reported to cause axillary artery occlusion.[20] Symptoms include arm pain, numbness, and severe fatigability. Absent brachial, radial, and ulnar pulses, coolness of the extremity, and a positive arteriogram are diagnostic. Treatment is thrombectomy.

Compression of the posterior circumflex humeral artery and the axillary nerve within the area defined by the teres minor superiorly, glenohumeral joint capsule and humerus laterally, long head of the triceps medially, and fascia and adipose tissue inferiorly has produced the quadrilateral-space syndrome in throwers (Level of Evidence C, nonquantitative review).[48] Overhead activity produces arm paresis, paresthesias, and shoulder pain and point tenderness at the space. Surgical decompression or modification of the overhead activity is recommended.

Primary or "effort" thrombosis of the axillary and subclavian veins is the most common vascular athletic problem and accounts for 2% of total venous thromboses. Vigorous repetitive activity (e.g., weightlifting, rowing, golf, swimming, volleyball, football, baseball) generally precedes such symptoms as arm swelling, pain, paresthesias, and fatigability. Examination reveals nonpitting edema, mottled skin, coolness, prominent superficial veins, and normal arterial and neurologic findings. A palpable axillary cord may be present. Venography confirms the diagnosis. Treatment options include rest, arm elevation, anticoagulation, thrombolytic therapy, and surgical thrombectomy.

Neural

Blunt trauma (e.g., a blow from a hockey stick) or a stretch injury (falling on the point of the shoulder) can damage the spinal accessory nerve (cranial nerve XI), a pure motor nerve (Level of Evidence C, nonquantitative review).[49] Persistent aching of the shoulder girdle, loss of shoulder shrugging, rotary winging of the scapula, and trapezius atrophy follow paralysis. Conservative therapy includes NSAIDs, a sling, and physical therapy, but if recovery is not apparent after 6 weeks exploration and neurolysis should follow.

Damage to the musculocutaneous nerve about the shoulder is rare but has been reported in competitive rowing, weightlifting, and model-airplane flying. Findings are weakness and wasting

of the biceps and brachialis muscles and sensory impairment of the radial aspect of the forearm. The differential diagnosis includes a C5–C6 radiculopathy, brachial plexus injury, or rupture of the bicipital tendon at the elbow. EMG can help differentiate these entities. Conservative therapy, particularly cessation of the inciting activity, yields a favorable prognosis.

Injuries to the long thoracic nerve, causing paralysis of the serratus anterior muscle, have been reported in rope skipping, shooting, throwing and racquet sports, golf, gymnastics, wrestling, and weightlifting. Complaints include aching or burning around the shoulder that may radiate down the arm or over the scapula and weakness of shoulder flexion and abduction. Weakness of shoulder shrug and winging of the scapula are common findings. An EMG is confirmatory and helps distinguish the condition from other scapular and glenohumeral pathologies. The prognosis is good for cases due to excessive or repetitive microtrauma, although cessation of activity, braces, and physical therapy may be required for 1 to 2 years. Prognosis is less favorable for lesions resulting from acute closed trauma.

The axillary nerve is acutely injured in at least 18% of anterior shoulder dislocations or following blunt trauma to the same area in such sports as football or wrestling. Chronic entrapment from repetitive microtrauma with overhead activities (e.g., racquet sports, gymnastics) occurs in association with the quadrilateral-space/tunnel syndrome. The tunnel is formed superiorly by the teres minor muscle; anteriorly by the glenohumeral ligament and subscapularis fascia; inferiorly by the teres major, venous plexus, and adipose tissue; laterally by the humerus and coracobrachialis and triceps tendons; and medially by the long head of the triceps and the inferior glenoid rim. Abduction rotation, internal rotation, and dorsal projection stress the nerve by traction.

Vague pain, paresthesias, and weakness (e.g., inability to screw in an overhead light bulb) can progress to pain and paresthesias radiating to the elbow and dorsal ulnar aspect of the hand, especially with abduction and external rotation (e.g., throwing or tennis serve), tenderness in the region of the posterior outlet of the tunnel (inferomedial humeral head between teres major and minor and long head of triceps), and eventual atrophy of the deltoid and teres minor.

Chronic microtrauma to the upper trunk of the brachial plexus may produce a severe and prolonged disability, particularly to the non-dominant side in hikers, climbers, and campers who carry heavy backpacks (backpacker's palsy). Motor and sensory deficits occur in the distribution of musculocutaneous and radial nerves. Shoulder strengthening exercises without the backpack are important initially. Reduced weight loads are indicated when trekking resumes. The differential diagnosis should include injuries to the posterior cord of the brachial plexus and the thoracodorsal nerve. An EMG is helpful with chronic symptoms. Rest, avoidance of aggravating maneuvers, NSAIDs, and injection therapy suffice in 60 to 80% of cases. Surgical decompression is indicated for refractory cases.

Suprascapular nerve injury is relatively common in athletes with a history of shoulder trauma causing traction on the nerve in the scapular notch (e.g., fall or dislocation). Poorly localized pain made worse while lying on the affected side (posterior subacromial position of the glenohumeral joint with radiation to the elbow), intact sensation, and insidious weakness of external rotation and abduction are typical. Atrophy of the supraspinatus and infraspinatus with normal deltoid function usually occurs. EMG and nerve conduction velocity (NCV) confirm polyphasic motor activity, diminished amplitude, and increased conduction time between Erb's point and muscle. The differential diagnosis should include rotator cuff pathology and backpacker's palsy. Rest, analgesics, and injection therapy usually provide a favorable prognosis. Surgical exploration and decompression are indicated for refractory cases (20%), with 60 to 70% showing improvement.

Other

Contusions (Chapter 34) commonly occur to the bones and muscles of the shoulder in collision/contact sports. A shoulder pointer represents a painful periosteal reaction following a contusive blow of the lateral clavicle or acromion. Myositis ossificans (Chapter 34) can occur 2 to 6 weeks after injury. The differential diagnosis includes cuff strain, AC separation, clavicular fracture, or subluxation/dislocation. Cumulative stress on the acromioclavicular joint due to strength training, repeated contusions, increased training intensity, and the earlier entry of the athlete into certain sports (e.g., weightlifting, football, swimming) can produce atraumatic osteolysis of the distal clavicle. The condition begins with insidious aching in the region of the AC joint several hours after exercise but progresses to earlier pain as the condition worsens. Eventually performance is impaired. Dips, push-ups, and bench pressing

especially aggravate the situation. The AC joint is tender and mildly prominent. Twenty percent of cases are bilateral. Standard radiographs reveal subtle late changes only, but 99mTc scintigraphy is highly sensitive and specific. The differential diagnosis includes other AC and glenohumeral pathology. Conservative therapy (NSAIDs, analgesics, physical therapy) produces a favorable prognosis provided that relative rest utilizing a modified exercise prescription (e.g., cross-training) is mandated. Surgical resection of the distal clavicle is reserved for chronic cases — the end result of continued microtrauma. A snapping scapula is due to the forceful sliding of a prominent superomedial scapular border over the underlying ribs and muscles. Surgical resection may be indicated following a period of conservative therapy.

SUMMARY

Sacrificing structural integrity for functional capacity, the peculiar anatomy and complex motion of the shoulder make it susceptible to acute injury (in collision and contact sports) and, more commonly, to chronic overuse injuries (from throwing, swimming, and racquet sports, for example). The capable sports physician must be familiar with the diagnosis and management of acute shoulder trauma such as dislocation and acromioclavicular sprains. Overuse problems such as the impingement syndrome and rotator cuff inflammation are particularly challenging, as a failure to adequately understand the mechanism of injury and natural history can result in a poor long-term outcome. Early recognition of injury patterns, aggressive non-operative intervention, and an appreciation of therapeutic options are essential to successful resolution and return of the athlete to accustomed levels of performance.

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46 ELBOW*

Joel Shaw, Francis G. O'Connor, and Robert P. Nirschl

ELBOW INJURIES

Epidemiology

As involvement in organized sports has increased in recent years, the frequency of sports-related injuries, including elbow injuries, has similarly increased. Multiple types of elbow injuries occur in athletes, most often due to chronic repetitive forces, elbow overload, poor technique, or improper equipment. These injuries are frequently encountered in throwing and racquet sports, such as tennis and baseball. Several studies have demonstrated that elbow disorders may account for 30 to 40% of injuries in recreational tennis players. Additionally, about 20% of overuse injuries in the young athlete involve the elbow.[1] In one study of young baseball pitchers, over a 1-year period the incidence of elbow injuries requiring treatment was 40%.[2]

Anatomy

The elbow joint is formed by three articulations that provide static and functional stability at this joint to allow for flexion, extension, supination, and pronation. Normal elbow motion is from about 0 to 135 degrees of flexion, and about 75 to 80 degrees of pronation and supination (Figure

* In the second edition, this chapter was authored by Brian C. Halpern.

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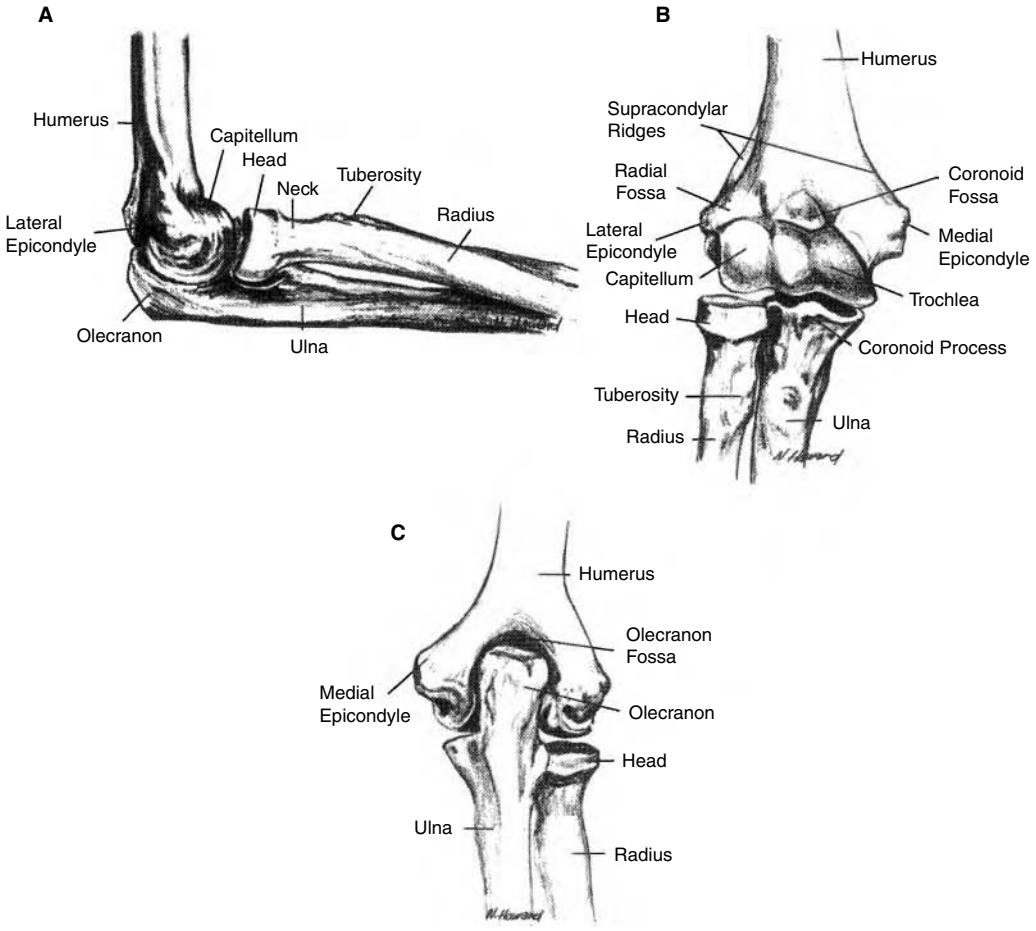

Figure 46.1 Bones of the elbow. (From O'Connor, F.G. et al., in *Handbook of Sports Medicine: A Symptom-Oriented Approach*, 2nd ed., Lillegard, W.A. et al., Eds., Butterworth-Heinemann, Boston, MA, 1999, pp. 141–157. With permission.)

46.1). The bony anatomy includes the two condyles of the humerus, the trochlea and capitellum, which articulate with the proximal ends of the radius and ulna. The trochlea, or medial condyle, is grooved, and articulates with the semilunar notch of the ulna to form the humeroulnar joint. This articulation forms a modified hinge that allows for flexion, extension, and stability. The capitellum, or spherical-shaped lateral condyle, articulates with the radial head to form a combination hinge and pivot joint called the humeroradial joint. This joint allows for flexion, extension, and axial rotation. Finally, the radial head articulates with the lesser sigmoid notch of the ulna to form the radioulnar joint, which also provides axial rotation.[3]

Due to the relative instability of the osseous articulations at the elbow, the ligaments are required to provide about 50% of elbow stability (Figure 46.2). The stronger of the collateral ligaments, the medial collateral ligament (MCL) complex is formed by anterior, posterior, and transverse ligaments. The anterior ligament provides about 70% of valgus stability and remains tight throughout the entire range of elbow flexion, providing the majority of the stability of this ligament. The posterior ligament only becomes tight past 90 degrees of flexion, providing minimal stability, and the transverse ligament does not appear to provide any significant stability. The lateral collateral ligament (LCL) complex provides both rotational and varus elbow stability. This complex originates at the lateral epicondyle and inserts along the annular ligament. Four ligaments form this complex, with the radial collateral ligament providing the majority of varus stability,

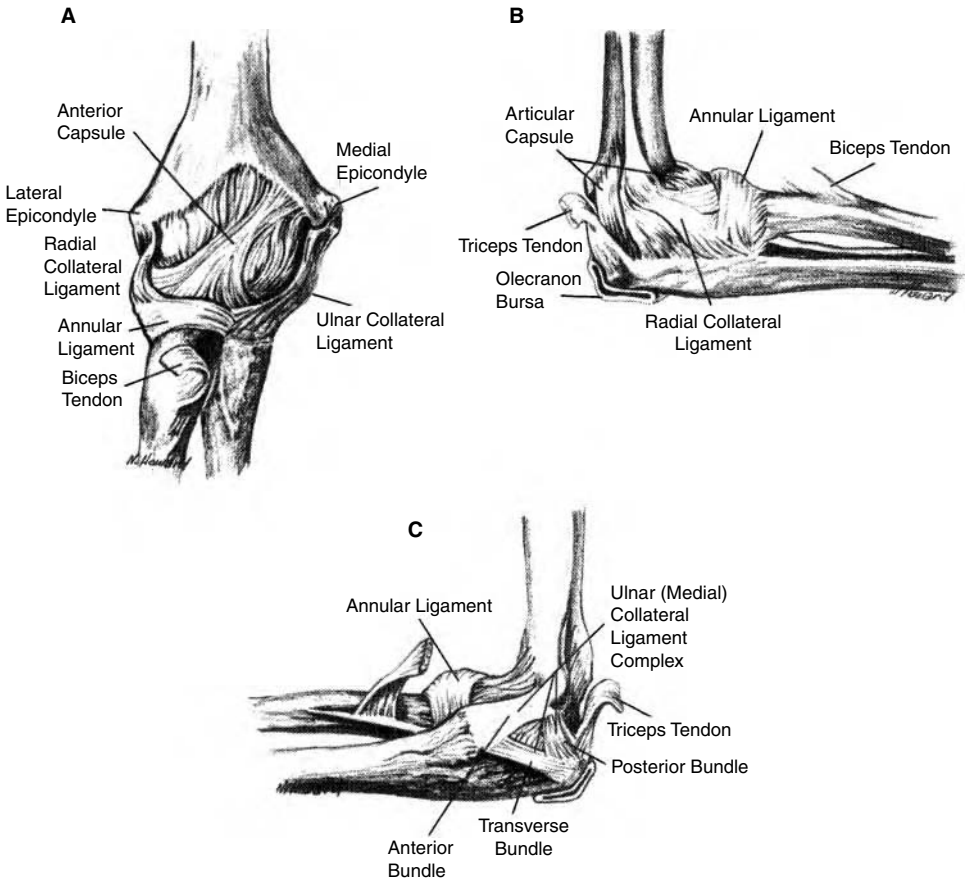

Figure 46.2 Ligaments of the elbow. (From O'Connor, F.G. et al., in *Handbook of Sports Medicine: A Symptom-Oriented Approach*, 2nd ed., Lillegard, W.A. et al., Eds., Butterworth-Heinemann, Boston, MA, 1999, pp. 141–157. With permission.)

remaining tight throughout flexion and extension, and the lateral ulnar collateral ligament providing inferior rotatory stability.[4]

Four major muscle activities are controlled by separate muscle groups passing through the elbow joint. Flexion is performed by the biceps brachii, brachioradialis, and brachialis muscles. Extension is controlled by the triceps and anconeus muscles. Supination is controlled by the supinator and biceps brachii muscles. Pronation involves the pronator quadratus, pronator teres, and flexor carpii radialis muscles. Also, the flexor pronator muscles of the wrist originate from the medial epicondyle, and the wrist extensors originate from the lateral epicondyle.[5]

The three principal nerves that cross the elbow joint complex are the median, ulnar, and radial nerves. The median nerve crosses the anterior elbow medial to the biceps tendon and the brachial artery before entering the pronator teres. The median nerve is responsible for pronation. The ulnar nerve passes posterior to the medial epicondyle in the cubital tunnel before entering the flexor carpi ulnaris. The ulnar nerve provides sensation to the ulnar two digits and motor function to the flexor carpi ulnaris, dorsal and palmar interossei, and the flexor digitorum of the ulnar two digits. The radial nerve emerges from the radial groove behind the humerus to pass anterior to the lateral epicondyle before dividing into superficial (sensory component) and deep (motor component–posterior interosseous) branches. The radial nerve is responsible for the majority of the extensor muscles of the arm.

History

The first step is to determine from the patient whether the injury occurred traumatically or gradually over time. An understanding of the recreational and occupational activities of the patient

TABLE 46.1
Traumatic Injuries

Medial	Lateral	Posterior
Medial epicondylar fracture	Lateral epicondylar fracture	Elbow dislocation
Supracondylar fracture	Capitellum fracture	Olecranon fracture
	Radial head fracture	
	Radial head subluxation	

TABLE 46.2
Overuse Injuries

Medial	Lateral	Posterior	Anterior
Medial epicondylitis	Lateral epicondylitis	Olecranon bursitis	Forearm splints
Ulnar collateral ligament sprain	Radial tunnel syndrome	Osteochondritis dissecans	Median nerve compression syndrome
Ulnar nerve entrapment	Posterior interosseous nerve syndrome	Olecranon impingement syndrome	Pronator syndrome
		Triceps tendinitis	Biceps tendinitis

may help determine the mode of the patient's elbow injury. From this determination, the examiner can then form a working differential diagnosis of common overuse and traumatic disorders that affect the elbow (Tables 46.1 and 46.2). Characteristics of the patient's pain, including timing, duration, intensity, location, character, frequency, and eliciting or relieving factors, will help direct the diagnosis. Relationship to activity will help determine the severity of the injury. Finally, the physician needs to look for loss of function or symptoms suggestive of nerve damage or compression. A history of prior treatment will also aid in establishing a treatment plan.

Physical Exam

Once the differential diagnosis is narrowed down by the history, the definitive diagnosis is often determined by the physical exam. Initial exam involves inspection of the elbow to look for bruising, atrophy, or swelling. Next, the patient should attempt full active range of motion to determine any loss in muscle function, mechanical blockade, or joint contracture. Loss of passive range of motion may indicate intraarticular blockade, dislocation, or fracture. Palpation of several areas is essential. Posterior palpation should include the olecranon, olecranon bursa, and the triceps. Palpation of both the medial and lateral epicondyle may suggest fracture, apophysitis, or epicondylitis. To test for inflammation or injury of the extensor tendons of the wrist (especially the extensor carpi radialis tendon in tennis elbow), resisted extension of the wrist will elicit pain at the lateral epicondyle (Figure 46.3). Similarly, resisted flexion of the wrist will illicit discomfort at the medial epicondyle in patients with inflammation of the flexor tendons of the wrist (Figure 46.4).

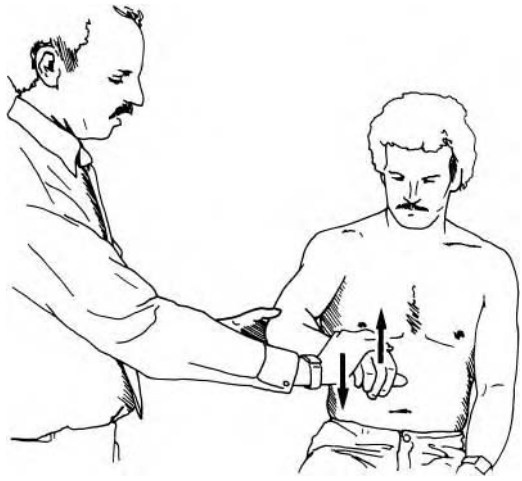

Figure 46.3 Testing for lateral epicondylitis.

Examination for ligamentous stability of the medial and lateral collateral ligaments should be performed. Valgus stress applied to the elbow in both full extension and in 20 degrees of flexion will determine the stability of the medial collateral ligament. Varus stress applied to the elbow will determine any damage to the lateral collateral

Figure 46.4 Testing for medial epicondylitis.

ligament. The examiner should note any laxity, decreased mobility, apprehension, or pain that may be present compared with the uninvolved side. Finally, a neurovascular exam should be performed. A more thorough review of the exam of the elbow can be found in Hoppenfeld[6] or other texts of physical examination.

Diagnostic Studies

Standard x-rays of the elbow include anteroposterior, lateral, olecranon, and oblique views. In the lateral view, the trochlea and capitellum should line up directly, and the ulna should be positioned directly below the radius. The anterior plane of the humerus (anterior humeral line) should intersect the middle third of the capitellum, while the midline of the radial cortex (central radial line) should intersect the anterior humeral line at the middle third of the capitellum. Derangements of these lines indicate an elbow dislocation. In traumatic elbow injuries, the physician should look for evidence of a joint effusion (fat pad sign). Fat pads are triangular-shaped radiolucencies either posterior or anterior to the distal humerus. An anterior fat pad is often visible in a normal elbow and is only considered abnormal when it is significantly raised or perpendicular to the humeral cortex. On the other hand, a posterior fat pad sign is evident of significant intraarticular injury, such as a fracture, dislocation, or an infection with joint effusion. In immature patients with ossification centers still unfused, it is often helpful to get bilateral radiographs for comparison.[7] Other studies may be helpful when specific injuries are being considered. Computed tomography (CT) scans are helpful to look at osteophytes, loose bodies, articular cartilage, and, in some cases, osteochondritis dissecans. Magnetic resonance imaging (MRI) is accurate in diagnosing ligament injuries, osteochondritis dissecans, and other osteochondral injuries. Electromyography (EMG) and nerve conduction studies should be used whenever nerve compression injuries are possible.

MEDIAL ELBOW INJURIES

MEDIAL ELBOW TENDINOSIS (GOLFER'S ELBOW)

I. Epidemiology

In adults, medial elbow tendinosis occurs about one tenth as often as lateral epicondylitis and is seen in sports that involve wrist snap and forearm pronation. Activities using this motion include forehand in tennis, baseball throwing (acceleration), volleyball spike, and the trailing arm in the golf swing. (8)

II. Mechanism of Injury

Tendon degeneration from repeated microtrauma to the flexor-pronator muscle group at its insertion at the medial epicondyle.

III. Anatomy

Enthesopathy of the flexor–pronator muscle group, including the pronator teres, flexor carpi ulnaris, flexor digitorum sublimis, and flexor carpi radialis muscles; involves angiofibroblastic degeneration of this group.

IV. Symptoms

Reports pain over the medial elbow and proximal forearm both during and after activity. Pain will improve with rest between activity, but gradually increases with activity. In severe cases, weakness in the wrist and hand flexion is noted.

V. Signs

Tender over the medial epicondyle and extending 1 to 2 inches distally along the pronator teres and flexor carpi radialis; pain with resisted palmar flexion, pronation, and flexion of the elbow; assess for instability and ulnar nerve pathology (Tinel's sign).

VI. X-Rays

Usually normal but may find extraarticular calcifications.

VII. Special Studies

None indicated.

VIII. Diagnosis
Medial elbow tendinosis (golfer's elbow).

IX. Differential Diagnosis
Osteochondritis dissecans (OCD), medial collateral ligament sprain, capsulitis, radiculopathy, osteoarthritis, ulnar neuritis, flexor–pronator strain.

X. Treatment
A. Initially: Use PRICEMM (prevention/protection, rest, ice, compression, elevation, modalities, and medications; see Chapter 27).
B. Long-term:[9] Injection therapy should only be used after attempting nonsteroidal anti-inflammatory drug (NSAID) treatment with the goal of pain relief to allow an adequate rehabilitation program, including stretching, ultrasound, and flexibility and strength training. Counter-force bracing should be worn. Proper technique should be learned to avoid excessive wrist flexion and pronation. Failure to respond to a 3-month trial of rehabilitation should result in a surgical referral for possible resection and repair of the medial flexor tendon.

XI. Complications
Recurrence.

XII. Prevention
Always warm-up, work on flexibility and strengthening of the flexor–pronator muscle group, control wrist flexion and pronation overuse during sporting activity.

ULNAR (MEDIAL) COLLATERAL LIGAMENT SPRAIN

I. Epidemiology
This sprain occurs primarily in throwing athletes (pitchers and javelin throwers) and in racquet sports.

II. Mechanism of Injury
Overuse microtrauma caused by repetitive valgus overload often due to poor mechanics or, less commonly, valgus trauma to the elbow associated with elbow dislocation.

III. Anatomy
Tear of the ulnar (medial) collateral ligament (usually anterior section of the ligament).

IV. Symptoms
Vague medial elbow pain that becomes worse with activity involving valgus stress and improves with rest.

V. Signs
Palpable tenderness inferior to the medial epicondyle, pain or instability caused by valgus stress testing of the elbow at 20 degrees of flexion, occasionally paresthesias or paresis due to ulnar nerve irritation.

VI. X-Rays
X-rays may indicate traction spurs, loose bodies, or heterotopic ossification of the ulnar collateral ligament. In children, instead of a torn ligament, epiphyseal separation of the medial epicondyle may occur.

VII. Special Studies
Positive gravity stress film in patients with a third-degree MCL sprain. Magnetic resonance imaging can effectively demonstrate partial and complete tears.

VIII. Diagnosis
Medial collateral ligament sprain (Javelin thrower's elbow) with or without instability.

IX. Differential Diagnosis
Medial epicondylar fracture, strain of the flexor pronator muscle group (medial elbow tendinosis), ulnar neuritis, osteoarthritis, medial apophysitis/avulsion in children.

X. Treatment
A. Initially: Rest, ice, and NSAIDS can be effective in partial thickness injury.
B. Long-term: Modify sport technique; wear a counter-force elbow sleeve. Failure to respond to rehab or chronic MCL insufficiency should result in a surgery referral. Ligament rupture requires surgical repair or reconstruction.

XI. Complications
Ulnar nerve entrapment, chronic instability, degenerative arthritis.

XII. Prevention
Adequate warm-up, flexibility and strength training, proper throwing mechanics.

ULNAR NERVE ENTRAPMENT

I. Epidemiology[10]
Ulnar nerve entrapment is the second most common compressive neuropathy in the upper extremity, and most common in the elbow. It occurs most commonly in throwing athletes, but also in those who play racquet sports, skiers, and weightlifters. It may also be related to cubitus valgus deformities.

II. Mechanism of Injury[11]
Direct trauma; compression related to a tight flexor carpi ulnaris muscle from repetitive overarm activities or elbow flexion; traction caused by valgus forces during the acceleration phase of throwing; ulnar nerve subluxation (congenital) with activity related incitement.

III. Anatomy
Inflammation, adhesions, and fibrosis in the cubital tunnel caused by compression (from retinaculum, muscles, tendons, ganglions), traction, impingement (from osteophytes), or subluxation. Zones of the medial epicondylar groove (Nirschl classification):

- Zone 1 — proximal to the medial epicondyle
- Zone 2 — at the level of the medial epicondyle
- Zone 3 — distal to the medial epicondyle

IV. Symptoms
Medial elbow discomfort spreading distally, paresthesias along the path of the ulnar nerve (the fifth finger and lateral aspect of the fourth finger), grip weakness, early hand fatigue, and clumsiness.

V. Signs
Tenderness along the cubital tunnel, positive Tinel's sign, grip and thumb-index pinch weakness; full elbow flexion may result in ulnar nerve subluxation and sudden symptoms.

VI. X-Rays
Usually unremarkable unless symptoms occur associated with arthritic spurs.

VII. Special Studies
Electromyography may demonstrate decreased conduction velocity at zone 3 and motor abnormalities in distal ulnar innervated muscles (about 50%).[10]

VIII. Diagnosis
Ulnar nerve injury, entrapment, compression, cubital tunnel syndrome.

IX. Differential Diagnosis
Medial elbow tendinosis, medial collateral ligament sprain, degenerative joint disease, cervical radiculopathy, thoracic outlet syndrome, other causes of neuropathy.

X. Treatment[11]
A. Initially: Rest, NSAIDs, soft protective pad, and therapeutic exercise for increased strength and flexibility at the elbow are helpful.
B. Long-term: Advise night splinting with elbow at 45 degrees of flexion and/or cortisone injection. Surgical referral for decompression or anterior transposition is recommended if condition is recalcitrant for 3 to 4 months or subluxation, ligamentous instability, or progressive motor or sensory deficits are noted.

XI. Complications
Tardy ulnar neuritis syndrome.

XII. Prevention
Elbow padding and training modification to avoid hyperflexion and valgus stress.

MEDIAL EPICONDYLE FRACTURE

I. Epidemiology[1]
This type of fracture accounts for 10% of elbow injuries in the pediatric population and is most common between 9 and 15 years of age. It is uncommon in younger age groups who have not yet undergone fusion of the medial epicondyle. It is seen in throwing athletes and in children after falling.

II. Mechanism of Injury
Repetitive microtrauma from valgus stress as the flexor muscles apply tension to the medial epicondyle; trauma due to a fall or a direct blow.

III. Anatomy
Partial or complete inferior displacement of the medial epicondyle sometimes associated with medial collateral ligament sprain; 50% occur with partial or complete posterolateral dislocation of the elbow. In some cases, the fragment may become trapped in the elbow joint.

IV. Symptoms
Pain, sometimes severe, over the medial epicondyle.

V. Signs
Tenderness and ecchymosis over the medial epicondyle.

VI. X-Rays
May show separation, asymmetry, increased density of the medial epicondylar apophysis, or fragmentation.

VII. Special Studies
Computed tomography scans provide good visualization of the fracture. The gravity stress test is positive with displaced fractures and associated tears of the MCL. The valgus stress test evaluates for elbow stability; a positive test requires surgical treatment to repair the ligaments and muscles that attach to the medial epicondyle.

VIII. Diagnosis
Medial epicondyle fracture; stress vs. avulsion.

IX. Differential Diagnosis
Medial collateral ligament strain.

X. Treatment
A. Initially: Use rest, ice, NSAIDs, and analgesics.
B. Long-term:[12] Displacement of <2 mm requires initial posterior splint immobilization. Usually within 1 to 2 weeks, when symptoms resolve, the splint may be removed to start active motion exercises. The patient may return to competitive activities when the elbow shows normal range of motion (ROM), strength, and endurance. Displacement of >2 mm or large fragment requires open reduction with internal fixation. ROM exercises may begin about 1 to 2 weeks after surgery with the help of a functional orthosis. Valgus instability, ulnar nerve injury, or entrapment of the epicondyle in the elbow joint necessitates open reduction. In most cases, throwing athletes will not be able to return to throwing for up to 12 weeks.

XI. Complications
Early: hypesthesia, parasthesia, paralysis (ulnar nerve entrapment); *late:* malunion, tardy ulnar palsy, atrophy, chronic instability.

XII. Prevention
Increased instruction and coaching, proper throwing mechanics, proper pregame warm-up followed by post-game icing; encourage and demand reporting of elbow soreness.

LATERAL ELBOW INJURIES

LATERAL ELBOW TENDINOSIS (TENNIS ELBOW)

I. Epidemiology
Tennis elbow is the most common overuse injury of the elbow and is most common in the 40- and 50-year-old age group. It is most often found in racquet sports but may also occur in baseball, swimming, gymnastics, fencing, golfing, and hammer throwing. Frequency includes 75% of club tennis players, 50% of expert females, and 30% of expert males. Causes include poorly fitted equipment, improper technique, and inadequate arm/shoulder fitness.

II. Mechanism of Injury
Repetitive loading of the extensor musculature of the wrist at its insertion onto the lateral epicondyle, causing microtrauma and overload to the tendons; most common with hyperpronation.

III. Anatomy
Degenerative tendinosis primarily of the extensor carpi radialis brevis but also including the extensor communis.

IV. Symptoms
Aching lateral elbow pain that tends to become worse with activity; in some cases, the pain may radiate into the proximal forearm. Initially, pain resolves with rest but may progress to pain that disrupts sleep. Severe cases may cause weakness in the hand and wrist.

V. Signs
Tenderness anterior, medial, and distal to the lateral epicondyle, directly over the extensor

brevis; pain with resisted wrist extension, especially with the elbow in full extension; mild decreased elbow extension.

VI. X-Rays
X-rays are usually normal but are used to rule out other diagnoses. They may demonstrate calcific deposits and epicondylar spurring in up to 20% of cases.

VII. Special Studies
None.

VIII. Diagnosis
Lateral elbow tendinosis/tennis elbow.

IX. Differential Diagnosis
Osteochondritis dissecans, lateral collateral ligament sprain, capsulitis/synovitis, radial tunnel syndrome, osteoarthritis, posterior interosseous nerve entrapment.

X. Treatment[13]
A. Initially: Use RICE, NSAIDs, and activity modification.
B. Long-term: Restore normal ROM; progressive strengthening exercises should include shoulder, arm, and forearm. Initial isometric exercises should be followed by gradual addition of submaximal isotonic exercises. Patient should not have any symptoms with appropriate rehab. Electrical stimulation, iontophoresis, and phonophoresis may also be tried. NSAID medication has been found to promote short-term relief of symptoms (Level of Evidence B, systematic review).[22] Lateral counterforce bracing has been recommended; however, a recent *Cochrane Review* stated that no definitive conclusions can be drawn concerning the effectiveness of orthotic devices for lateral elbow tendinosis (Level of Evidence B, systematic review).[23] Alter sports technique to avoid excessive wrist extension. Steroid injection should be used infrequently and then with the sole purpose of pain relief to allow aggressive rehab. Healing occurs through rehabilitation, not by steroid injection.[14] An evidence-based review did find a short-term benefit at 4 weeks of injectable corticosteroid vs. oral NSAID (Level of Evidence B, systematic review).[24] Surgery is recommended for recalcitrant cases; however, no clinical trials of surgery for lateral elbow pain have been published (Level of Evidence B, systematic review).[25]

XI. Complications
Recurrence.

XII. Prevention
Proper equipment and technique; flexibility and strengthening of forearm, wrist, and finger extension.

RADIAL TUNNEL SYNDROME[11]

I. Epidemiology
Radial tunnel syndrome is seen in weightlifters, bowlers, rowers, swimmers, golfers, and athletes in racquet sports.

II. Mechanism of Injury
Repetitive microtrauma/repetitive pronation; direct trauma to the proximal forearm extensor muscles.

III. Anatomy
Compression of the radial nerve at the radial tunnel; forearm pronation or resisted supination resulting in entrapment of the motor branch of the radial nerve under the fibrous arcade of the supinator muscle.

IV. Symptoms
Lateral elbow pain medial and distal to the lateral epicondyle radiating to the dorsal forearm; aggravation of pain by pronation and supination; paresthesias; finger extensor and supinator weakness; night pain in some cases.

V. Signs
Tenderness over the forearm extensors distal and medial to the lateral epicondyle; finger and wrist extensor weakness; positive Tinel's sign distal and medial to the lateral epicondyle; pain with resisted supination of the forearm or extension of the long finger; sensory defects of the dorsal thumb, index, and long fingers.

VI. X-Rays
Unremarkable.

VII. Special Studies
Nerve conduction studies evaluate delay of radial nerve conduction across the elbow. EMG detects motor dysfunction of the radial nerve innervated distal muscles. Diagnostic lidocaine block of the nerve (1 mL of 1% lidocaine four fingerbreadths distal to the lateral epicondyle) results in improved pain. Conversely, a more proximal injection at the lateral epicondyle does not result in pain relief.[15] Evidence of wrist drop confirms that the injection has reached the posterior branch of the radial nerve.

VIII. Diagnosis
Radial tunnel syndrome.

IX. Differential Diagnosis
Diabetes (polyneuropathic), polyarteritis nodosa, lateral epicondylitis, forearm strain, heavy metal poisoning.

X. Treatment
A. Initially: Use rest, NSAIDs, wrist splinting.
B. Long-term: Comprehensive rehabilitation should focus on flexibility, strength training, and endurance. Surgery for nerve decompression should be considered in cases that do not respond after 3 to 6 months of conservative therapy.

XI. Complications
Chronic disability.

XII. Prevention
None.

POSTERIOR INTEROSSEOUS NERVE SYNDROME[11]

I. Epidemiology
This syndrome occurs in throwing and racquet sports and sometimes in contact sports such as football, hockey, and rugby.

II. Mechanism of Injury
Compression; repetitive supination and pronation, resulting in microtrauma of the interosseous nerve; direct trauma.

III. Anatomy
Entrapment of the posterior interosseous nerve due to swelling by inflammation of the supinator muscles.

IV. Symptoms
Primary symptom is weakness of wrist extension, as opposed to radial tunnel syndrome, where the primary symptom is pain. No complaints of pain or paresthesia (e.g., dysfunction does not include the sensory component of the radial nerve).

V. Signs
Weakness of resisted wrist extension, weakness of finger extension at the MCP joint, radial wrist deviation, normal sensation.

VI. X-Rays
None indicated.

VII. Special Studies
Electromyography and nerve conduction velocity (NCV) may have results similar to those for radial tunnel syndrome.

VIII. Diagnosis
Posterior interosseous nerve syndrome.

IX. Differential Diagnosis
Lateral elbow tendinosis, forearm strain, diabetic neuropathy, polyarteritis nodosa, heavy metal poisoning.

X. Treatment
Essentially the same as for radial tunnel syndrome.

XI. Complications
Tardy posterior interosseous nerve syndrome.

XII. Prevention
None.

RADIAL HEAD FRACTURE

I. Epidemiology
Radial head fracture occurs in any sport involving contact and collision activities.

II. Mechanism of Injury
Fall on an outstretched arm with the elbow extended; compression and shear force on the radial head caused by valgus force on the elbow; axial compressive loading on the radial head.

III. Anatomy
Mason's classification:

- Type I — nondisplaced fractures
- Type II — displaced fractures including impaction, depression, and angulation
- Type III — comminuted fractures

Complex fractures are associated with other injuries such as other fractures, ligamentous injuries, and dislocation.

IV. Symptoms
Generalized lateral elbow pain (acute traumatic onset).

V. Signs
Tenderness at the radial head; edema and swelling of the lateral elbow; pain with flexion or rotation of the elbow/forearm; decreased supination and pronation; decreased elbow flexion and extension.

VI. X-Rays
Consider multiple views to identify fractures at all angles. A posterior pad sign may indicate an intraarticular fracture.

VII. Special Studies
Computed tomography is more detailed for differentiating Mason classification.

VIII. Diagnosis
Radial head fracture.

IX. Differential Diagnosis
Forearm strain, contusion elbow sprain, lateral elbow tendinosis.

X. Treatment
A. Initially: Use RICE, NSAIDs, and analgesics.
B. Long-term: For type I, use a sling, elbow immobilizer, or posterior splint for 1 to 2 weeks with early ROM exercises within 2 to 3 days. Painful hemarthrosis may require aspiration. Type II or greater requires orthopedic consultation. Although nonoperative treatment may be possible, the injury should be evaluated for necessity of surgical treatment (Level of Evidence C, consensus/expert opinion).[4]

XI. Complications
Nonunion, decreased ROM (especially elbow extension or elbow rotation), persistent pain, chronic stiffness, chronic aching, or osteoarthritis; complications are most common in complex fractures.

XII. Prevention
None.

OSTEOCHONDRITIS DISSECANS[16]

I. Epidemiology
Osteochondritis dissecans is most common in males 9 to 15 years of age. The diagnosis is often made 3 to 4 years after symptoms appear, with mean age at diagnosis of 17 to 20 years of age.[17] It usually occurs in baseball players but may also occur in wrestling, football, gymnastics, shooting, shotput, and golf. It can be the end-stage of lateral compression elbow injuries.

II. Mechanism of Injury
Lateral compression injury; repetitive pressure of the radial head against the capitellum resulting in shear force across the joint.

III. Anatomy
Injury of an articular surface caused by separation of cartilage from subchondral bone;[18] it is associated with avascular necrosis, hypertrophy, and fragmentation. The most common location in the elbow is the anterolateral surface of the humeral capitellum.

IV. Symptoms
Pain with activity that improves with rest (90% of patients); swelling and stiffness; occasionally clicking, locking, or a loose body sensation.

V. Signs
Decreased range of motion (55% of patients), palpable crepitus, radiohumeral tenderness.

VI. X-Rays
Depression in the capitellum with radiolucency, loose bodies, hypertrophy of the radial head.

VII. Special Studies
Bone scans are sensitive but may signal another type of injury. CT may be helpful in identifying small, intraarticular loose bodies. MRI is sensitive and specific, especially with normal plain films, and can determine fragment detachment, staging, and early lesions (contusion or stress lesions prior to fragmentation).

VIII. Diagnosis
Osteochondritis dissecans stage _____.

Stage 1 — Intact fragment without displacement of the fragment or fracture of the articular cartilage.
Stage 2 — Partial detachment of the fragment and fracture of the articular cartilage.
Stage 3 — Complete detachment of the fragment resulting in loose bodies.

IX. Differential Diagnosis
Arthirits; Panner's osteochondrosis — ages 7 to 12, usually has normal ROM, has no loose bodies, involves the entire ossification center, and improves well with conservative treatment.

X. Treatment
A. Initially: Stop activity causing injury. Use NSAIDs, ice, stretching, strengthening. Correct technique.
B. Long-term (for persistent pain): Apply splint and initiate physical therapy. Surgery is required in some cases, especially higher stage lesions, to remove loose bodies and to drill in the OCD bed to stimulate repair (Level of Evidence C, expert opinion).[10] Consultation is required for stages 2 and 3, as prognosis may be poor.

XI. Complications
Degenerative arthritis, limited elbow extension, recurrent loose bodies, inability to throw competitively.

XII. Prevention[16,19]
Limit throwing and pitches, correct biomechanics.

POSTERIOR ELBOW INJURIES

OLECRANON BURSITIS

I. Epidemiology
Olecranon bursitis is most commonly seen in dart throwers or in football players (wide receivers, defensive players), wrestlers, and basketball players who land on hard surfaces.

II. Mechanism of Injury
Irritation of the olecranon bursa through direct trauma or repetitive friction.

III. Anatomy
Olecranon bursa inflammation.

IV. Symptoms
Bursal swelling with no or minimal pain.

V. Signs
Fluctuant, nontender swelling. Pain or signs of inflammation require ruling out infection.

VI. X-Rays
Generally unremarkable but may have spur at the tip of the olecranon.

VII. Special Studies
None indicated.

VIII. Diagnosis
Olecranon bursitis.

IX. Differential Diagnosis
Septic bursitis, gout, pseudogout, triceps tendinitis, degenerative joint disease (olecranon fossa).

X. Treatment
Asymptomatic cases are frequently self-limiting. Fluid may be resorbed with resolution of swelling. Initial treatment of symptomatic cases includes RICE, short-term NSAIDs, and an elbow protective pad. In the long term, aspiration may be needed either to relieve discomfort or to send samples for laboratory testing to evaluate for infection or crystals. Steroid injections are rarely needed or helpful (Level of Evidence C, expert opinion).[20]

Consultation: referral for bursectomy for recurrent swelling or catilagenous chips

XI. Complications
Septic bursitis

XII. Prevention
Elbow pads in high-risk sports.

OLECRANON IMPINGEMENT SYNDROME[4]

I. Epidemiology
This syndrome occurs in pitchers, throwers (such as javelin), and those who play racquet sports due to valgus stress and snapping of the elbow at full extension. It is also associated with hyperextension in boxers during jabs.

II. Mechanism of Injury
Impingement of the olecranon in the olecranon fossa from overload during valgus extension.

III. Anatomy
The stress of this repetitive overload causes inflammation and degeneration, resulting in synovitis, osteophytes, and loose bodies.

IV. Symptoms
Posterior elbow pain and clicking, greatest in extension; locking and catching consistent with mechanical blockade.

V. Signs
Tenderness and swelling of the posterior elbow; pain worse with extension; crepitation and limitation of full extension revealed by ROM; pain with combination of extension and valgus stress; subtle instability in some cases.

VI. X-Rays
X-rays often reveal osteophytes, loose bodies, and olecranon hypertrophy; occasionally may find olecranon tip fracture.

VII. Special Studies
Magnetic resonance imaging or CT arthrography may be helpful in severe cases.

VIII. Diagnosis
Olecranon impingement syndrome.

IX. Differential Diagnosis
Olecranon stress fracture, olecranon bursitis, triceps tendinosis.

X. Treatment
A. Initially: Use RICE and NSAIDs.
B. Long-term: Initiate physical therapy with therapeutic exercises to increase the strength and flexibility of the elbow musculature.

Consultation: surgical referral required for persistent pain, loose bodies, or mechanical blockade for arthroscopic excision

XI. Complications
Olecranon stress fracture, arthritis, persistent loss of extension.

XII. Prevention
Correct throwing biomechanics (e.g., improve follow-through); improve flexibility and strengthening; do adequate warm-up.

TRICEPS TENDINITIS/TENDINOSIS

I. Epidemiology
Triceps tendinitis/tendinosis occurs in sports requiring repetitive elbow extension such as weightlifting, boxing, gymnastics, throwing, and racquet sports.

II. Mechanism of Injury
Repetitive extension of the elbow resulting in triceps tendon overload.

III. Anatomy
Degeneration/inflammation of the triceps tendon and muscle at the olecranon insertion.

IV. Symptoms
Posterior elbow pain, worse with forceful or full extension.

V. Signs
Tenderness at or above the insertion of the triceps tendon at the olecranon, increased pain and decreased range of motion with resisted extension; a palpable depression just proximal to the olecranon with severe pain and swelling is suggestive of triceps tendon rupture.

VI. X-Rays
Usually unremarkable, but may have calcification in the tendon.

VII. Special Studies
None indicated.

VIII. Diagnosis
Triceps tendinitis/tendinosis.

IX. Differential Diagnosis
Olecranon bursitis, degenerative joint disease, olecranon fracture, triceps strain, olecranon impingement syndrome.

X. Treatment
A. Initially: Use RICE and NSAIDs
B. Long-term: Physical therapy is directed at triceps strengthening and increased flexibility. Exercise techniques should be modified. Steroid injections should be avoided due to the risk of tendon rupture. Condition usually responds to conservative therapy.

Consultation: refractory cases

XI. Complications
Loose body, chondromalacia, tendon rupture (rare but requires immediate surgical referral; occurs most often with a history of falling on an outstretched arm, and exam shows severe pain, swelling, and a palpable depression).

XII. Prevention
Correct throwing mechanics, adequate warm-up, improved stretching and strengthening.

OLECRANON FRACTURE
I. Epidemiology
Olecranon fracture occurs in contact/collision sports and is often associated with coexistent posterior elbow dislocation.

II. Mechanism of Injury
Direct blow to the posterior elbow; fall on an outstretched arm, especially associated with a posterior elbow dislocation.

III. Anatomy
Important to classify non-displaced vs. displaced fracture.

IV. Symptoms
Posterior elbow pain, swelling due to intraarticular component, ulnar nerve neuropathy.

V. Signs
Decreased range of motion, especially decreased extension; posterior elbow effusion; tenderness with palpation of olecranon.

VI. X-Rays
X-rays reveal fractures of the olecranon (displaced vs. non-displaced).

VII. Special Studies
None indicated

VIII. Diagnosis
Olecranon fracture (displaced vs. non-displaced)

IX. Differential Diagnosis
Posterior elbow dislocation, olecranon bursitis, triceps tendinitis/tendinosis.

X. Treatment

- Non-displaced fractures — Use immobilization in a posterior splint with the elbow flexed at 90°. Range of motion exercises should include supination/pronation exercises at 2 to 3 days; flexion/extension exercises can begin at 2 weeks. Immobilization may be discontinued at 6 weeks, which is the time when fractures usually achieve adequate union for ADLs (activities of daily living).
- Displaced fractures — Referral to an orthopedic surgeon is required for reduction and internal fixation, followed by early motion to avoid elbow stiffness. Small fractures (involving less than 30% of the olecranon) may be treated with excision and reattachment of the triceps tendon.

XI. Complications
Residual stiffness/restricted ROM, nonunion, arthritis.

XII. Prevention
None.

ANTERIOR ELBOW INJURIES
MEDIAN NERVE COMPRESSION SYNDROME/PRONATOR SYNDROME
I. Epidemiology
This syndrome is seen in sports that involve repetitive pronation and a forceful grip, such as throwing, racquet sports, weight lifting, archery, rowing, gymnastics, and arm wrestling. It is believed that hypertrophy of the pronator teres in these type of athletes may predispose them to compression.[11]

II. Mechanism of Injury
Repetitive microtrauma, direct trauma.

III. Anatomy
The syndrome is the result of compressive entrapment of the median nerve just distal to the elbow. Three possible areas exist for compression of the nerve as it crosses the cubital fossa of the elbow joint: the head of the pronator teres, the bicipital aponeurosis, and the fibrous arch of the flexor digitorum superficialis.

IV. Symptoms
Insidious onset of pain in the antecubital fossa or proximal forearm that is related to activity (e.g., pronation) and radiates; numbness and parasthesias may occur in the forearm and hand. Loss of index and long finger flexion occurs in chronic cases.

V. Signs
Sensory deficit in the forearm and hand (median nerve distribution); positive Tinel sign in the proximal forearm; pain provoked by resisted forearm pronation; papal sign (compromise of index and long-finger active flexion) in chronic cases; atrophy and weakness of the thenar intrinsic muscles in chronic cases.

VI. X-Rays
X-rays are normal in most cases.

VII. Special Studies
Electromyography is only positive in chronic cases. Nerve conduction velocity tests often show delayed conduction in the upper forearm and are also helpful for ruling out carpal tunnel syndrome. Negative EMG and NCV testing does not rule out pronator syndrome.

VIII. Diagnosis
Median nerve compression syndrome, pronator teres syndrome, anterior interosseous syndrome.

IX. Differential Diagnosis
Biceps tendinosis/strain, stenosing tenosynovitis, forearm splints.

X. Treatment
A. Initially: Recommend relative rest, NSAIDs, and immobilization.
B. Long-term: Rehabilitative exercise program goals should be to increase flexibility and to balance the strength of the forearm, focusing on the forearm pronators. Surgical decompression is indicated for cases that fail to respond to conservative treatment after 3 to 6 months, show axonal damage by EMG, or show persistent loss of function.[11]

Consultation: refractory cases, as discussed above

XI. Complications
Loss of motor function.

XII. Prevention
None.

BICEPS TENDINITIS/TENDINOSIS

I. Epidemiology
Biceps tendinitis/tendinosis is found in athletes performing activities that require repetitive biceps function, such as weightlifters, boxers, bowlers, and gymnasts.

II. Mechanism of Injury
Repetitive flexion of the elbow resulting in biceps overload.

III. Anatomy
Overuse microtrauma of the biceps tendon resulting in degeneration/inflammation of the biceps tendon and muscle.

IV. Symptoms
Anterior elbow pain, weakness of elbow flexion.

V. Signs
Tenderness with direct palpation of the biceps tendon, increased pain with resisted elbow flexion and supination.

VI. X-Rays
X-rays are usually normal but may show calcification of the biceps tendon.

VII. Special Studies
None indicated.

VIII. Diagnosis
Biceps tendinitis/tendinosis.

IX. Differential Diagnosis
Median nerve compression syndrome, forearm splints, biceps strain.

X. Treatment[13]
A. Initially: Use RICE and NSAIDs.
B. Long-term: Rehabilitation should be directed at increasing the strength and flexibility of the biceps mechanism. Modify technique and training patterns to prevent repeat and continuing injuries. Avoid steroid injections due to risk of tendon rupture.

Consultation: refractory cases that may suggest musculotendinous tear or nerve entrapment

XI. Complications
Tendon rupture — distal biceps tendon rupture necessitates immediate referral for surgical repair. Symptoms that suggest complete rupture include sudden and severe pain, marked swelling, and a provoking event associated with strenuous flexion against a heavy load.

XII. Prevention
Improve the strength and flexibility of the biceps brachii, correct sports specific mechanics, do adequate warm-up.

FOREARM SPLINTS
I. Epidemiology
Most often seen in young gymnasts, especially males, due to training on the pommel horse.

II. Mechanism of Injury
Forearm overuse, especially repetitive wrist extension.

III. Anatomy
Believed to be similar to shin splints. One hypothesis is that forearm splints are caused by strain and overuse of the extensor carpi ulnaris at its origin.[21]

IV. Symptoms
Lateral forearm pain that is worse with precipitating activity.

V. Signs
Exacerbation of forearm pain with resisted wrist extension.

VI. X-Rays
Normal.

VII. Special Studies
None.

VIII. Diagnosis
Forearm splints/wrist splints.

IX. Differential Diagnosis
Forearm strain, median nerve compression syndrome, stress fracture of the ulna.

X. Treatment
A. Initially: Ice before and after practice. Taping or counter-force bracing of the forearm or wrist at the site of pain will promote an earlier return to activity.
B. Long-term: Forearm strengthening should focus on the wrist extensors. Improving technique and avoiding overtraining will prevent further injury.

XI. Complications
None.

XII. Prevention
Improve technique and prevent overtraining.

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47 FOREARM INJURIES

James L. Lord

INTRODUCTION

Injuries to the forearm occur at an estimated frequency of 5 to 7% of all sports injuries. The forearm is vulnerable to injury because it is often the leading body part in contact or collision injuries such as those between players in football and soccer, between stick and forearm in ice hockey and lacrosse, or from any fall onto the outstretched arm. This chapter discusses some of the important anatomic considerations required to adequately evaluate a variety of forearm injuries, and it discusses a logical approach to some of the more common osseous and nonosseous injuries to the forearm likely to be encountered by the primary care sports physician. In addition, general treatment protocols for several of the more common injuries that lend themselves to conservative nonoperative management are presented.

ANATOMY OVERVIEW

The radius and ulna represent the infrastructure of the forearm. They are approximately equal in length and articulate proximally and distally at their respective proximal and distal radioulnar joints, which are true synovial joints having a joint capsule and synovial fluid. They are bound proximally by the capsule of the elbow and the annular ligament. Distally, the radioulnar joint is supported by the capsule of the wrist joint, by the anterior and posterior radioulnar ligaments, and by the triangular fibrocartilaginous complex (TFCC). The shafts of the radius and ulna are joined by the interosseous membrane, an important supporting structure in the forearm, with fibers originating proximally on the radius and inserting distally and obliquely on the ulna. In addition, the radius and ulna are joined by the supinator, pronator teres, and pronator quadratus muscles, which play an important role as stabilizers of the radial and ulnar shafts and which are responsible for significant bony displacement associated with some forearm fractures. The ulna acts primarily as a fixed strut. The radius rotates around this strut during supination and pronation.

When evaluating the bony anatomy of the distal forearm radiographically, three important anatomic measurements require careful consideration when determining the alignment of a distal radius fracture (Figure 47.1). The numbers to remember are 11, 23, and 12. On a lateral radiograph of the distal forearm, volar tilt of the radius is approximately 11°. On the AP view of the distal forearm, the radius inclines approximately 23°, and the tip of the radial styloid is approximately 12 mm distal to the distal ulnar articular surface. Injuries involving the radial styloid may also involve the median nerve and the extensor pollicis longus tendon.

The soft tissues of the forearm are arranged in two main compartments, both of which are subdivided into superficial and deep compartments. One compartment is dorsal to the bones and interosseous membrane and one is volar to these structures. The dorsal compartment contains the extensor and supinator muscles of the wrist, hand, and forearm, and the volar compartment contains the flexor and pronator muscles of the wrist, hand, and forearm. Nerves and blood vessels in each compartment lie between the deep and superficial muscle layers.

CLINICAL APPROACH TO FOREARM INJURIES

The essential component of a detailed history is first to determine as accurately as possible the mechanism of injury. This helps to narrow the

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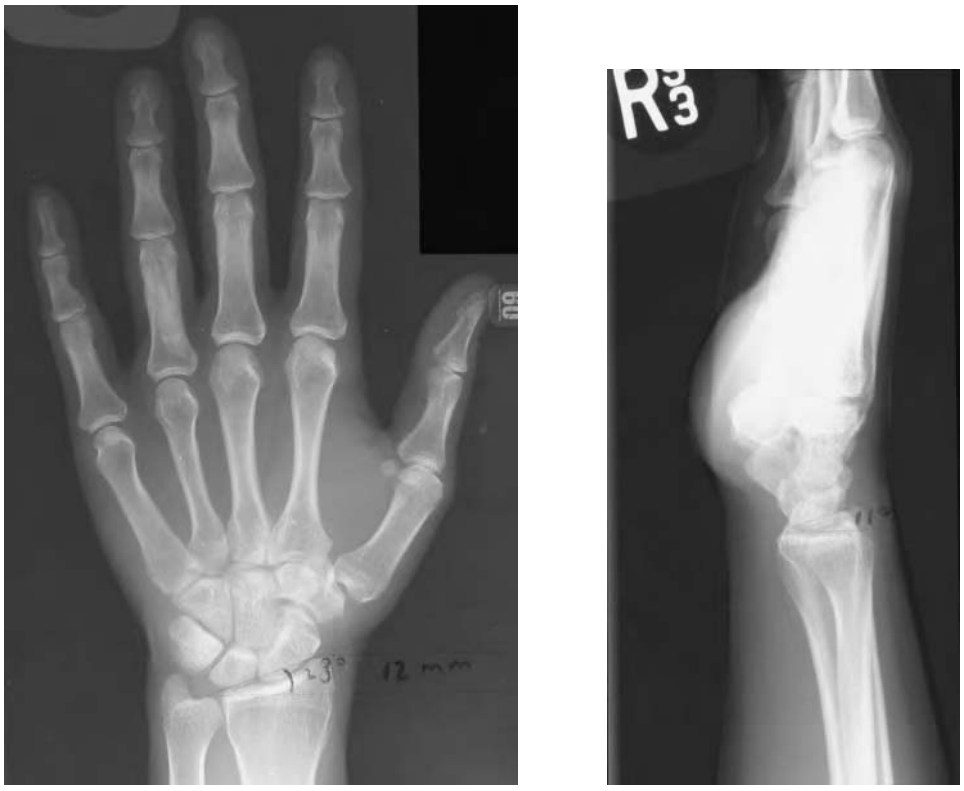

Figure 47.1 (left) Anteroposterior view: normal average radial length and radial inclination; (right) lateral view: normal volar tilt of the distal radius.

diagnostic possibilities (Table 47.1). Axial loading injuries from falls on the outstretched hand/extended wrist (FOOSH injuries) most commonly result in either a fracture or a fracture/dislocation. A direct blow or contusive injury produces fractures of the radius and/or ulna, subperiosteal hematomas of the radius or ulna, or muscle contusions. Acute muscle strains with or without rupture of the muscle fibers may be produced by tension-overload injuries such as those seen with rock climbing. Repetitive microtrauma (overuse injuries) results in injuries such as delayed-onset muscle soreness (DOMS), tenosynovitis syndromes such as DeQuervain's disease, intersection syndrome, or isolated extensor or flexor tendinopathies. Various nerve entrapment syndromes such as those involving the median nerve, the anterior interosseous nerve (AIN), or the posterior interosseous nerve (PIN) are also probably examples of overuse type injuries.

Examination of the forearm yields the best results when performed in rote sequence from exam to exam. The suspected injury site should first be inspected and palpated carefully, then both inspection and palpation should be extended along the entire length of the radius and ulna, including wrist and elbow, and then over the dorsal and volar surfaces of the soft tissues of the forearm. Comparison with the opposite, presumably uninjured, forearm is frequently desirable. Many injuries of the forearm can result in an acute compartment syndrome that represents a true orthopedic emergency; therefore, a careful neurovascular exam distal to the injury site should be done next in the sequence. Three parameters to assess are color, motion, and sensation (remember CMS). The classic signs of an acute compartment syndrome are the 5 P's (Table 47.2): pain disproportionate to the injury, pallor, pulselessness, parasthesias, and paralysis. For suspected overuse injuries or musculotendinous strains, resisted flexion and extension and resisted radial and ulnar deviation can help to localize and define the nature of the injury. Tinel testing (tapping over a superficial nerve) coupled with appropriate sensorimotor testing in the hand and wrist can aid in the diagnosis of nerve entrapment syndromes.

TABLE 47.1
Differential Diagnosis by Mechanism of Injury

Fall on the Outstretched Hand (FOOSH)
Fracture
Fracture/dislocation
Contusion
Fracture
Subperiosteal hematoma
Muscle contusion
Tension Overload
Muscle strain
Rupture of the flexor digitorum superficialis muscle
Overuse (Repetitive Microtrauma)
Delayed-onset muscle soreness (DOMS)
Tendinopathies
DeQuervain's tenosynovitis
Intersection syndrome
Tendinitis of various isolated tendons
Nerve entrapment syndromes
Anterior interosseous nerve (AIN) syndrome
Posterior interosseous nerve (PIN) syndrome

TABLE 47.2
Signs of Acute Compartment Syndrome (The 5 P's)

Pain disproportionate to the injury
Pallor
Pulselessness
Parasthesias
Paralysis

Initial imaging for all forearm injuries includes AP and lateral plain radiographs and should include both wrist and elbow. Computed tomography imaging is usually unnecessary. Magnetic resonance imaging or ultrasound imaging may be necessary to evaluate for a suspected interosseous membrane injury.

OSSEOUS INJURIES

A detailed discussion of the management of complicated fractures or fracture/dislocations, especially those requiring manipulation or surgical intervention, is beyond the scope of this chapter (refer to appropriate texts on fracture management).[1–8] Five fractures that frequently fall within the scope of management for primary care sports physicians are:

- Torus (or buckle) fractures with less than 10° of angulation in children
- Extraarticular distal radius fractures that do not require closed or open reduction
- Some nightstick fractures of the ulna
- Type I radial head fractures
- Type II radial head fractures with no mechanical block

Distal radius fractures represent a substantial number of pediatric forearm fractures. The torus or buckle fracture of the radius (Figure 47.2) by definition is a plastic deformity of the bone that occurs in young children between the diaphysis and the metaphysis of the radial shaft. These fractures do not display a tendency to displace, and they are not synonymous with a greenstick fracture, in which separation of the cortex on the tension side of the fracture has occurred (Figure 47.3). A greenstick fracture can displace with time. One prospective randomized control study, reported in the *Journal of Bone and Joint Surgery* (British edition) in 2001, showed that an appropriate soft forearm splint with rigid stays applied for 3 weeks was as effective as a rigid cast in treating torus fractures of the forearm in children, and no difference in complications was found (Level of Evidence A, randomized controlled trial).[9]

Nondisplaced, nonangulated, extraarticular distal radius fractures (Figure 47.4) in adolescents and young adults can be treated with a double sugar-tong splint or a long-arm cast for 4 to 6 weeks (Level of Evidence C, consensus expert opinion).[6] Obtain repeat X-rays on the first follow-up visit at 1 to 2 weeks and then again at 4 to 6 weeks.

A nightstick fracture of the ulnar shaft falls within the scope of primary care if it is not comminuted or displaced and if apposition of the fracture fragments ≥ 50% and angulation < 10°.[4] Initial treatment can be accomplished with a long-arm posterior splint for 7 to 10 days or until pronation and supination are no longer painful. Then, either a short-arm posterior splint or a functional brace may be used for 4 to 6 weeks (Level of Evidence A, prospective randomized controlled trial).[15] The patient should have repeat X-rays every week for 3 weeks to check for

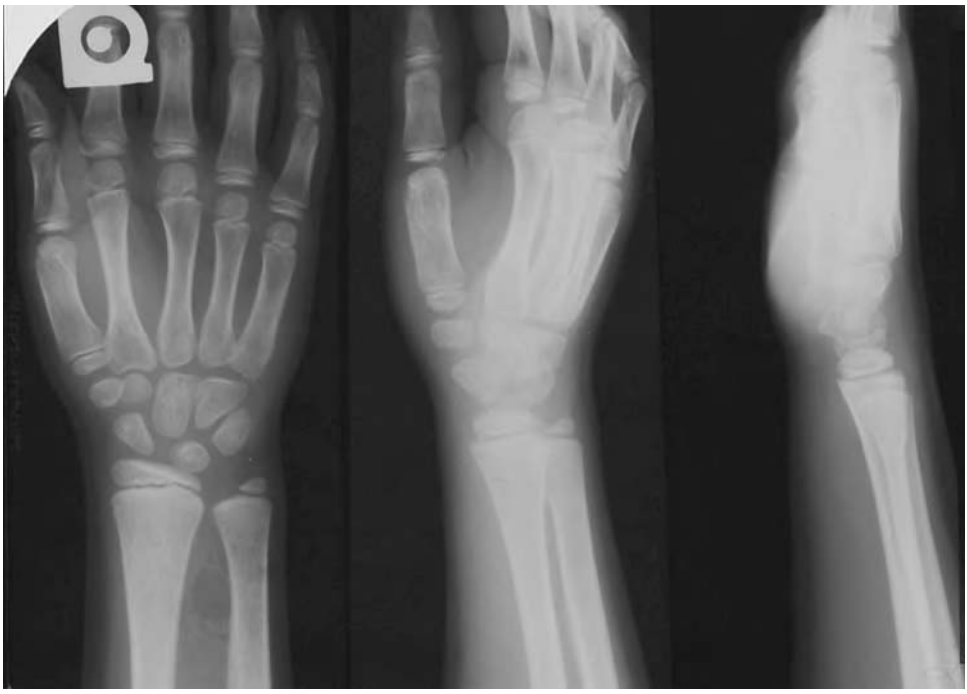

Figure 47.2 Torus fracture of distal radius.

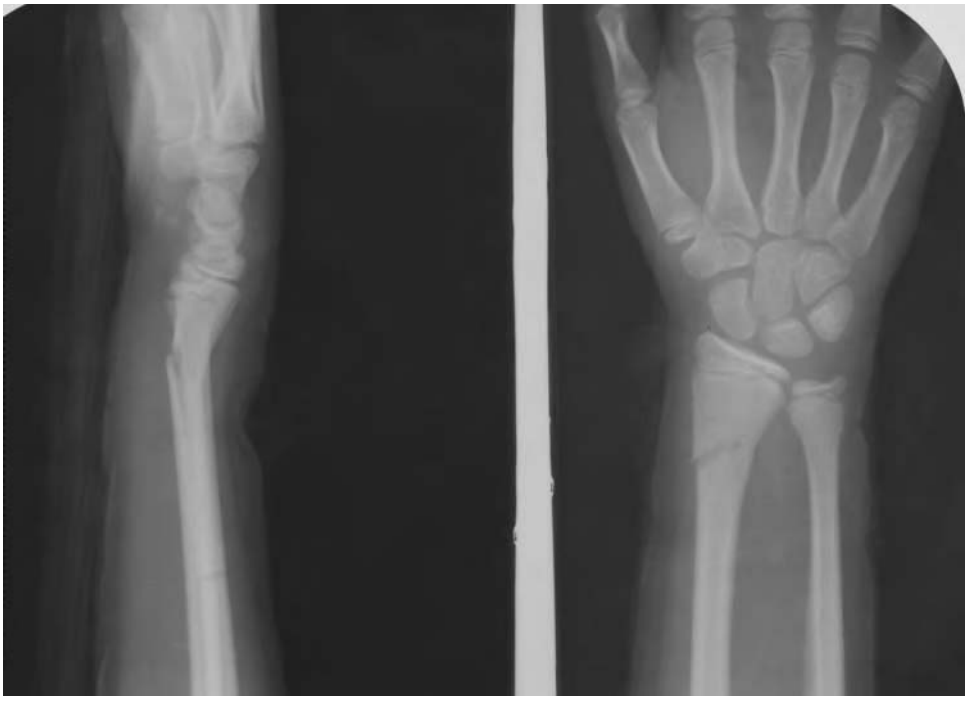

Figure 47.3 Greenstick fracture of radius, torus fracture of ulna.

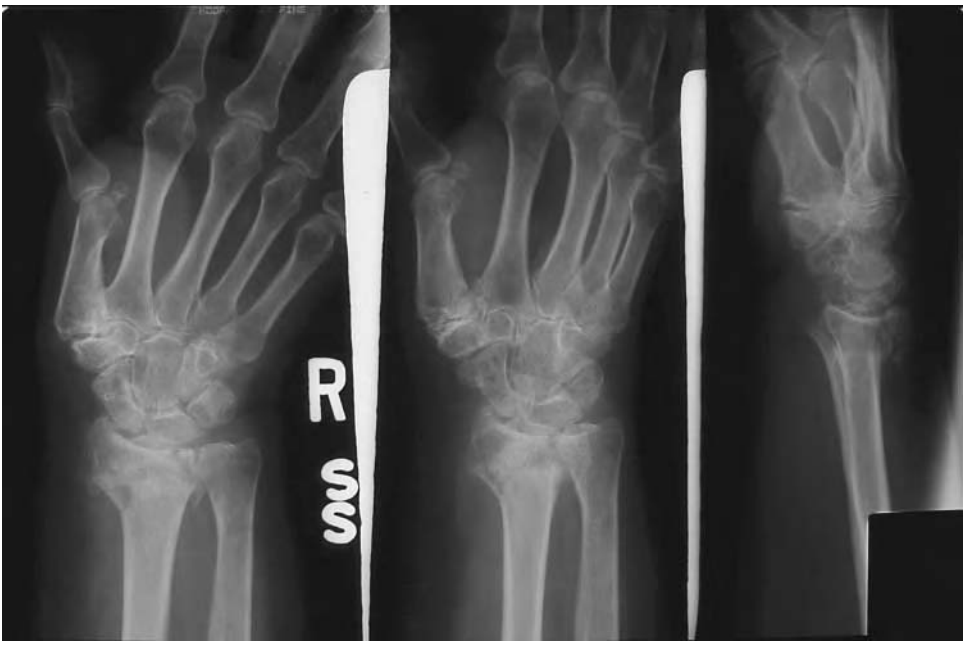

Figure 47.4 Extraarticular nondisplaced distal radial fracture.

TABLE 47.3
Radial Head Fracture Types

Type I	Nondisplaced; no mechanical block
Type II	Displaced >2 mm; mechanical block, or more than a marginal lip of the radial head
Type III	Comminuted

alignment and subsequent displacement and then at 6 weeks to document bony union.

Radial head fractures are classified as one of three types based on total joint surface involvement and degree of displacement (Table 47.3, Figure 47.5). Treatment of type I fractures (Figure 47.6) and type II fractures with no evidence of mechanical block involves use of a sling for comfort only, usually 1 to 2 weeks (Level of Evidence C, consensus expert opinion).[5] The key to management of both types is early mobilization to prevent loss of full range of motion of the elbow. Physical therapy may be necessary later in the course of recovery if acceptable progress in achieving full range of motion, especially the last 10° of extension, is lacking. The decision to treat or refer type II fractures is dependent upon detecting any mechanical block to motion at the outset. This can be done at the time of the first exam by aspirating the hemarthrosis and instilling local anesthetic into the joint for pain relief in order to fully assess range of motion.

Radial head fractures in the skeletally immature population pose a different set of problems.[8] Salter–Harris I and II type fractures are more common and complication rates higher. Referral to an orthopedist is generally recommended for these.

At times, primary care sports physicians will find themselves in situations where they are faced with fractures that are obviously more complex than those discussed above. Examples of common potentially complicated fractures involving the distal radius are *Colles' fracture*, with dorsal angulation and shortening of the distal fragment (resulting in the classic "dinner fork" deformity); *Smith's fracture* (or reverse Colles' fracture), with volar angulation and shortening (the "garden spade" deformity); and *Barton's fracture*, a fracture/dislocation of the distal radius. With this injury, either the dorsal or volar aspect of the distal radial articular surface is sheared off, resulting in a dislocation of the radiocarpal joint.

Combined radius and ulna fractures may present with very impressive angulation and/or displacement of the fracture fragments due to muscular contraction forces of either the dorsal or volar compartment muscles (see Figure 47.5).

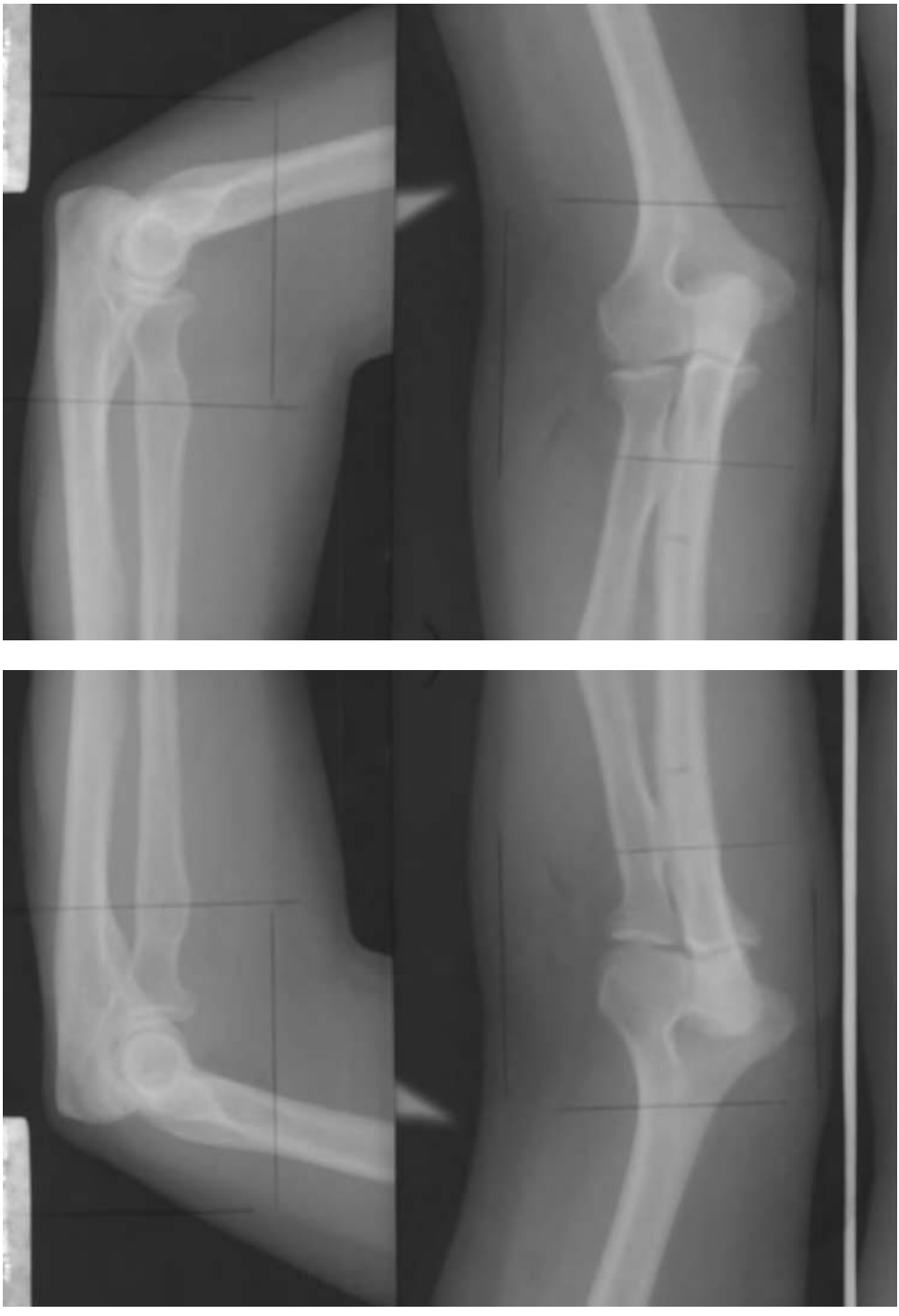

Figure 47.5 Type I radial head fracture.

Fracture/dislocations involving the shaft of either the radius or ulna include *Galeazzi's fractures*, with fracture of the radial shaft and concomitant dislocation of the distal radioulnar joint (DRUJ), and *Monteggia's fractures*, which are shaft fractures combined with dislocation of the radial head.

An initial clinical evaluation as detailed previously with careful attention to the neurovascular exam is still very appropriate in these situations.

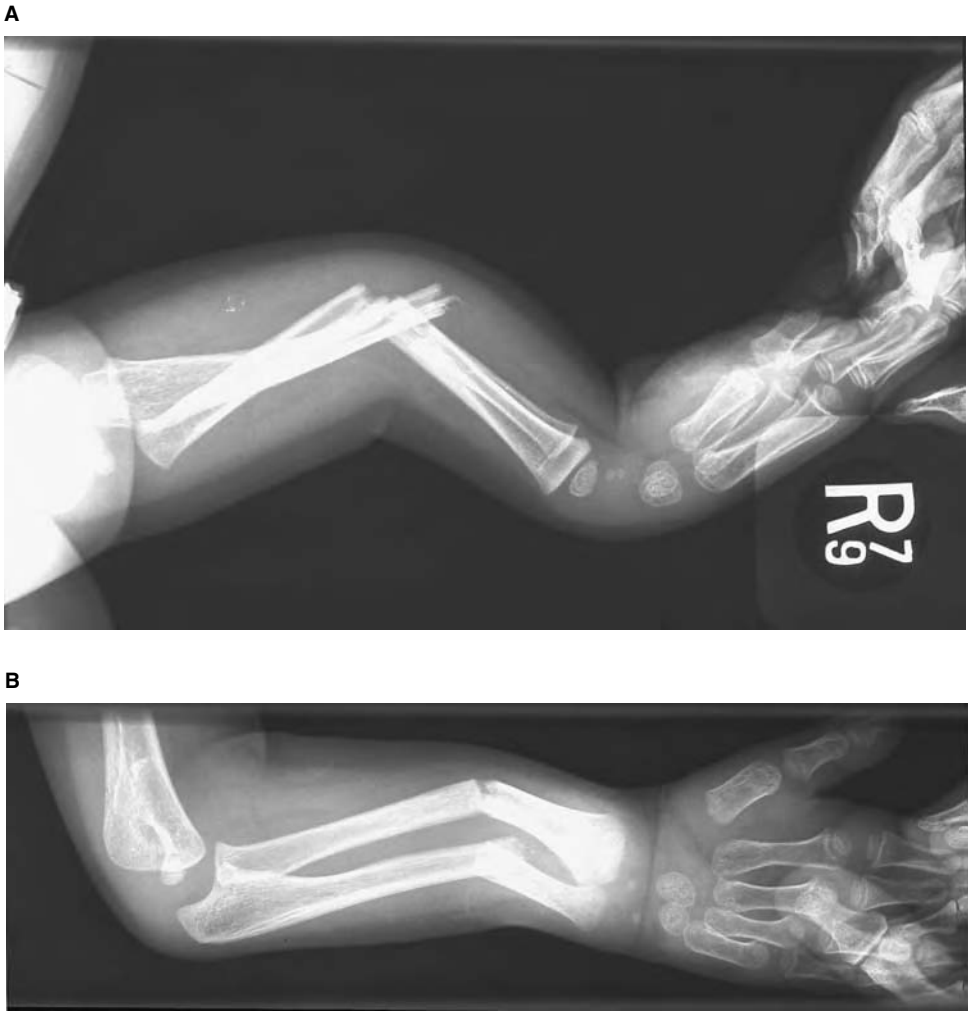

Figure 47.6 (A) Anteroposterior view and (B) lateral view: transverse fractures with radial angulation and shortening of both radius and ulna.

Most of these complex injuries will require some sort of closed reduction or surgical intervention followed by prolonged immobilization along with close follow-up to watch for any serious complications (Level of Evidence C, consensus expert opinion). Early consultation and referral to an orthopedist frequently comprise the most prudent course.

SOFT-TISSUE INJURIES[10–11]

Contusive injuries to the forearm may result in a fracture as noted above; however, with a less violent blow or with a blow padded by some piece of protective equipment, the resulting injury may only involve soft tissues such as a subperiosteal hematoma of either the radius or ulna or a forearm muscle contusion. The exam may reveal either a localized painful mass of the bone with or without superficial ecchymosis or a localized area of tenderness within the involved muscle with or without a mass or bruising. Standard musculoskeletal first-aid modalities including relative rest, ice, compression and elevation (RICE) are generally adequate and very little else is needed as long as appropriate systematic recovery occurs within a reasonable timeframe[12] (Level of Evidence C, consensus opinion). If recovery is slower than expected, more extensive evaluation for other injuries using magnetic resonance imaging or ultrasound may be necessary.[13] Muscles and tendons in the forearm are also subject to repetitive microtrauma from overuse injuries.

Tenosynovitis syndromes such as DeQuervain's disease and intersection syndrome are discussed in Chapter 48.

Delayed-onset muscle soreness frequently follows prolonged isometric contraction of the forearm muscles with weight training or power lifting as well as in predominantly upper extremity sports such as crew. Treatment mainly involves tincture of time and alteration of the training regimen. Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) may be needed for temporary relief of pain during the acute phase.

Nerve entrapment syndromes originating in the forearm are less common overuse injuries. Three syndromes that are worth noting are the *pronator syndrome*, in which the median nerve can become entrapped at one of two sites, either between or just distal to the two heads of the pronator teres muscle in the proximal forearm; the *anterior interosseous nerve syndrome*, which involves a branch of the median nerve; and the *posterior interosseous nerve syndrome*, which involves a branch of the radial nerve. Tinel testing and a sensorimotor exam of the wrist and hand can help with the diagnosis. Treatment consists of various combinations of wrist splinting, relative rest, or surgical decompression.

Tension-overload syndromes are most likely to occur in sports such as rock climbing.[13] One such injury is a rupture or partial rupture of the flexor digitorum superficialis muscle that occurs with difficult hand-hold maneuvers. Treatment is usually conservative, consisting of relative rest and minor pain-control medications if necessary.

SUMMARY

Key to accurate diagnosis and management of forearm injuries is a systematic approach that consists of taking an injury-based history, a thorough exam of the entire length of the forearm (including the wrist and the elbow), a careful neurovascular exam, and appropriate imaging studies. Primary care sports physicians, depending upon their degree of individual expertise, are capable of managing many of these injuries, but, at the very least, they need to have an in-depth understanding of the common forearm injuries and know which ones are appropriate for them to manage and which ones require consultation and/or referral. A favorable outcome is the number one goal no matter how that is accomplished.

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48 HAND AND WRIST INJURIES

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THE HAND

Its high physical profile and large range of functional activities make the hand susceptible to a wide variety of sports-related trauma. Regardless of the patient's level of proficiency or age, all hand injuries require a careful medical evaluation, including appropriate x-rays. Even common abrasions and contusions should not be quickly dismissed as trivial. Local swelling, ecchymosis, and tenderness may indicate a more serious injury to underlying structures. Important treatment is delayed without early accurate assessment. A relatively simple problem requiring a splint or cast for a successful outcome may progress to a chronic condition requiring complicated surgery which may fail to restore normal strength and mobility. Nonetheless, the competent sports physician should be able to appropriately manage the majority of hand injuries.

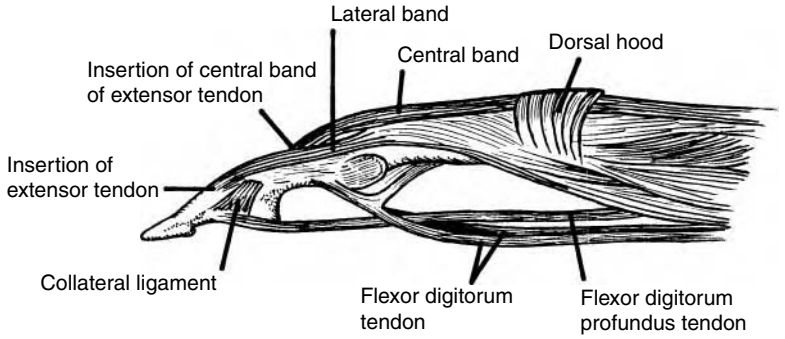

Figure 48.1 Lateral view of the flexor and extensor mechanism of the finger.

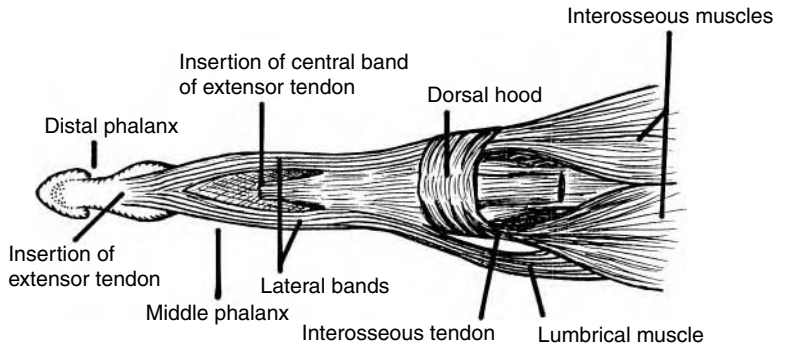

Figure 48.2 Dorsal view of the extensor mechanism of the finger.

Epidemiology

The true incidence of hand injuries in athletes is difficult to determine because many athletes either ignore the injury or continue to play with a splint. The injuries are therefore considered insignificant and often are not recorded in statistical records. McGrew et al.[1] reported that 11% of 1286 injuries in a primary care sports medicine setting were to the fingers and hand.

Anatomy

The nine finger flexors and the median nerve pass into the hand through the carpal tunnel beneath the transverse carpal ligament. The five deep flexors pass through the superficialis tendons to the distal phalanx of each finger and thumb, and four superficial flexors insert on the middle phalanx of each finger (Figure 48.1). The flexors pass beneath a series of ligaments between the distal palmar crease and the distal interphalangeal joint. These annular ligaments create "pulleys" (A1–A5 and C1–C3) and prevent the tendons from bowstringing. Tendon repair in this so-called surgical "no-man's land" is therefore often unrewarding due to adhesions that form between the lacerated tendon ends and these ligaments.

The volar plate is a thickened portion of the joint capsule on the volar aspect and is a static stabilizer against hyperextension forces. Disruption will lead to chronic instabilities if it is not allowed to heal in its proper anatomic position. The collateral ligaments afford medial and lateral stability and are at maximal tautness at 70° flexion for the metacarpophalangeal (MCP), 30° for the proximal interphalangeal (PIP), and 15° for the distal interphalangeal (DIP). When the hand is immobilized for any length of time, the collateral ligaments will contract; therefore, these joints should be immobilized, flexed at the above angles (collaterals maximally lengthened) to prevent permanent contractures. When describing these collateral for orientation, one can describe them as the radial or ulnar collateral.

The extensor tendons enter the dorsal extensor hood at the distal end of the metacarpals (Figure 48.2). Distal to the sagittal fibers of the hood enter the transverse and oblique fibers from the interosseous and lumbrical muscles. These blend

into the sides of the extensor hood over the proximal phalanx and flex the MCP joint by pulling on the extensor hood. The central portion of this extensor complex travels distally over the PIP joint and inserts into the proximal dorsal aspect of the middle phalanx to extend the PIP. The lateral bands continue radially and ulnarly and insert onto the proximal dorsal aspect of the distal phalanx to extend the DIP.

Pathogenesis
Most injuries to the hand resulting in significant disability are caused by direct or indirect trauma with subsequent disruption of a static (capsule, volar plate, collateral ligament, or bony) or dynamic (tendon, dorsal hood) stabilizer. Intricate interactions of the bones, ligaments, capsule, and tendons allow for exquisite function yet stability in the hand. Disruption of any of these may lead to significant functional disability. Central tendon rupture leads to "boutonnière" deformity, volar plate disruption to dorsal instability or a "swan-neck" deformity, angulated fractures to decreased grip function, etc.

Symptoms
Clues to significant injuries can be any of the following: severe deformity on initial examination, incomplete reduction of the initial deformity, loss of normal alignment or joint motion (including catching, locking, or crepitus) during active or passive flexion and extension, an increase of more than 20% of the normal range of motion in any plane during passive testing, or pain or muscle contraction significant enough to inhibit testing.

Signs
With the wrist in mild dorsiflexion, the resting posture of the fingers form a gentle flexed curve. Any extension deviation from the normal semiflexed position suggests a flexor tendon injury. The arm is then pronated with the wrist slightly flexed. An excessive drop may suggest an extensor tendon or acute nerve (radial or C6 root) injury. The patient is then observed while actively flexing and extending the fingers, thumb, and wrist, looking for deviation from normal. With gentle flexion, all of the fingers should generally point towards the scaphoid tuberosity. Any deviation suggests a rotational deformity of a phalanx or metacarpal. Active flexion and extension may be hampered by a disrupted tendon, ligament, or fracture, and an unstable joint may sublux. Sensation can be tested by asking the athlete to distinguish between the feel of the rough edge of a quarter and a smooth-edged coin or by two-point discrimination (normal, 5 mm or less). Specific findings are discussed under individual injuries.

Radiographs/Laboratory
X-rays should be taken for any injury with bony tenderness, angulation, rotation, or instability. Posterior-anterior (PA), lateral, and oblique views will generally suffice. Laboratory studies are usually not indicated except for suspected septic joints, for which a complete blood count and erythrocyte sedimentation rate are useful.

Special Studies
Coronal magnetic resonance imaging (MRI) may be useful in demonstrating tendon rupture.

Treatment
Treatment principles for stable injuries of the hand are generally the same for most hand injuries and can be anticipated to take from 1 to 6 weeks. Rest and elevation are advised until the soft-tissue swelling is stable. Early active range of motion for non-injured joints is essential to minimize contractures and encourage use of the flexor and extensor mechanisms. Splinting should be minimized as soon as the acute reactive stage of inflammation has subsided by changing over to minimal, removable, protective splinting and encouraging active range of motion as soon as comfort allows. Return to full function is allowed as symptoms subside. Open reduction and internal fixation (ORIF) is generally considered if a fracture involves more than 25 to 30% of an articular surface, or if significant angulation, displacement, or rotation is present that cannot be reduced and maintained by closed manipulation and immobilization.

SPRAINS AND STRAINS

MALLET FINGER
See Figure 48.3.

I. Epidemiology
Originally described in baseball but can occur in any activity where the finger is subject to "jamming"; it is frequently missed initially, with subsequent deformity and medico-legal consequences.

II. Mechanism of Injury/Pathogenesis
Axial load against an actively extending finger.

III. Anatomy
Can result in a dorsal bony avulsion or a grade III (complete disruption) injury to the extensor digitorum tendon.

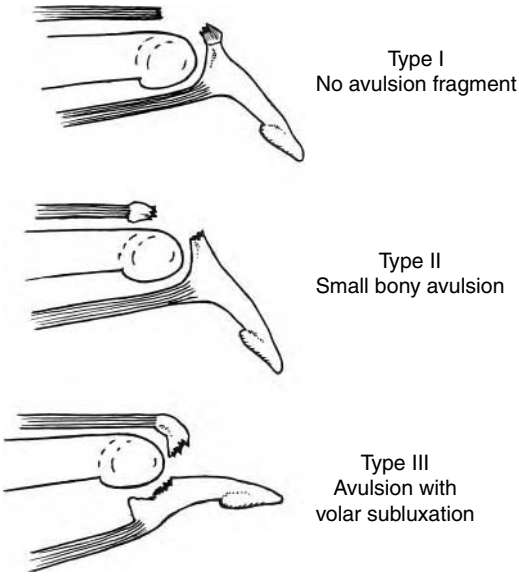

Figure 48.3 Types of rupture of extensor digitorum tendon at its insertion onto the proximal dorsal distal phalanx ("mallet" or "baseball" finger).

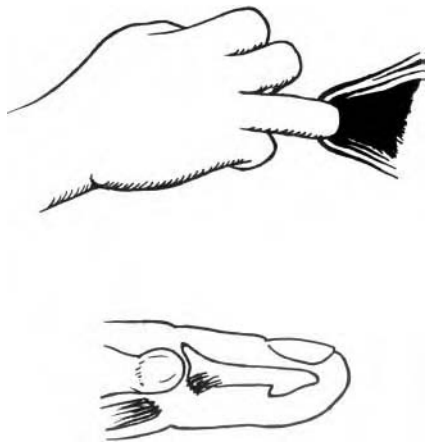

Figure 48.4 Mechanism for rupture of the flexor digitorum profundus tendon ("jersey finger").

IV. Symptoms
Pain at the distal interphalangeal joint.

V. Signs
Inability to extend the isolated DIP, tenderness over the dorsal proximal aspect of the distal phalanx.

VI. X-Rays/Laboratory
X-rays may show a bony avulsion from the dorsal proximal distal phalanx (seen in approximately 20 to 30% of cases).

VII. Special Studies
None indicated.

VIII. Diagnosis
"Mallet" or "baseball" finger.

IX. Differential Diagnosis
"Jammed" finger, DIP dislocation, collateral ligament tear, fracture.

X. Treatment
A. Initially: Treat with RICE protocol and analgesics as needed.
B. Long-term: No avulsion fracture — splint DIP fully extended for 6 to 8 weeks straight and an additional 6 to 8 weeks if engaged in athletic activity; the splint should be worn 100% of the time and, when removed for personal care, the DIP should be maintained in extension. Bony avulsion — if less than 30% of joint space is involved, dorsal finger splint should be worn in full extension for 4 weeks; if more than 30% of joint space is involved, refer for possible ORIF.

XI. Complications
Permanent DIP extensor lag if untreated; watch for pressure necrosis from splint.

XII. Prevention
Proper technique

JERSEY FINGER (FOOTBALL FINGER)
See Figure 48.4.

I. Epidemiology
Common in football, rugby, martial arts, or any sport where grabbing an opponent's clothing can occur.

II. Mechanism of Injury/Pathogenesis
Forced extension of the distal phalanx while actively flexing the DIP (e.g., athlete grabbing onto a jersey).

III. Anatomy[2]
Caused by either a grade III tear or a bony avulsion fracture of the flexor digitorum profundus tendon. An avulsion fracture of the volar lip of the distal phalanx limits retraction and enables repair by ORIF. A large avulsion fragment will limit retraction to the A4 pulley (type III). Pure tendon avulsions may retract to the PIP at the A2 pulley (type II) or palm (type III). If retracted to the palm, the blood supply via the vincula brevum and longum is compromised. Buscemi and Page[3] recommend the addition of a type VI injury in cases of tendon avulsion with a separate intraarticular fracture of the distal phalanx.

IV. Symptoms
Pain and swelling at the DIP.

V. Signs
Unable to flex the isolated DIP with localized tenderness at the level of retraction of the avulsed segment. The flexor digitorum profundus is examined by holding the PIP straight and asking the athlete to flex the DIP. The superficialis is tested by holding the MCP straight and asking the athlete to flex the PIP.

VI. X-Rays/Laboratory
Posterior-anterior, lateral, and oblique views will document an avulsed fragment and may help localize the level of retraction.

VII. Special Studies
None indicated.

VIII. Diagnosis
Jersey/football finger.

IX. Differential Diagnosis
"Jammed" DIP, collateral ligament tear, fracture of distal phalanx.

X. Treatment[2,4]
A. Initially: Treatment is RICE and analgesics as needed.
B. Long-term: Refer for surgical repair — Type I within 1 week; types II and III, within 3 weeks (beware that Type II may convert to Type III). If referral is delayed, reconstruction may be required rather than repair.

XI. Complications
Permanent loss of flexion, or reconstruction if diagnosis made late.

fragment (Level of Evidence B, pro-

XII. Prevention
Proper technique.

PIP VOLAR PLATE RUPTURE (WITHOUT DISLOCATION)

I. Epidemiology
Common in volleyball, football, or any sport where the finger is subject to hyperextension.

II. Mechanism of Injury/Pathogenesis
Hyperextension injury causing the distal portion of the volar plate to rupture from its attachment to the middle phalanx.

III. Anatomy
Loss of the volar stabilizing force of the PIP allows the extensor tendon to gradually pull the PIP into a hyperextension deformity (reverse or pseudoboutonnière) over time.

IV. Symptoms
Pain and swelling at the PIP joint.

V. Signs
The PIP is in varying degrees of hyperextension with maximal tenderness over the volar aspect of the PIP. With active extension and flexion, the hyperextended PIP often "locks" in the extended position with an inability to initiate flexion. DIP range of motion (ROM) is normal.

VI. X-Rays/Laboratory
Posterior-anterior, lateral, and oblique views may show an avulsion fragment at the base of the middle phalanx.

VII. Special Studies
None indicated.

VIII. Diagnosis
Proximal interphalangeal volar plate rupture.

IX. Differential Diagnosis
"Jammed" finger, collateral ligament sprain, flexor tendon avulsion with retraction to PIP, PIP dislocation, fracture.

X. Treatment
A. Initially: Treat with RICE and analgesics as needed. Early active mobilization provides the best results. In a study of 190 patients, excellent or good results were achieved in 98% with early mobilization, independent of size or displacement of the volar fracture spective cohort study).[5]

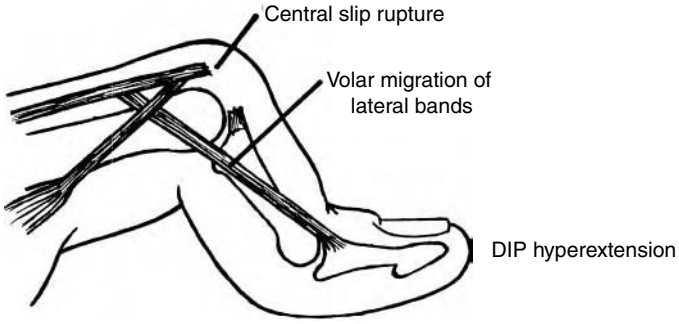

Figure 48.5 Central slip of the extensor mechanism rupture. Note the volar migration of the lateral bands leading to flexion of the PIP and extension of the DIP ("boutonnière" deformity).

B. Long-term: Wear dynamic extension block splint for 3 weeks with the PIP at 20 to 30° of flexion (allows full flexion, but limits extension to 30°), then buddy tape, followed by progressive resistance exercises (PREs). Surgery is indicated for PIP contracture.

XI. Complications
Hyperextension deformity (reverse boutonnière) if untreated.

XII. Prevention
Proper technique.

CENTRAL SLIP AVULSION
See Figure 48.5.

I. Epidemiology
Contact and collision sports.

II. Mechanism of Injury/Pathogenesis
Volar-directed force on the middle phalanx against a semiflexed finger attempting to extend; volar dislocation of PIP.

III. Anatomy
Disruption of the central slip of the extensor digitorum communis tendon over the PIP joint allows for migration of the lateral bands volar to the axis of the joint.

IV. Symptoms
Pain and swelling over the PIP joint.

V. Signs
The PIP is in 15 to 30° of flexion with point tenderness over the dorsal lip of the middle phalanx. Early on the patient may have limited extension, appearing stronger if extension is tested at 0° and weaker if tested at 30°. Later, an inability to actively extend the PIP with the DIP hyperextended ("boutonnière" deformity) occurs.

VI. X-Rays/Laboratory
X-rays may show an avulsion fracture at the dorsal base of the middle phalanx.

VII. Special Studies
None indicated.

VIII. Diagnosis
Central slip avulsion.

IX. Differential Diagnosis
"Jammed" finger, collateral ligament tear, volar plate disruption, dislocation, fracture.

X. Treatment[2,4]
A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: PIP is splinted in full extension for 4 to 5 weeks and further protected during sporting activity for an additional 6 to 8 weeks. While splinted, the DIP should be allowed to flex to help relocate the lateral bands back to their normal position. If an avulsion fragment involves >1/3 of the joint, the patient should be referred for possible ORIF.

XI. Complications
"Boutonnière" deformity if untreated.

XII. Prevention
Proper technique.

COLLATERAL LIGAMENT TEARS

I. Epidemiology
Collision and contact sports.

II. Mechanism of Injury/Pathogenesis
Result from valgus or varus stress to the PIP, DIP, or MCP.

III. Anatomy
Causes partial or complete tears of the ulnar or radial collateral ligaments.

IV. Symptoms
Pain and swelling at the involved joint.

V. Signs
Laxity with valgus or varus stress; the joint may be stable or unstable with active flexion and extension.

VI. X-Rays/Laboratory
X-rays may show avulsion fracture from capsular insertion.

VII. Special Studies
None indicated.

VIII. Diagnosis
Collateral ligament sprain/tear.

IX. Differential Diagnosis
Volar plate injury, flexor or extensor tendon strain, fracture, dislocation.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed
B. Long-term: Stable with active ROM — buddy tape finger-to-finger adjacent to side of injury for 3 weeks. Unstable with active ROM or obvious angulation — refer for possible surgical repair.

XI. Complications
Unstable joint if complete tear is inadequately treated.

XII. Prevention
Proper technique.

TRAUMATIC DISLOCATION OF THE EXTENSOR HOOD

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis[2,4]
Caused by direct blow to the flexed MCP or by flexion and ulnar deviation force across the MCP.

III. Anatomy
Disruption of the sagittal fibers (usually radial), allowing the extensor tendon to sublux off the apex of the MCP into the valley between the MC heads.

IV. Symptoms
Pain and swelling over the dorsum of the MCP.

V. Signs
The MCP is tender dorsally with inability to actively extend the MCP joint from a flexed position; after passive extension of the joint, the patient is able to maintain extension.

VI. X-Rays
Usually normal.

VII. Special Studies
None indicated.

VIII. Diagnosis
Traumatic dislocation of the extensor hood.

IX. Differential Diagnosis
Dislocated MCP, fracture, collateral ligament sprain.

X. Treatment
A. Initially: treated with RICE, splinting, and analgesics as needed
B. Long-term: Splint the MCP in full extension with the PIP free for 4 weeks. Active ROM exercises are begun at 4 weeks with the splint worn at all other times. Splint is discontinued at 8 weeks. Injuries >10 days old or those failing conservative management should be referred for possible surgical correction (Level of Evidence C, consensus/expert opinion).[6]

XI. Complications
If unrecognized early, condition will require surgical correction.

XII. Prevention
Proper technique.

Figure 48.6 Ulnar collateral ligament rupture, type 4. The free distal end of the ulnar collateral ligament folds back on itself under or over the adductor aponeurosis, which prevents healing.

"SKIER'S" OR "GAMEKEEPER'S" THUMB[7–9]

See Figure 48.6.

I. Epidemiology

Initially observed in Scottish gamekeepers, who would kill hares by placing the thumb and index fingers around the neck to hyperextend it. This abduction force resulted in gradual weakening of the ulnar collateral ligament and joint instability over time. The injury is now almost exclusively traumatically induced in sports such as skiing.

II. Mechanism of Injury/Pathogenesis

Hyperabduction of the thumb MCP joint (e.g., the classic fall on a ski pole, causing the thumb to be held while the remainder of the hand plunges into the snow).

III. Anatomy[8]

The four classes of ulnar collateral ligament (UCL) sprain are:

Type 1 — avulsion fracture, no displacement
Type 2 — avulsion fracture, displaced
Type 3 — torn ligament, stable in flexion
Type 4 — torn ligament, unstable in flexion

IV. Symptoms

Pain over the UCL area, weak and painful pinch.

V. X-Rays

X-rays should be performed prior to any stress testing in order to reveal any avulsion fragment; if an avulsion fracture is evident, stress testing should not be done. A displaced fracture with >2 mm displacement or a rotated fracture should be considered type 4.

VI. Signs[8]

Tenderness and swelling over the ulnar aspect of the thumb MCP. Stress testing should be performed if there is no evidence of avulsion fracture and is performed as follows:

1. The area is anesthetized with either a local block or median and radial nerve blocks at the wrist.
2. The thumb metacarpal is stabilized with one hand and a valgus stress placed on the MCP with the MCP in full flexion. Testing is done in full flexion because with extension or slight flexion the normally taut volar plate gives the MCP stability.
3. Complete rupture (type 4) is suspected if angulation is 15° greater than the normal thumb or absolute angulation is greater than 35°.
4. Angulation less than described above is type 3 and considered stable.

VII. Special Studies

Arthrogram will show extravasation of dye in complete ruptures. MRI may be useful to diagnose Stener lesions or grade III tears (Level of Evidence C, consensus/expert opinion). Sensitivities of 96% and specificity of 95% have been reported.[10]

VIII. Diagnosis

Skier's/gamekeeper's thumb, type _____.

IX. Differential Diagnosis

Dislocation, volar plate injury, sprain, fracture.

X. Treatment[7–9]

A. Initially: Treat with RICE and analgesics as needed.
B. Long-term:
   Type 1 — thumb spica cast with MCP in full extension for 4 weeks
   Type 2 — refer for ORIF
   Type 3 — thumb spica cast with IP free and MCP flexed 20° for 3 weeks
   Type 4 — refer for ORIF

XI. Complications
Loss of effective thumb apposition if inadequately treated.

XII. Prevention
Ski pole with sword-type grip, proper technique.

FRACTURES

DISTAL PHALANGEAL FRACTURE

I. Epidemiology
Collision and contact sports.

II. Mechanism of Injury/Pathogenesis
Compression or crush force.

III. Anatomy
The fracture fragments of the distal phalangeal (DP) are rarely displaced but result in extensive soft-tissue damage, including subungual hematomas and nail-bed injury.

IV. Symptoms
Pain and swelling of the distal phalanx.

V. Signs
A subungual hematoma is often the primary source of pain; look for nail matrix disruption.

VI. X-Rays
X-rays generally show non-displaced fracture fragments.

VII. Special Studies
None indicated.

VIII. Diagnosis
Fracture of the distal phalanx.

IX. Differential Diagnosis
Soft-tissue injury or isolated subungual hematoma.

X. Treatment[11]
A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: A protective splint is used for symptomatic relief rather than fracture stabilization. Subungual hematomas are drained. A disrupted nail matrix is repaired with 6-0 absorbable suture and the wound treated as a compound fracture.

XI. Complications
A disrupted nail matrix interposed between fracture fragments can cause a permanent nail deformity or delayed union of the fracture.

XII. Prevention
Protective equipment, proper technique.

MIDDLE PHALANGEAL FRACTURE

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Direct trauma or twisting

III. Anatomy
The fractures tend to be transverse, are generally angulated palmarly, and are often unstable due to the opposing forces of the dorsal extensor tendon and the flexor digitorum superficialis (FDS) palmarly.

IV. Symptoms
Pain and swelling.

V. Signs
Tenderness and swelling over middle phalanx with varying degrees of deformity; always check for rotational deformity.

VI. X-Rays
X-rays show degree of angulation or displacement.

VII. Special Studies
None indicated.

VIII. Diagnosis
Middle phalangeal fracture, stable (reduction achieved and maintained) or unstable.

IX. Differential Diagnosis
Soft-tissue injury.

X. Treatment[11]
A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: Stable, non-displaced, and non-angulated — buddy tape; use thermoplastic splint for sport activity. Stable, minimal angulation — immobilize with the MCP flexed 70°, PIP flexed 45°, and DIP free, buddy taping

to control rotation. Remove splint in 3 to 4 weeks and begin ROM exercises; the splint should be worn during sporting activities for an additional 9 to 10 weeks. Unstable (displaced, angulated, unable to hold reduction) — refer to orthopedics.

XI. Complications
Rotational or angular deformity if mistreated initially.

XII. Prevention
Proper technique, protective equipment.

PIP FRACTURE DISLOCATION

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Caused by an axial load on a semi-flexed finger.

III. Anatomy
The middle phalanx shears dorsally, impacting the palmar articular surface of the middle phalanx with the condyles of the proximal phalanx.

IV. Symptoms
Pain and swelling over the PIP.

V. Signs
Subtle dorsal prominence over the PIP, with localized tenderness.

VI. X-Rays
X-ray shows impaction fracture; the proximal aspect of the middle phalanx is dorsally displaced and the palmar articular fragment is maintained palmarly.

VII. Special Studies
None indicated.

VIII. Diagnosis
Fracture dislocation of the PIP, small volar impaction fracture.

IX. Differential Diagnosis
Volar plate avulsion fracture, PIP dislocation, proximal or middle phalangeal fracture, sprain.

X. Treatment[4,12]
A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: Small fragment without dislocation — buddy tape. Larger fragment but <40% of articular surface — closed reduction followed by extension block splint with PIP in 30 to 60° of flexion for 3 weeks. Fragment >40% of articular surface — surgical consultation for ORIF.

XI. Complications
Post-traumatic arthritis.

XII. Prevention
Proper technique, protective equipment.

PROXIMAL PHALANGEAL FRACTURES

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Direct trauma.

III. Anatomy
Most fractures are spiral or oblique, tend to shorten, and are therefore unstable. These are difficult to treat due to the compact anatomy of extensor hood, lateral bands, and flexor tendons surrounding it. Scarring or displacement disturbs the tendon balance.

IV. Symptoms
Pain, swelling, variable degree of deformity.

V. Signs
Tenderness, swelling, varying degrees of shortening, angulation, or rotation, tuning fork test.

VI. X-Rays
X-rays will reveal type and extent of the injury.

VII. Special Studies
None indicated.

VIII. Diagnosis
Proximal phalangeal fracture; type: spiral, oblique, or transverse.

IX. Differential Diagnosis
Soft-tissue injury.

X. Treatment[4,11]

A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: Fracture stability and early ROM are critical to successful treatment. For stable fractures, immobilize with wrist in slight extension, MCP in 70° flexion, PIP and DIP joints free, buddy taping to adjacent finger for 3 to 4 weeks. Buddy taping is then continued until asymptomatic. For unstable (usual type) fractures, refer to orthopedics.

XI. Complications

Scarring, angulation, or shortening, resulting in poor tendon function.

XII. Prevention

Proper technique, protective equipment.

METACARPAL FRACTURES

I. Epidemiology

Fifth MC neck fractures common in boxing and martial arts; can occur in any contact or collision sport.

II. Mechanism of Injury/Pathogenesis

Direct trauma from either an axial load or compressive forces.

III. Anatomy

Neck fractures tend to angulate volarly to a significant degree. Shaft fractures are frequently stabilized by the intrinsic muscles; 60% are angulated >40°, and angulation up to 70° does not result in significant functional disability. The second and third digits are necessary for a power grip and much less angulation (<10°) is acceptable here than for the fourth and fifth.

IV. Symptoms

Pain and swelling.

V. Signs

Varying degrees of angular or rotational deformity.

VI. X-Rays

X-rays confirm fracture and degree of angulation/displacement.

VII. Special Studies

None indicated.

VIII. Diagnosis

Metacarpal neck or shaft fracture.

IX. Differential Diagnosis

Soft-tissue injury, MCP dislocation.

X. Treatment[11]

A. Initially: treated with RICE and analgesics as needed.
B. Long-term:
   1. For the reduction technique, the fracture site is anesthetized with hematoma block. The MCP is flexed 90° and the direction and force of the displacement/angulation are reversed. After reduction, the wrist is placed in a well-molded ulnar gutter splint incorporating the fourth and fifth fingers with the MCP flexed 70°. Post-reduction x-rays should confirm adequate reduction. Splint is worn for 4 weeks and early ROM exercises begun to prevent stiffness.
   2. Fifth MC neck fractures should be reduced to <30°, especially in boxers or baseball players who may have significant functional compromise with an angulation of 30°. Dorsal angulations <30° demonstrate no significant loss in grip strength (Level of Evidence B, clinical cohort study).[13]
   3. Second and third MC neck fractures should be reduced if angulated >10° and casted with the MCP at 70° for 4 weeks.
   4. MC shaft fractures should be immobilized with the adjacent finger, with the MCP flexed 70° and PIP slightly flexed. Splint is removed after 10 days and active ROM exercises begun. The splint is reapplied if the fracture site remains tender.
   5. Unstable fractures should be referred to orthopedics.

XI. Complications

Higher risk of refracture in sports such as boxing or martial arts if reduction inadequate; cosmetic deformity with excess angulation; loss of power grip with second and third MC fracture angulation >20°; pressure necrosis with "overtreatment" of fifth MC fracture.

XII. Prevention
Proper punching technique, protective gear.

THUMB CMC FRACTURE DISLOCATION: BENNETT'S FRACTURE
I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Axial and abduction forces to the thumb.

III. Anatomy
The anterior oblique carpometacarpal (CMC) ligament holds the palmar fragment in its normal anatomic position. The abductor pollicis longus pulls the MC shaft fragment radial and dorsal.

IV. Symptoms
Pain and swelling over base of thumb CMC.

V. Signs
Variable degree of deformity over the thumb CMC.

VI. X-Rays
Posterior-anterior, lateral, and oblique views will show the palmar fragment ranging in size from a small avulsion fracture to a large triangular fragment.

VII. Special Studies
None indicated.

VIII. Diagnosis
Bennett's CMC fracture dislocation.

IX. Differential Diagnosis
Scaphoid fracture, trapezium fracture, CMC dislocation, soft-tissue injury.

X. Treatment
A. Initially: Treat with RICE and analgesics as needed.
B. Long-term: These are unstable; refer to orthopedics.

XI. Complications
Often unstable if not surgically reduced and fixed.

XII. Prevention
Proper technique, protective gear.

DISLOCATIONS

DIP JOINT DISLOCATION
I. Epidemiology
Collision or contact sports

II. Mechanism of Injury/Pathogenesis
Hyperextension, varus or valgus forces.

III. Anatomy
Rare injury due to the short lever arm of the distal phalanx and strong collateral ligaments; often, dislocations are compound due to the dense cutaneous ligaments that anchor the overlying skin.

IV. Symptoms
Pain and swelling over the DIP joint.

V. Signs
Dorsal or lateral angulation of the DIP joint.

VI. X-Rays
X-rays will show the angulation and associated fractures.

VII. Special Studies
None indicated.

VIII. Diagnosis
Distal interphalangeal dislocation.

IX. Differential Diagnosis
Collateral ligament tear, distal phalangeal fracture, sprain, mallet finger, jersey finger.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: Reduction technique — anesthetize with digital block; middle phalanx is stabilized with one hand and the dorsal base of the distal phalanx is pushed into reduction. Post-reduction should be splinted in slight flexion for 10 to 12 days. The rare irreducible dislocation should be referred for open reduction.

XI. Complications
Joint stiffness if immobilized too long.

XII. Prevention
Proper technique.

PIP DORSAL DISLOCATION (COACH'S FINGER)

I. Epidemiology
Collision or contact sports; most common dislocation of the hand and wrist.[1]

II. Mechanism of Injury/Pathogenesis
Hyperextension injury with resultant disruption of the volar plate at its attachment to the middle phalanx.

III. Anatomy
Loss of the volar stabilizing force causes the phalanx to ride dorsally on the proximal phalanx, producing a "bayonet" deformity.

IV. Symptoms
Pain and swelling over PIP.

V. Signs
Deformity and inability to move PIP.

VI. X-Rays
X-rays will reveal a dorsally displaced middle phalanx, parallel to proximal phalanx with some retraction.

VII. Special Studies
None indicated.

VIII. Diagnosis
Proximal interphalangeal dislocation.

IX. Differential Diagnosis
Collateral ligament tear, PIP fracture/dislocation, volar plate rupture, sprain.

X. Treatment[4,14]
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: Reduction technique — anesthetize with metacarpal block; the middle phalanx is grasped with one hand, giving slight hyperextension of the PIP. The other hand grasps the proximal phalanx and that thumb pushes the middle phalanx into reduction. Longitudinal traction of the middle phalanx may allow soft-tissue interposition into the PIP and should be avoided. Post-reduction should be placed in a dorsal extension block splint with the PIP blocked at 20 to 30° of flexion but allowed to flex for 3 weeks. Follow with buddy taping until symptoms resolve.

XI. Complications
Chronic dorsal instability if inadequately treated acutely.

XII. Prevention
Proper technique and protective gear.

PIP PALMAR (VOLAR) DISLOCATION

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Caused by torsional or shearing stress applied to a semiflexed joint.

III. Anatomy
The above forces result in rupture of one collateral ligament from its proximal attachment and the central slip insertion, allowing the proximal phalangeal condyle to buttonhole through the torn extensor mechanism. The torn collateral ligament may become entrapped between the middle and proximal phalanges, preventing closed reduction.

IV. Symptoms
Pain and swelling over the PIP.

V. Signs
Tenderness over the PIP, especially dorsally and on the side; varying degrees of angular or rotational deformity.

VI. X-Rays
Posterior-anterior, lateral, and oblique views shows volar displacement of the middle phalanx.

VII. Special Studies
None indicated.

VIII. Diagnosis
Proximal interphalangeal palmar dislocation.

IX. Differential Diagnosis
Collateral ligament tear, dorsal dislocation, volar plate rupture, fracture.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: Closed reduction may be attempted but these are frequently irreducible or unstable. If reduction is successful (post-reduction films show normal congruence of joint surfaces), the treatment is the same as for central slip avulsions. Irreducible dislocations should be referred to orthopedics.

XI. Complications
Unstable joint due to soft-tissue interposition.

XII. Prevention
Proper technique, protective gear.

MCP DISLOCATION
I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Torsional or shear forces across the MCP.

III. Anatomy
With simple dislocations, the volar plate remains attached and the proximal phalanx rests perpendicular to the MC. With complex dislocations, the MC head goes through the volar plate, causing a buttonhole effect, and rests between the lumbricals radially and long flexors ulnarly.

IV. Symptoms
Pain, swelling, and stiffness at the MCP joint.

V. Signs
Variable degree of deformity; in simple dislocations, the proximal phalanx is dorsally angulated 60 to 90° but complex dislocations are more subtle, with the involved digit (usually the index finger) slightly hyperextended and ulnar deviated, with dimpling on the palmar surface of the MCP.

VI. X-Rays
Lateral views show hyperextended MCP for simple dislocations. PA views for complex dislocation show widened joint space with asymmetric inclination of proximal phalanx toward the more ulnar finger. Lateral views may show sesamoid interposition between the proximal phalanx and MC.

VII. Special Studies
None indicated.

VIII. Diagnosis
Metacarpophalangeal dislocation (simple or complex).

IX. Differential Diagnosis
Traumatic dislocation of the extensor hood, fracture, collateral ligament tear.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: For simple dislocation, use the same technique as for PIP dorsal dislocation. For complex dislocation, reduction may be attempted if injury is acute and no swelling has occurred:
1. If the deformity is exaggerated and the base of the proximal phalanx is pushed over the articular surface, no longitudinal traction is applied, as this will tighten the entrapment described above. Once reduced, this dislocation is stable and the finger is buddy taped and early ROM begun.
2. Complex dislocations are generally irreducible and should be referred to orthopedics.
3. If after two attempts the reduction is unsuccessful, splint and refer to othopedics.

XI. Complications
Unstable joint if not reduced.

XII. Prevention
Proper technique, protective gear.

LACERATIONS
EXTENSOR TENDON LACERATION
I. Epidemiology
Hockey, field hockey, lacrosse, collision, or contact sports.

II. Mechanism of Injury/Pathogenesis
Laceration over the extensor tendon.

III. Anatomy
Proximal interphalangeal and DIP extension is still possible via the lumbricals and extensor juncturae tendinum if the MCP is flexed.

IV. Symptoms
Bleeding, pain, inability to straighten finger.

V. Signs
Laceration over tendon (lacerated tendon edges may be visualized); inability to actively extend the PIP and DIP joints with the MCP in full extension; passive ROM is full.

VI. X-Rays
X-rays may show an associated fracture.

VII. Special Studies
None indicated.

VIII. Diagnosis
Extensor tendon laceration.

IX. Differential Diagnosis
Superficial skin laceration, partial tendon laceration.

X. Treatment
Debride and irrigate wound; splint wrist in extension with a volar splint to relax the tendon. Refer within 48 hours.

XI. Complications
May require reconstruction with late diagnosis.

XII. Prevention
Proper technique, protective gear.

FLEXOR TENDON LACERATION

I. Epidemiology
Hockey, field hockey, lacrosse, collision, or contact sports.

II. Mechanism of Injury/Pathogenesis
Laceration over flexor tendon.

III. Anatomy
Flexor tendons course through an intricate system of pulleys and sheaths; neurovascular bundle is in close proximity.

IV. Symptoms
Bleeding, pain, inability to flex finger.

V. Signs
Laceration over flexor tendon. Inability to actively flex DIP (profundus) or PIP (superficialis). Capillary refill may be delayed with vascular injury. Two-point discrimination will be abnormal with nerve injury.

VI. X-Rays
X-rays may show an associated fracture.

VII. Special Studies
None indicated.

VIII. Diagnosis
Flexor tendon laceration.

IX. Differential Diagnosis
Skin laceration, cellulitis, flexor tenosynovitis.

X. Treatment
After thorough irrigation and debridement of wound, skin may be closed loosely with interrupted 5-0 nylon and the wrist splinted dorsally in 45° flexion, with the MCPs 60 to 80° and IPs slightly flexed. Patients should be referred to a hand surgeon within 48 hours.

XI. Complications
May require reconstruction if diagnosis delayed; infection.

XII. Prevention
Proper technique, protective gear.

FINGERTIP OR NAIL LACERATION

I. Epidemiology
Collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Direct trauma.

III. Anatomy
Lacerations may involve the nail, nail bed, small avulsions of the pulp, larger pulp amputations, or bony amputations.

IV. Symptoms
Bleeding, pain.

V. Signs
Laceration, avulsion, or amputation.

VI. X-Rays
X-rays will document bone involvement.

VII. Special Studies
None indicated.

VIII. Diagnosis
Fingertip laceration, avulsion, or amputation; nail laceration.

IX. Differential Diagnosis
Crush injury.

X. Treatment
Treat with protective dressing and analgesics as needed. Document tetanus status; dirty wounds may warrant coverage with first generation cephalosporin.

- Laceration: Irrigate and debride under digital nerve block; close with simple interrupted 5-0 non-absorbable suture.
- Small amputations (<1 cm): After thorough irrigation apply sterile dressing.
- Larger amputations: Irrigate, apply sterile dressing, and refer immediately to hand surgeon.
- Bony amputation: Place amputated part in waterproof bag, then in ice water. Do not soak directly in ice water. Refer immediately.
- Lacerated nails: Trim or remove nail. If nail bed is involved, it must be repaired. The nail is removed and the nailbed repaired using 5-0 or 6-0 absorbable sutures.

XI. Complications
Cellulitis, deep space infection.

XII. Prevention
Proper technique, protective equipment.

INFECTIONS

FLEXOR TENOSYNOVITIS

I. Epidemiology
Rock climbing, mountaineering, collision, or contact sports.

II. Mechanism of Injury/Pathogenesis
Puncture wound or laceration over flexor mechanism.

III. Anatomy
Flexor sheath provides a tight lubricated path for the tendon to glide. When infected, the sheath fills with pus and rapidly progresses to a deep palmar space infection.

IV. Symptoms
Pain with finger flexion, swelling.

V. Signs
Overlying puncture wound or laceration, tenderness over flexor tendon sheath, symmetric swelling of the digit, pain with passive extension, flexed posturing of the digit. Warmth and erythema of soft tissues.

VI. X-Rays
X-rays may show soft tissue swelling.

VII. Special Studies
None indicated.

VIII. Diagnosis
Septic flexor tenosynovitis.

IX. Differential Diagnosis
Flexor tendon rupture, cellulitis.

X. Treatment
Immediate referral for incision and irrigation.

XI. Complications
Destruction of the flexor mechanism with delayed treatment.

XII. Prevention
Proper technique, protective equipment.

SEPTIC ARTHRITIS/CLENCHED FIST LACERATION

I. Epidemiology
Rock climbing, mountaineering, collision or contact sports.

II. Mechanism of Injury/Pathogenesis
Open skin wound with (septic arthritis) or without (cellulitis) communication with a joint. Most commonly secondary to "clenched fist" lacerations over fourth or fifth MCP joints. Organisms include *Eikenella corrodens*, anaerobes, staphylococci, and streptococci for human bites; *Pasteurella multicida*, for animal bites.

III. Anatomy
Skin laceration communicates with the joint space, allowing for infection.

IV. Symptoms
Swelling and pain over involved joint.

V. Signs
Tenderness, warmth, and erythema over involved joint; pain with axial compression with septic joint.

VI. X-Rays
X-rays may show associated fracture or possibly air in joint.

VII. Special Studies
Complete blood count (CBC), erythrocyte sedimentation rate (ESR), and cultures; methylene blue dye can be injected into the joint to look for extravasation if capsular disruption is suspected.

VIII. Diagnosis
Septic arthritis, "fight bite," or cellulitis.

IX. Differential Diagnosis
Extensor tendon laceration, superficial laceration.

X. Treatment
Septic arthritis should be referred immediately to orthopedics; cellulitis should be treated with penicillin plus first-generation cephalosporin or ampicillin/clavulanic acid.

XI. Complications
Joint destruction with delay in treatment of septic arthritis.

XII. Prevention
Proper technique, protective gear.

PALMAR SPACE INFECTION

I. Epidemiology
Rock climbing, mountaineering, collision, or contact sports.

II. Mechanism of Injury/Pathogenesis
Laceration or puncture wound to palmar area.

III. Anatomy
Can involve the web space, deep space of the thenar space, mid-palmar space, or Parona's space in the forearm.

IV. Symptoms
Pain, redness, warmth over involved area.

V. Signs
Associated puncture wound or laceration, tenderness.

VI. X-Rays
Usually normal, may show embedded foreign body.

VII. Special Studies
Complete blood count and ESR; cultures should be obtained prior to antibiotic therapy.

VIII. Diagnosis
Palmar space infection.

IX. Differential Diagnosis
Superficial cellulitis, flexor tenosynovitis.

X. Treatment
Tetanus toxoid as needed, surgical drainage.

XI. Complications
Progression of infection, scarring, adhesions with delayed treatment.

XII. Prevention
Proper technique, protective gear.

OTHER

TRIGGER FINGER

I. Epidemiology
Rowing, rock climbing, or any activity requiring repetitive finger flexion.

II. Mechanism of Injury/Pathogenesis
Nonspecific flexor tenosynovitis from over demand.

III. Anatomy
Most common in the flexor tendons of the thumb, middle, and long fingers; inflammatory/degenerative nodule on the flexor digitorum profundus (FDP) catching on the A1 pulley.

IV. Symptoms
Difficulty straightening involved finger (triggering), especially in morning; variable degree of pain.

V. Signs
Variable amount of tenderness over flexor tendon sheath aggravated by active finger flexion or passive extension; palpable nodule in flexor tendon sheath just proximal to the head of the metacarpal.

VI. X-Rays
Not indicated (Level of Evidence C, consensus/expert opinion).[15]

VII. Special Studies
None indicated.

VIII. Diagnosis
Flexor tenosynovitis, trigger finger.

IX. Differential Diagnosis
Infectious flexor tenosynovitis.

X. Treatment
For early or no triggering, splint finger at night. For triggering, inject flexor tendon sheath through a mid-lateral approach over distal one third of the proximal phalanx while the patient resists with active flexion. Repeat in 6 to 8 weeks, and splint at night. Resistant cases may require surgery, which should be a consideration after one to two injections.

XI. Complications
Infection from non-sterile injection.

XII. Prevention
Proper technique.

SUMMARY OF THE HAND
The diverse function and complex structure of the hand and fingers make it a highly visible target in a wide variety of sports particularly basketball, football, wrestling, and gymnastics. The spectrum of trauma is vast, ranging from skin abrasions and contusions to displaced intraarticular fractures and complex ligamentous and tendon problems. Many of these injuries often appear trivial at the time of injury and yet may lead to considerable long-term impairment. A high degree of suspicion, clear understanding of anatomy and biomechanics, accurate evaluation, and early aggressive therapy including a comprehensive rehabilitation program will allow the competent primary care physician to maximize the functional outcome at the earliest possible time.

THE WRIST
General Principles
The wrist is a complex joint capable of motion in three planes. It provides the foundation for force transfer from the forearm and hand and postures the latter for fine motor activity and power grip. The wrist can sustain injury in a wide variety of sports and recreational activities in which weight bearing, twisting, throwing, and impact occur. Because of its diverse functional role, the wrist is particularly vulnerable to injury. Diagnosis and treatment of wrist trauma are especially complex due to its integrated multiply articulated anatomy. Nonetheless, a detailed understanding of anatomy, pathomechanics, and early diagnosis and treatment are crucial for the best possible outcome and maximal function. Long-term outcome is predicated on early diagnosis, treatment, and a sound rehabilitation program.

Epidemiology
Ninety percent of traumatic wrist injuries are secondary to falls where the wrist is dorsiflexed and resisting external forces. This mechanism predisposes the wrist to compressive injuries dorsally, such as scaphoid impactions seen in gymnasts and weightlifters, or tension injuries on the volar side with disruption of the volar ligaments. Ligamentous injuries are uncommon in prepubertal children. Overuse (over demand) injuries result from repetitive microtrauma to the involved structures. Excessive use of the wrist demanded by such sports as fencing, gymnastics, racquet sports, bowling, weightlifting, etc. predisposes to such injuries.

Pathogenesis
Acute
Most wrist injuries are caused by a fall on an outstretched hand (FOOSH), resulting in a dorsiflexion motion with resultant tension on the volar side and compression on the dorsal side.

Chronic
Tendinitis (inflammation of the tendon itself) and tendinosis (degeneration of the tendon) as well as tenosynovitis (inflammation of the tendon sheath) are caused by an over-demand of the involved tendon. The tendon responds first with inflammation and hemorrhage, followed by cellular invasion, collagen production, maturation, and strengthening. Tendinosis, with it degenerative tissue changes, occurs when chronic inflammation goes unresolved.[16]

Anatomy
Functional Anatomy
Wrist motion is passive as no tendons originate from or insert into the carpal bones except for the sesamoidal pisiform. The wrist bones are arranged in two rows, proximal and distal, with only the scaphoid bridging the two (Figure 48.7). Under compressive loads, this scaphoid bridge prevents the two rows from collapsing in a zigzag pattern. This same positioning also places the

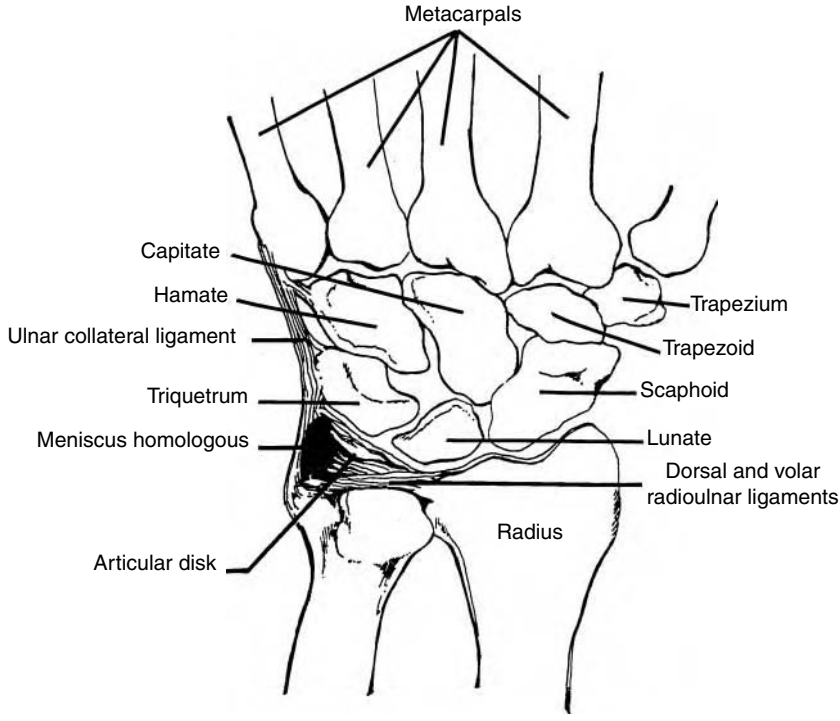

Figure 48.7 The carpal bones and the triangular fibrocartilage complex (TFCC). The proximal row consists of the triquetrum, lunate, and scaphoid. The distal row is comprised of the hamate, capitate, trapezoid, and trapezium. The scaphoid bridges these two rows and is the only bone stabilizing the carpus; therefore, it is subject to injury.

scaphoid and its surrounding ligaments at greatest risk of injury with compressive loads.

Ligamentous Anatomy

The volar aspect of the wrist is stabilized by a series of ligaments configured in a double inverted V pattern with the apices pointing toward the fingers (Figure 48.8). The more proximal V is formed by two intracapsular, extrinsic ligaments: the radiolunate and ulnolunate. The distal V is formed by two intracapsular and intrinsic ligaments: the capitoscaphoid ligament on the radial side and the capitotriquetral ligament on the ulnar side. A potential weak space exists between these two V's and over the capitolunate articulation, called the "space of Porrier." This may be responsible for the perilunar instabilities in hyperextension injuries.

Symptoms

Acute

Significant pain and/or swelling after trauma to the wrist implies a significant osseous or ligamentous injury that may lead to instability and must be carefully investigated. In general, the more pain and swelling associated with an injury, the more serious the injury is likely to be.

Chronic

Pain with active and/or passive motion is the hallmark symptom with the tendinitides or tenosynovitis. Pain with resisted extremes of motion is described with impingement lesions, and sensory changes or weakness with neurologic lesions. Tendinosis causes pain that tends to only partially respond to conventional therapies.

Signs

Acute

The physical exam is generally nonspecific, showing diffuse pain and/or swelling. The range of motion is often limited and the pain may be localized over an area of fracture or ligamentous injury.

Chronic

Swelling and erythema are variable but most prominent with tenosynovitis. The involved tendon is painful with exacerbation of that pain upon

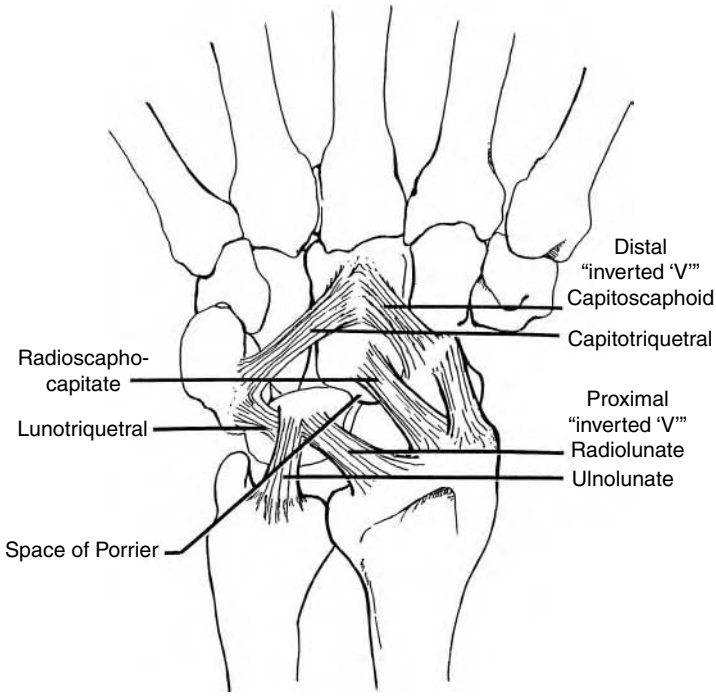

Figure 48.8 The stabilizing volar ligaments of the wrist.

passive stretching or active use. Sensory deficits or motor weakness are found with neurologic lesions. Tendinopathies may feel thickened or have a "rubbery" consistency when palpated.

X-Rays

Acute

Traumatic injuries to the wrist should be evaluated with more than simple PA and lateral views. A six-view wrist trauma series should be ordered, including PA, lateral, right and left oblique, and PA in radial and ulnar deviation, and with a clenched fist, especially if generalized wrist pain is noted.[17] These views can bring out subtle dynamic instabilities not readily seen on non-stressed views.

Chronic

Posterior-anterior, lateral, and oblique views will delineate exostoses related to impingements, old fractures, avascular necrosis (AVN), or chronic instabilities. Conventional tomography in the lateral projection, spot radiographs, and special views (e.g., carpal tunnel) are used to define pathology in poorly visualized areas (e.g., fractures of the hamate hamulus and palmar aspects of the pisiform and triquetrum).

Special Studies

Computed tomography (CT) scanning is useful in evaluating fractures of the hook of the hamate bone, subluxations at the distal radioulnar joint, fracture healing of the scaphoid, and joint fusions of the trapezium, scaphoid, and trapezoid bones. MRI or MR arthrogram is the procedure of choice for carpal ligament tears.[18] MRI is excellent for the diagnosis of avascular necrosis following scaphoid fracture, Kienbock's disease, carpal tunnel syndrome, tendinitis, tendinosis, and tendon ruptures. Radionuclide bone scans are quite useful in localizing injury to the wrist's complex bony anatomy. CT scanning is an excellent study to follow an abnormal bone scan finding.

Treatment

Acute

Most instabilities should be referred to a hand surgeon for definitive treatment; specific injury management is described under individual injuries.

Chronic

The acute pain and inflammation are controlled with ice massage for 20 minutes 4 times a day for the first 48 to 72 hours and are treated with acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs) and by avoiding any activity that

aggravates the pain. The athlete may participate in pain-free activities, which may necessitate any or all of the following: protecting the injured tendon with a wrist splint or brace and continuing the sport, decreasing the intensity or duration of the activity to a level that is pain free, or temporarily substituting another sport or activity that does not cause the wrist to be painful.

After 48 to 72 hours, when swelling has subsided, healing and range of motion are promoted using contrast baths. The wrist is placed in a container of hot water (102 to 105°F) for 4 minutes, during which the athlete performs active ROM exercises. The wrist is then placed in ice water for 1 minute. This cycle is repeated 4 times, always ending with the ice water, and should be done 2 to 3 times a day until painless and full range of motion is achieved.

Ultrasound or phonophoresis is useful at tendon–bone or tendon–muscle interfaces to provide a deep-heating action with possibly some anti-inflammatory benefit. Relative rest or splinting is continued as necessary to allow pain-free activity. Steroid injection in the tendon sheath for tenosynovitis can be useful in cases resistant to more conservative measures, but care must be taken to inject only the sheath and avoid the tendon itself.

When pain is absent at rest and swelling has subsided, high-repetition, low-resistance progressive resistance exercises for wrist flexion, extension, supination, and pronation are initiated using surgical tubing or free weights. The athlete begins with 30-second stretches of the anterior and posterior forearm muscles 3 times, followed by 3 sets of 10 to 15 repetitions of each exercise. The amount of resistance is increased as pain resolves. The forearm stretches are repeated and ice massage is applied to the involved tendon for 10 minutes. For chronic tendinopathies, the same modalities may be applied. An eccentric loading type of strengthening program over several months may help alleviate pain.

SPRAINS/INSTABILITIES

RADIAL LIGAMENT INJURIES

I. Epidemiology

Collision or contact sports, gymnastics, rodeo, parachuting, mountain climbing, skiing; sprains are uncommon in prepubertal children.

II. Mechanism of Injury/Pathogenesis

- Traumatic hyperextension and/or rotational stress to the wrist.
- Scapholunate dissociation (rotatory subluxation of the scaphoid): Normally, the scaphoid palmar flexes (becoming more vertical) with radial deviation of the wrist. The intact scapholunate ligament will force the lunate to move with the scaphoid, resulting in the lunate also palmarflexing with radial deviation. With a ruptured scapholunate ligament, the scaphoid will still palmarflex with radial deviation but the lunate will now dorsiflex, resulting in a vertical scaphoid (rotatory subluxation) and a dorsiflexed lunate. Because the lunate is essentially a passive, intercalated segment, this is called a dorsal intercalated segment instability pattern (DISI) (Figure 48.9).
- Perilunate and lunate dislocations: A severe hyperextension injury to the wrist may result in tension rupture to the volar radioscaphoid and scapholunate ligaments, freeing the proximal pole of the scaphoid, while compressive forces wedge the capitate between the scaphoid and lunate. Continued dorsiflexion will cause the distal carpal row to "peel" away from the lunate and maintain a position dorsal to the lunate and radius, a perilunate dislocation. Further force will rupture the dorsal-restraining radiocarpal ligament, allowing the lunate to flip palmarward (spilled teacup sign) as the distal carpal bones relocate, resulting in a lunate dislocation.

III. Anatomy

Instabilities at the radial aspect of the wrist center around injuries to the ligaments surrounding the scaphoid; these include scapholunate dissociation (also called rotatory subluxation of the scaphoid), dorsal perilunar dislocation, and lunate dislocation; grades 1 to 3 (see Chapter 34).

IV. Symptoms

Pain, swelling, and difficulty moving the wrist.

V. Signs

Scapholunate Dissociation

Tender, swollen wrist has a limited range of motion. Watson's scaphoid test may be positive and is performed as follows: The patient's hand

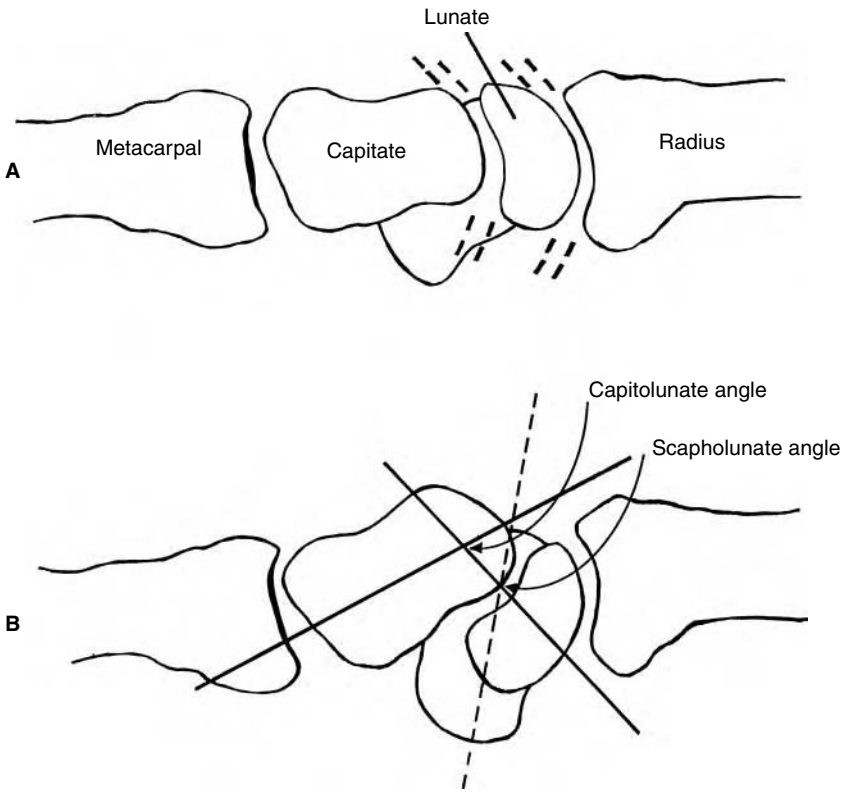

Figure 48.9 (A) The four C's (distal radius, proximal lunate, distal lunate, proximal capitate) on a lateral radiograph should line up in a normal wrist. (B) The capitolunate angle is normally 0 ± 15 and the scapholunate angle should be 30 to 60°. This example shows a DISI pattern with an increase in both the scapholunate and capitolunate angles.

is placed in ulnar deviation (making the scaphoid horizontal) and neutral. The examiner places his or her thumb on the scaphoid tuberosity and the four fingers of the same hand on the dorsal aspect of the distal radius. The thumb pushes on the scaphoid in an attempt to keep it from becoming vertical as the patient radially deviates the hand. With rupture of the scapholunate ligaments, the thumb will effectively prevent the scaphoid from palmarflexing and force the proximal pole to rotate dorsally, causing pain and/or a painful click.[18]

Perilunate and Lunate Dislocations

Tender, swollen wrist with marked limitation in range of motion; particular attention must be paid to median nerve function after this injury.

VI. X-Rays

See Figure 48.9.

Scapholunate Dissociation

Posterior-anterior radiographs (with and without a clenched fist) will show a scapholunate space >3 mm (Terry Thomas sign) or a space greater than that seen between the other carpal bones and a ring sign (the scaphoid appears short and the end-on projection of the rotated scaphoid gives a ringed appearance). Lateral views show the lunate is dorsiflexed (DISI pattern) with a capitolunate angle of >15°. The scapholunate angle is >65 to 70°. If the plain PA and lateral views are normal but suspicion remains, a full traumatic wrist series should be ordered.

Perilunate and Lunate Dislocations

The lunate appears three-sided rather than its normal trapezoidal configuration on PA projections. A true lateral view shows the distal carpal row dorsal to the lunate and radius in a perilunate dislocation and shows a "spilled teacup" sign (the lunate faces palmarward and rests volar to the

radius and distal carpal row) with a lunate dislocation.

VII. Special Studies
Magnetic resonance imaging, arthrography, or diagnostic arthroscopy may be useful with suspected scapholunate injury with equivocal plain radiographic findings.[18]

VIII. Diagnosis
Scapholunate dissociation, perilunate dislocation, lunate dislocation.

IX. Differential Diagnosis
Scaphoid fracture, wrist sprain, ulnar instability.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.[18]
B. Long-term: Referral to a hand surgeon for definitive treatment within 1 week of the injury is preferred.

XI. Complications
Chronic instability, pain, decreased range of motion, and arthritis with delayed diagnosis.

XII. Prevention
Proper technique and appropriate equipment.

ULNAR LIGAMENT INJURIES

I. Epidemiology
Collision or contact sports, gymnastics, rodeo, parachuting, mountain climbing, skiing; sprains are uncommon in prepubertal children.

II. Mechanism of Injury/Pathogenesis
Traumatic hyperextension or rotational injury of the wrist; instabilities on the medial aspect of the wrist center around disruption of the stabilizing triquetrolunate or triquetrohamate ligaments.

III. Anatomy (Grades 1 to 3)
Triquetrolunate Injury
The bony configuration of the carpal bones guides the triquetrum into dorsiflexion with ulnar deviation. Its ligamentous attachments to the rest of the proximal carpal row cause the lunate and scaphoid also to dorsiflex. With rupture of the ligament connecting the triquetrum and lunate (triquetrolunate ligament), the triquetrum will still dorsiflex with ulnar deviation, but the lunate and scaphoid will now be volarflexed. This results in a volar intercalated segment instability (VISI).

Triquetrohamate Instability
The loss of the bony geometric influence of the hamate on the triquetrum allows for a VISI pattern with triquetrohamate ligament rupture.

IV. Symptoms
Swollen, painful joint with limited range of motion.

V. Signs
Triquetrolunate Injury
A painful click may occur as the wrist is compressed and moved from ulnar to radial deviation. The lunotriquetral ballotment test may be positive and is performed as follows: The lunate is stabilized with the examiner's thumb and index finger. With the other hand, the examiner grasps the patient's pisiform and triquetrum and moves them in a volar and dorsal direction, assessing laxity, crepitus, and pain.

Triquetrohamate Instability
A painful click may occur over the ulnar aspect of the wrist with ulnar deviation and pronation.

VI. X-Rays
See Figure 48.9.

Triquetrolunate Injury
Plain PA and lateral radiographic views are often normal. PA views may show a break in the arc between the lunate and triquetrum, and lateral views may show a dorsiflexed triquetrum with a volarflexed scaphoid and lunate (VISI pattern). The scapholunate angle is >70° when positive.

Triquetrohamate Instability
Lateral X-rays may show a volarflexed lunate (VISI pattern) with an acute unstable injury.

VII. Special Studies
Triquetrolunate Injury
If clinical suspicion remains with negative plain films, fluoroscopy, an arthrogram, or arthroscopy may be necessary. An MRI or MR arthrogram may provide the most diagnostic information.

Triquetrohamate Instability
Ulnar instabilities are often dynamic and not identified early on with plain static views. Videofluoroscopy is the most useful tool in evaluating dynamic instabilities, and if suspicion remains, these injuries should be referred to an orthopedic surgeon for further evaluation and treatment.

VIII. Diagnosis
Triquetrolunate instability, triquetrohamate instability.

IX. Differential Diagnosis
Radial ligament instability, fracture, sprain.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: Refer to hand surgeon for definitive treatment.

XI. Complications
Chronic instability, pain, decreased range of motion, and arthritis with delayed diagnosis.

XII. Prevention
Proper technique.

ACUTE SUBLUXATION OF THE EXTENSOR CARPI ULNARIS TENDON

I. Epidemiology
Collision or contact sports, gymnastics, rodeo, parachuting, mountain climbing, skiing.

II. Mechanism of Injury/Pathogenesis
Acute forceful supination of the wrist.

III. Anatomy
Results in tears of the retinaculum over the sixth compartment which allow the extensor carpi ulnaris (ECU) tendon to snap out of the groove.

IV. Symptoms
Painful "snapping" sensation over the dorsal ulnar aspect of the distal ulna with wrist supination and ulnar deviation.

V. Signs
Tenderness over dorsal and ulnar aspect of distal ulna, with palpable tendon subluxation with wrist supination.

VI. X-Rays
X-rays will rule out associated fracture.

VII. Special Studies
None indicated.

VIII. Diagnosis
Subluxation of extensor carpi ulnaris tendon.

IX. Differential Diagnosis
Triangular fibrocartilage complex tear (TFCC tear), ulnar instability, fracture, ECU tendinitis.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: Immobilization for 3 to 4 weeks allows the retinaculum to heal over; chronic injuries may require surgical correction.

XI. Complications
Recurrent painful snapping if untreated.

XII. Prevention
Proper technique.

TRIANGULAR FIBROCARTILAGE COMPLEX (TFCC) INJURY

I. Epidemiology
Collision or contact sports, gymnastics, rodeo, parachuting, mountain climbing, skiing.

II. Mechanism of Injury/Pathogenesis
Subject to injury with dorsiflexion and rotation forces.

III. Anatomy
(See Figure 48.7.) The TFCC suspends the carpal bones from the radius, serves as a major stabilizer of the radioulnar joint, and functions as a cushion for ulnar axial loads. It is comprised of a triangular fibrocartilage, a meniscus homolog, an articular disc, the ulnar collateral ligament, dorsal and volar radioulnar ligaments, and the extensor carpi ulnaris sheath.

IV. Symptoms
Pain with ulnar deviation of the wrist, variable amount of swelling.

V. Signs
Physical exam may reveal ulnar head instability manifested by dorsal subluxation of the ulna. Volar compression between the ulnar head and triquetrum may cause pain or crepitus. A painful clunk in the ulnar region may occur with passive supination or pronation of the wrist. The lift test involves having a patient lift himself or herself out of a chair by weight bearing with wrists extended. One study found this 100% sensitive for a TFCC tear.[19]

VI. X-Rays
Plain radiographs in neutral position may show a fracture or ulnar subluxation (the ulnar styloid should be in the center of the ulnar head).

VII. Special Studies
Magnetic resonance imaging is a reliable method for evaluating the TFCC because it obviates the need for a neutral wrist position. Arthrography is a good method to detect tears of the TFCC but can be difficult to interpret. An MRI arthrogram may be the most sensitive noninvasive test to detect a TFCC injury. Arthrogram is considered the most reliable and gold standard (Level of Evidence C, consensus/expert opinion).[19]

VIII. Diagnosis
Triangular fibrocartilage complex tear.

IX. Differential Diagnosis
Ulnar ligament instability; subluxation of extensor carpi ulnaris, fracture, sprain.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed
B. Long-term: Immobilize for 2 to 3 weeks and re-examine. A steroid injection may be attempted if pain persists after immobilization. Arthroscopy may be indicated if pain persists after these methods have failed. Depending on the location and extent of the tear, as well as the length of the ulnar, a ulnar shortening procedure may be advised.[19] Arthroscopic repair of 35 patients with TFCC injuries had a good outcome in 29 of 35 patients in a retrospective study (Level of Evidence B, systematic review).[20]

XI. Complications
Persistent pain, clicking.

XII. Prevention
Proper technique.

STRAINS
TENOSYNOVITIS
I. Epidemiology
DeQuervain's syndrome, the most common of the tenosynovitides; intersection syndrome, which is more common in athletes such as weightlifters and rowers; extensor carpi radialis brevis tenosynovitis (the second most common), where extensor tendons that are involved in overuse syndromes of the wrist and are frequently used in sports requiring repetitive wrist acceleration and deceleration.

II. Mechanism of Injury/Pathogenesis
Caused by overuse (over-demand) of the tendons and their sheaths surrounding the wrist.

III. Anatomy (Grades 1 to 3)
A. DeQuervain's syndrome: The abductor pollicis longus and extensor pollicis brevis in the first dorsal compartment over the dorsal and radial aspect of the wrist are highly prone to tendinitis and tenosynovitis from repetitive wrist motion.
B. Intersection syndrome: Similar to DeQuervain's disease but occurs more proximally, where the abductor pollicis longus and extensor pollicis brevis cross the radial wrist extensors.[21]
C. Extensor carpi radialis brevis tenosynovitis: Inflammation of the second dorsal compartment on the dorsal radial aspect of the wrist.
D. Extensor pollicus longus (EPL) tenosynovitis: Involves the third dorsal compartment. The EPL tendon passes through a narrow curved tunnel around Lister's tubercle at the middle of the radius. It is relatively thin there and is vulnerable to rupture if untreated or if injected with a steroid.
E. Common extensor tenosynovitis: Involves the extensor digitorum and extensor indicis tendons of the fourth compartment.
F. Extensor carpi ulnaris (ECU) tenosynovitis: Involves the sixth dorsal compartment.

IV. Symptoms
Pain with active use of involved tendon or passive stretching; variable degree of swelling and erythema.

V. Signs
A. DeQuervain's syndrome: Tenderness and swelling are observed over the tendons lateral to the radial styloid. Finkelstein's test is positive and is performed as follows: The patient's thumb is grasped by the four fingers of the same hand and a clenched fist is made.

The patient then deviates the wrist ulnarly, which will result in significant pain over the radial aspect if the tendons are inflamed.

B. Intersection syndrome: Tenderness and swelling of the dorsolateral forearm are noted approximately 4 to 8 cm proximal to the wrist.
C. Extensor carpi radialis/brevis tenosynovitis: Tenderness is localized over the second dorsal compartment on the dorsal radial aspect of the wrist.
D. Extensor pollicis longus tenosynovitis: Tenderness and swelling are noted over the EPL (third dorsal compartment)
E. Common extensor tenosynovitis: Tender, erythematous "goosefoot" swelling can be observed over the mid-dorsum of the wrist.
F. Extensor carpi ulnaris tenosynovitis: The close proximity of the ECU tendon to the triangular fibrocartilage complex makes differentiating between ECU tenosynovitis and old TFCC injuries as the source of pain more difficult. Local pain and tenderness may not be limited to the ECU tendon sheath in tenosynovitis.

VI. X-Rays
Can rule out underlying osseous pathology and reveal soft-tissue swelling.

VII. Special Studies
For extensor carpi ulnaris tenosynovitis, injection of the ECU tendon sheath with a local anesthetic may help differentiate tenosynovitis from TFCC pathology.

VIII. Diagnosis
Tenosynovitis: DeQuervain's syndrome, intersection syndrome, extensor carpi radialis brevis tenosynovitis, extensor pollicis longus tenosynovitis, common extensor tenosynovitis, extensor carpi ulnaris tenosynovitis.

IX. Differential Diagnosis
Tendinitis, cellulitis, TFCC tear, impingement.

X. Treatment
Treat with rest, frequent icing, analgesics, ergonomic adjustments, short-term splinting of the wrist. Consider early injection for DeQuervain's and intersection syndromes. In a pooled qualitative literature evaluation, an 83% cure rate was achieved with injection alone for DeQuervain's syndrome (Level of Evidence B, systematic review).[22] For extensor pollicis longus tenosynovitis, treatment is the same except steroid injections should not be given. DeQuervain's disease may require surgical decompression for resistant cases. Consider surgical management only after 4 months of symptoms and failure of two well-placed injections.[22]

XI. Complications
Usually none; persistent symptoms may require surgical correction.

XII. Prevention
Ergonomic assessment of the workplace and avoidance of repetitive activities.

TENDINITIS

I. Epidemiology
Flexor carpi radialis tendinitis is common in racquet sports. Flexor carpi ulnaris/pisiform tendinitis is the most common wrist flexor predisposed to tendinitis.[21] Flexor digitorum tendinitis is more common in sports requiring repetitive pulling (e.g., rowing) or prolonged pressure on the palms (cycling).

II. Mechanism of Injury/Pathogenesis
Overuse (over-demand) of the wrist.

III. Anatomy
Flexor Carpi Radialis (FCR) Tendinitis
The FCR tendon passes through a groove in the trapezium and passes near the scaphotrapezial and metacarpotrapezial joints as it approaches its insertion onto the volar surface of the second metacarpal. Inflammation in these joints may either cause or be caused by FCR inflammation.

Flexor Carpi Ulnaris/Pisiform Tendinitis
Its broad area of insertion into the pisiform and hypothenar fascia may cause vague pain anywhere from the forearm to the ulnar side of the hand, but it is usually well localized. FCU tendon inflammation may irritate the nearby ulnar nerve, causing paresthesias.

Flexor Digitorum Tendinitis
These tendons pass through the carpal tunnel and can both mimic and cause carpal tunnel syndrome, including median nerve compression.

IV. Symptoms
Pain with active use or passive stretching of the involved tendon. For flexor carpi ulnaris tendinitis, the broad area of insertion of the flexor carpi ulnaris tendon into the pisiform and hypothenar fascia may cause vague pain anywhere from the forearm to the ulnar side of the hand but is usually well localized to the pisiform area.

V. Signs
Flexor Carpi Radialis Tendinitis
Volar radial wrist pain aggravated by resisted wrist flexion.

Flexor Carpi Ulnaris/Pisiform Tendinitis
Flexor carpi ulnaris tendon inflammation may irritate the nearby ulnar nerve, causing paresthesias. Moderate to marked swelling is observed over the pisiform, with increased pain on medial and lateral movement of the pisiform.

Flexor Digitorum Tendinitis
Pain over the carpal tunnel is present and limited mobility of the fingers and hypoesthesia in the median nerve distribution may be noted. Weakness of the thenar muscles and radial two lumbricals indicates motor damage to the median nerve.

VI. X-Rays
X-rays will rule out degenerative changes, avascular necrosis, fracture. For flexor carpi ulnaris/pisiform tendinitis, an oblique radiograph may reveal a calcific deposit near the insertion of the FCU.

VII. Special Studies
A three-phase bone scan can confirm active bony or soft tissue inflammation of the involved tendon. An MRI may demonstrate edema of the tendon or fluid in the tendon sheaths.

VIII. Diagnosis
Flexor carpi radialis tendinitis, flexor carpi ulnaris/pisiform tendinitis, flexor digitorum tendinitis.

IX. Differential Diagnosis
Tenosynovitis, arthritis, chronic carpal instability, avascular necrosis, carpal tunnel syndrome.

X. Treatment
Treatment for flexor carpi radialis tendinitis is as outlined for tenosynovitis. Initial treatment for flexor digitorum tendinitis is as outlined for tenosynovitis; if pain is resistant to conservative treatment or motor weakness is noted, a carpal tunnel release and synovectomy may be necessary. Treatment for flexor carpi ulnaris/pisiform tendinitis is as outlined for tenosynovitis; a steroid injection can be directed into the calcific deposit or the tendon sheath.

XI. Complications
Persistent pain with continued over-demand.

XII. Prevention
Proper racquet holding and grip size; ergonomic assessment and corrections.

IMPINGEMENT SYNDROMES
SCAPHOID IMPINGEMENT
I. Epidemiology
Repetitive wrist-dorsiflexion sports such as weightlifting or gymnastics.

II. Mechanism of Injury/Pathogenesis
Repetitive abutment between scaphoid and radius with wrist dorsiflexion leads to damage to the capsule, ligament, and/or articular surface.[23]

III. Anatomy
The proximal scaphoid and the radius abut with forced hyperextension of the wrist.

IV. Symptoms
Dorsal wrist pain with resisted extension.

V. Signs
The resultant inflammatory response causes the symptoms in this syndrome. Physical exam shows tenderness dorsally between the proximal scaphoid and the radius with the wrist slightly flexed and ulnarly deviated. Forced passive dorsiflexion of the wrist worsens the pain.

VI. X-Rays
In chronic cases, lateral radiographs may show a bony hypertrophic ridge on the scaphoid rim.

VII. Special Studies
None indicated.

VIII. Diagnosis
Scaphoid impingement syndrome.

IX. Differential Diagnosis
Arthritis, tendinitis, chronic fracture or instability, radial styloid impingement syndrome.

X. Treatment
Treatment is as outlined for tenosynovitis. A dorsal wrist block splint may be tried. Steroid injection along the dorsal rim of the scaphoid may be attempted if conservative therapy fails, and resistant cases may require surgery.

XI. Complications
Progression of symptoms and scaphoid rim hypertrophy if repetitive trauma persists; chronic pain.

XII. Prevention
Prevent hyperextension of the wrist and/or use a dorsal-extension-block splint at the wrist.

RADIAL STYLOID IMPINGEMENT

I. Epidemiology
Most common in golf.

II. Mechanism of Injury/Pathogenesis
Usually caused by a mechanism similar to a golfer decelerating the club head during a backswing; analogous to scaphoid impingement discussed above, except for its location.

III. Anatomy
The radial scaphoid and the lateral radius abut with forced radial deviation of the wrist.

IV. Symptoms
Pain with radial deviation of the wrist (e.g., decelerating the club head during a backswing).

V. Signs
Tenderness is generally more prominent on the palmar aspect of the radial styloid and pain is present with forced radial deviation.

VI. X-Rays
Degenerative changes in the radioscaphoid joint may be evident on radiographs.

VII. Special Studies
None indicated.

VIII. Diagnosis
Radial styloid impingement syndrome.

IX. Differential Diagnosis
Scaphoid fracture, chronic carpal instability, tenosynovitis, scaphoid impingement.

X. Treatment
Treatment is as outlined for tenosynovitis.

XI. Complications
Progressive degeneration of the radioscaphoid joint if aggravating activity continues.

XII. Prevention
Slowing or changing the mechanics of the backswing may help (i.e., decreased wrist dorsiflexion); golf swing analysis by professional.

TRIQUETROHAMATE IMPINGEMENT SYNDROME

I. Epidemiology
Floor exercises, racquet sports, and side horse routines.

II. Mechanism of Injury/Pathogenesis[24]
Results from acute or chronic wrist extension and ulnar deviation.

III. Anatomy
Inflammation over the dorsal triquetrohamate joint.

IV. Symptoms
Pain on wrist extension and ulnar deviation.

V. Signs
Tenderness is present over the triquetrohamate joint dorsally, with increased pain on forced wrist extension and ulnar deviation.

VI. X-Rays
May show old fracture, instability, or degenerative changes.

VII. Special Studies
None indicated.

VIII. Diagnosis
Triquetrohamate impingement syndrome.

IX. Differential Diagnosis
Triangular fibrocartilage complex tear, extensor tendinitis/tenosynovitis, old ulnar instability.

X. Treatment
Treatment is as outlined for tenosynovitis; steroid injection along triquetral ridge may be used for resistant cases.

XI. Complications
Traumatic arthritis.

XII. Prevention
Proper technique.

FRACTURES

SCAPHOID FRACTURE

I. Epidemiology
Of all carpal bone injuries 70% are fractures of the scaphoid.[24] Most common in contact and collision sports but can occur with any fall.

II. Mechanism of Injury/Pathogenesis
Forced dorsiflexion injury to the wrist, most commonly the result of a fall on an outstretched hand.

III. Anatomy
The scaphoid has a key stabilizing role as it bridges the proximal and distal carpal rows (see Figure 48.7). It is also vulnerable to avascular necrosis (AVN) after a fracture due to its dependence on a single interosseous blood supply that enters distally and runs proximally. The more proximal the fracture, the more delayed the healing and the higher the risk of AVN. Fractures of the distal one third average 8 weeks to heal, while waist fractures require 3 months. Fractures of the proximal one third require 4 months or more.

IV. Symptoms
Pain, swelling, and limited range of motion.

V. Signs
Physical exam shows tenderness and/or swelling in the anatomic "snuff box" formed by the tendons of the extensor pollicis brevis and abductor pollicis longus radially and the extensor pollicis longus ulnarly. Limited range of motion. Tenderness over the scaphoid tubercle.

VI. X-Rays
A fracture line may be visualized with a scaphoid series. If no fracture line is visualized on x-ray but clinical suspicion persists, the wrist should be immobilized in a thumb spica cast and repeat radiographs taken in 10 days to 2 weeks (and even 4 and 6 weeks if still symptomatic).

VII. Special Studies
If a more expedient diagnosis is desired, a bone scan will be positive within 72 hours of injury. A CT scan or MRI may used for immediate diagnosis if indicated.

VIII. Diagnosis
Scaphoid fracture

IX. Differential Diagnosis
Scapholunate dissociation, lunate or perilunate dislocation, wrist sprain, distal radius fracture.

X. Treatment
A. Initially: Treat with RICE, thumb spica splinting, and analgesics as needed.
B. Long-term: Non-displaced scaphoid fractures can be treated in a long arm thumb spica cast (with the MP joints of the fingers included) for 6 weeks, followed by a short arm thumb spica cast until radiographic evidence of healing is found.[25] Immobilization is then discontinued for activities of daily living and rehabilitation, but the wrist needs to be protected from impact loading for an additional 3 months.[25] An effective soft but rigid scialastic cast as described by Bergfield is useful.[26] Fractures of the proximal third, displaced fractures, or those delayed in presentation (more than 2 weeks) are at high risk for AVN and non-union and should be referred to an orthopedic surgeon.

XI. Complications
Avascular necrosis with subsequent chronic pain and instability, non-union.

XII. Prevention
Proper technique and protective equipment (e.g., wrist guards).

HOOK OF HAMATE FRACTURE

I. Epidemiology
More common in racquet sports and baseball.

II. Mechanism of Injury/Pathogenesis
Trauma to the hypothenar area from a direct blow or from the proximal end of a racquet or baseball bat.

III. Anatomy
The hook of the hamate forms the radial border of Guyon's canal and, because of its perpendicular orientation to the rest of the carpal bones, is vulnerable to fractures.

IV. Symptoms
Pain and variable amount of swelling over the hamate.

V. Signs
Tenderness over the hamate, either palmar or dorsal.

VI. X-Rays
Routine views are often normal. Supination oblique and carpal tunnel views (Hart–Gaynor or supination oblique projection) should be ordered.

VII. Special Studies
Bone scan if x-rays are negative; CT if bone scan is positive.

VIII. Diagnosis
Hook of hamate fracture.

IX. Differential Diagnosis
Guyon's canal syndrome, ulnar artery aneurysm, ulnar instability, bruise.

X. Treatment
A. Initially: Treat with RICE, splinting, and analgesics as needed.
B. Long-term: For a stable, non-displaced fracture, initially immobilize with wrist in slight flexion, MCP of ring and little finger flexed to 90°, with base of thumb included.[24] Radiographic and/or tomographic surveillance should be done every 2 weeks to assure non-displacement; anticipate 8 to 12 weeks of healing time. For a displaced fragment, refer to orthopedics.

XI. Complications
Non-union for displaced fractures.

XII. Prevention
Proper racquet handling technique, proper grips.

RADIAL EPIPHYSEAL INJURIES[27]

I. Epidemiology
Collision or contact sports; one of the most common epiphyseal injuries.

II. Mechanism of Injury/Pathogenesis
Fall on outstretched hand; dorsiflexion injury of the wrist, such as with losing control of weight bar in a military press.

III. Anatomy
Salter–Harris type 1 and type 2 most common.

IV. Symptoms
Pain, swelling, and variable amount of deformity at distal radius.

V. Signs
Tenderness, swelling, and variable degree of deformity at distal radius.

VI. X-Rays
Posterior-anterior and lateral will define the Salter–Harris type and amount of angulation; initial films may look completely normal.

VII. Special Studies
None indicated.

VIII. Diagnosis
Epiphyseal fracture of the distal radius.

IX. Differential Diagnosis
Scaphoid fracture, radial carpal instability, Bennett's fracture/dislocation.

X. Treatment[27]
A. Initially treated with RICE, splinting, and analgesics as needed
B. Long-term: Closed reduction under hematoma block should be performed by a physician experienced in fracture reduction. Two thumbs are placed over the dorsum of the distal fracture and a distal and downward pressure is applied. The amount of reduction depends on the age and maturity of the fracture. A long arm cast is applied with the wrist flexed 25° and ulnarly deviated 15° with forearm supination; the cast is molded over the dorsal carpus and volar concavity of the distal forearm. The cast is bivalved and tightened with tape in 3 to 5 days. Immediate post-reduction x-rays should be done to confirm adequate reduction; in 5 days, take another x-ray. Cast should be worn for 5 weeks.

XI. Complications[27]
Manipulation after 1 to 2 weeks can damage the growth plate; severe growth plate damage can result in significant future deformity.

XII. Prevention
Proper technique and protective gear (e.g., wrist guards) may be helpful.[26]

NERVE ENTRAPMENT SYNDROMES

MEDIAN NERVE: CARPAL TUNNEL SYNDROME

I. Epidemiology
More common with sports involving repetitive wrist motion, such as rowing and racquet sports.

II. Mechanism of Injury/Pathogenesis
Swelling in the rigid tunnel can compress the median nerve and result in sensory and motor deficits.

III. Anatomy
The median nerve at the level of the carpal tunnel contains the sensory branches to the radial 3.5 digits and a motor branch to the thenar eminence.

IV. Symptoms
Wrist pain, numbness in radial 3.5 digits.

V. Signs
Phalen's wrist flexion test reproduces symptoms, and Tinel's sign is positive in approximately 45% of cases. Decreased vibratory sensation in the radial 3.5 digits is the first neurologic finding, followed by decreased two-point discrimination.

VI. X-Rays
Usually not helpful.

VII. Special Studies
Electromyograms may be normal in 25% of patients with CTS but are useful to exclude other etiologies of neurogenic wrist pain. Thyroid function tests including thyroid-stimulating hormone (TSH) and diabetes screen if indicated. MRI of the carpal tunnel and median nerve has fair accuracy in assessing nerve health and degree of swelling.[28]

VIII. Diagnosis
Carpal tunnel syndrome.

IX. Differential Diagnosis
Median nerve entrapment at the elbow, carpal tunnel syndrome secondary to hypothyroidism or diabetes, flexor tendinitis, double crush injury associated with cervical radiculopathy.

X. Treatment
A. Initial: Treatment is conservative, consisting of splinting the wrist in 20 to 30° of dorsiflexion (especially at night), NSAIDs, and relative rest of the wrist.
B. Long-term: A steroid injection may be given with persistent symptoms, as follows. A long, 25-gauge needle is inserted just proximal to the distal flexion crease between the palmaris longus and flexor carpi radialis tendons, piercing the flexor retinaculum. If the needle insertion induces median nerve paresthesias, it must be withdrawn and redirected. Injections of 1 cc of 1% lidocaine and 1 cc of a steroid preparation are given. A single steroid injection of 15 mg of methylprednisolone acetate into the carpal tunnel was found to be more effective than a 10-day course of oral prednisolone up to 3 months after treatment.[29] Successful response to surgery may be predicted by positive response to injection (Level of Evidence B, retrospective cohort study).[30] Resistant cases should be referred for a possible carpal tunnel release if conservative measures have failed after 8 to 12 weeks or sooner if evidence of thenar muscle wasting is found.

XI. Complications
Thenar atrophy, median nerve palsy.

XII. Prevention
Ergonomic assessment and correction of biomechanical factors.

MEDIAN NERVE: PRONATOR TERES SYNDROME

I. Epidemiology
More common in sports requiring repetitive forced wrist pronation, such as baseball and racquet sports.

II. Mechanism of Injury/Pathogenesis
Compression of the median nerve at the elbow.

III. Anatomy[31]
Three most common sites of compression are:

- At a thickened lacertus fibrosis at its connection to the pronator teres.
- At the pronator teres (PT) from a thickened PT with compression between the two heads; fibrous bands in the muscle; or anomalous course of the median nerve.
- At a thickened fibrous arch of the flexor digitorum superficialis.

IV. Symptoms
Insidious onset of anterior, proximal forearm pain; occasional hand pain, with pain worse upon forearm pronation and wrist flexion. Early forearm fatigue and improvement with rest. Occasional paresthesias in the median nerve distribution (radial 3.5 digits).

V. Signs
Tenderness over the pronator teres, positive Tinel's sign over proximal forearm. Increased symptoms with resisted elbow flexion at 120 to 130° and forearm supination indicate compression at the lacertus fibrosis. Forearm pronation and wrist flexion can cause impingement at the pronator teres. Compression at the flexor digitorum superficialis causes increased pain with resisted flexion of the middle finger.

VI. X-Rays
X-rays are generally not helpful, but may show an exostosis-causing impingement.

VII. Special Studies
Electromyography and nerve conduction study abnormalities may or may not be present.

VIII. Diagnosis
Pronator teres syndrome (median nerve compression).

IX. Differential Diagnosis
Carpal tunnel syndrome, double crush injury from cervical radiculopathy, brachial neuritis, anterior interosseous syndrome.

X. Treatment
Treatment includes relative rest (especially avoiding pronation), NSAIDs, stretching, local steroid injections, short-term elbow immobilization at 90° with wrist in neutral position.[32] Refer for possible surgery if axonal loss is present on EMG or if symptoms persist.

XI. Complications
Muscle weakness with advanced cases.

XII. Prevention
Proper technique and ergonomic assessment.

MEDIAN NERVE: ANTERIOR INTEROSSEOUS SYNDROME

I. Epidemiology
Not common with sporting activity but can occur.

II. Mechanism of Injury/Pathogenesis[31]
Compression of the anterior interosseous branch of the median nerve from fascial bands of the deep head of the PT, tendinous origin of the flexor digitorum superficialis (FDS), or anomalous muscles.

III. Anatomy
The anterior interosseous nerve (AIN) is a motor branch that exits above the FDS arch and innervates the flexor pollicis longus, flexor digitorum profundus, and pronator quadratus.

IV. Symptoms
Proximal forearm pain worse with exercise; weak pinch.

V. Signs
Weak FPL, PQ, and FDP. Inability to form an O with forefinger and thumb; no sensory findings.

VI. X-Rays
X-rays are generally not helpful except to rule out bony exostosis.

VII. Special Studies
Electromyograms are useful and may determine the site and extent of damage.

VIII. Diagnosis
Anterior interosseous syndrome.

IX. Differential Diagnosis
Brachial neuritis, pronator teres syndrome.

X. Treatment
Treatment is as for pronator teres syndrome, above.

XI. Complications
Progressive atrophy and weakness of the FPL, pronator quadratus (PQ), and FDP if untreated.

XII. Prevention
Ergonomic assessment and correction.

ULNAR NERVE: GUYON'S CANAL SYNDROME[31]

I. Epidemiology
Most common in cycling and weightlifting.

II. Mechanism of Injury/Pathogenesis
Chronic repetitive insults to the hypothenar area resulting in inflammation in Guyon's canal and ulnar nerve irritation.

III. Anatomy
The ulnar nerve traverses Guyon's canal between the pisiform and hamate and divides into a superficial terminal branch (supplying sensation to the ulnar palm, fifth digit, and ulnar side of the fourth digit) and a deep terminal branch (innervating the interossei, hypothenar muscles, lumbricals 3 and 4, adductor pollicis brevis, and the deep head of the flexor pollicis brevis). The deep terminal branch distal to the branch supplying the hypothenar muscles is most commonly affected. The second, less common, syndrome involves both the deep and superficial branches with compression proximal to Guyon's canal.

IV. Symptoms
Weakness of intrinsic muscles and/or paresthesias in ulnar 1.5 digits.

V. Signs
With deep terminal ulnar nerve involvement (most common), there is weakness in the interosseus, third and fourth lumbricals, adductor pollicis brevis, and flexor pollicis brevis. The hypothenar muscles may be spared since the compression is usually distal to this nerve branch. Proximal Guyon's canal involvement results in weakness in all of the ulnar-innervated muscles plus numbness in the ulnar 1.5 digits.

VI. X-Rays
Carpal tunnel views should be done to assess for a hamate fracture or exostosis.

VII. Special Studies
Electrophysiologic studies help to localize the lesion.

VIII. Diagnosis
Guyon's canal syndrome (ulnar nerve compression).

IX. Differential Diagnosis
Ulnar artery aneurysm, old hamate hook fracture.

X. Treatment
Protective padding over the hypothenar area and NSAIDs. Resistant cases or cases with evidence of motor loss should be referred to an orthopedic surgeon.

XI. Complications
Ulnar neuropathy with intrinsic muscle weakness.

XII. Prevention
Wearing padded gloves while cycling or weightlifting; proper positioning of hands on handlebars.

SUMMARY OF THE WRIST
The injured wrist of an athlete deserves careful, thorough evaluation. Its central position to the transmission of forces and complex anatomy make it uniquely vulnerable to many modes of trauma. While many vague wrist injuries improve with temporary and conservative therapy, a wrist "sprain" should be a diagnosis of exclusion. A missed scaphoid fracture or undiagnosed ligamentous instability can lead to prolonged impairment and the need for protracted intervention. The identification of a specific injury pattern will define a more precise treatment plan and, when appropriate, a more immediate and aggressive intervention permitting return to sport in a timely manner while avoiding unwanted chronic pain or impairment.

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PART V
SPORTS INJURIES: THE LOWER EXTREMITY

49 INJURIES OF THE PELVIS AND HIP*

Jennifer A. Hager and Kenneth M. Bielak

PELVIS AND HIP INJURIES

The pelvis is the focal point of upper and lower extremity balance and determines the center of gravity of the body, located 1 inch anterior to S2. The pelvis acts as the hinge of the two halves of the body and has more muscle attachments than any other bone in the body. The muscles closest to the hip are some of the most powerful muscle

* In the second edition, this chapter was authored by Kenneth M. Bielak and John M. Henderson.

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groups of the body. Because of this central role in all of body mechanics, injuries to the hip can affect the central axis of the body, and vice versa. An injury to one muscle can lead to compensatory actions by other muscles, resulting in overuse and leading to further problems. Accurate diagnosis is imperative if one is to treat and rehabilitate these dysfunctions effectively, as they are compounded by the number of muscles and actions that center on the hip. The pelvis also protects and supports the lower abdomen and its contents, including the genitourinary and intestinal tracts.

Epidemiology

Injuries to the hip and pelvis comprise less than 3% of all injuries to the lower extremity; these are the least frequent sites of injury.[1,2] The most common sports that produce injury to the hip are running and jumping sports, which primarily use the muscles of the hip for locomotion; among gymnasts, hip injuries rank third in frequency and are more likely to be acute rather than chronic injuries.[3]

Anatomy

The pelvis consists of three bones: the ilium, ischium, and pubis (Figure 49.1). The three articulations of the pelvic girdle are the sacroiliac, the sacrococcygeal, and the interpubic, with additional ligaments between the sacrum and ischium. The femur attaches to the lateral pelvis with the acetabulum formed superiorly by the ilium, posteroinferiorly by the ischium, and anteroinferiorly by the pubis. The obturator foramen is formed by the pubis, ischium, and ilium and is covered by the obturator membrane. The greater sciatic foramen is formed by the sacrotuberous ligament, which divides the area of the sciatic notches into the greater and lesser sciatic foramen (Figure 49.2). Through the greater sciatic foramen passes the piriformis muscle, superior gluteal vessels and nerve, inferior gluteal vessels and nerve, internal pudendal vessels and nerve, sciatic nerve, posterior femoral cutaneous nerve, and nerves to the obturator internus and quadratus femoris muscles. Through the lesser sciatic foramen passes the tendon of the obturator internus muscle, the nerve to obturator internus muscle, and the internal pudendal vessel and nerve.

Mechanisms of Injury

In general, most injuries are the result of direct acute trauma (contact/collision sports) or overuse (noncontact and endurance sports). Neurovascular injuries generally result from compression (acute blunt trauma, chronic swelling, and inflammation). In terms of age patterns, adolescents are most vulnerable because of the increasing demands of sports on their slower maturing musculoskeletal systems. At the other end of the spectrum, older athletes are vulnerable to the cumulative effects of recurring injury, increasing inelasticity, and diminished restorative abilities.

History

The following questions will provide a thorough database to aid in diagnosis:

- What is the present problem? When did it first occur?
- How did it occur? What was the mechanism of injury?
- Did you hear any "pop" or "snap" at the time of injury?
- Was disability immediate? How long before symptoms of pain, swelling, disability began?
- Where is the pain localized? What movements or positions aggravate the symptoms? What relieves the symptoms?
- Did you have any preexisting problems prior to this injury?
- Have you had any previous surgeries, medical problems, or use of medications for this or other problems?
- Do you experience any night pain? Any pain at rest?
- Any numbness, tingling, or radiation of pain?
- What are the limitations to your daily activities?
- Describe the limitations to ambulation.
- Any previous treatment measures? Outcome?

Physical Exam and Radiology/Laboratory

Observation

As the patient moves around the room, observe gait, stance, and mobility with change of position, and look for inefficient movements or use of support devices.

Gait Analysis

Evaluate for evidence of a limp, which can be described as antalgic (avoiding pain), circumducted (circular motion), Trendelenburg (sagging gluteus), or short leg (shortened leg, hip, or knee contracture). The affected side is compared with the contralateral side.

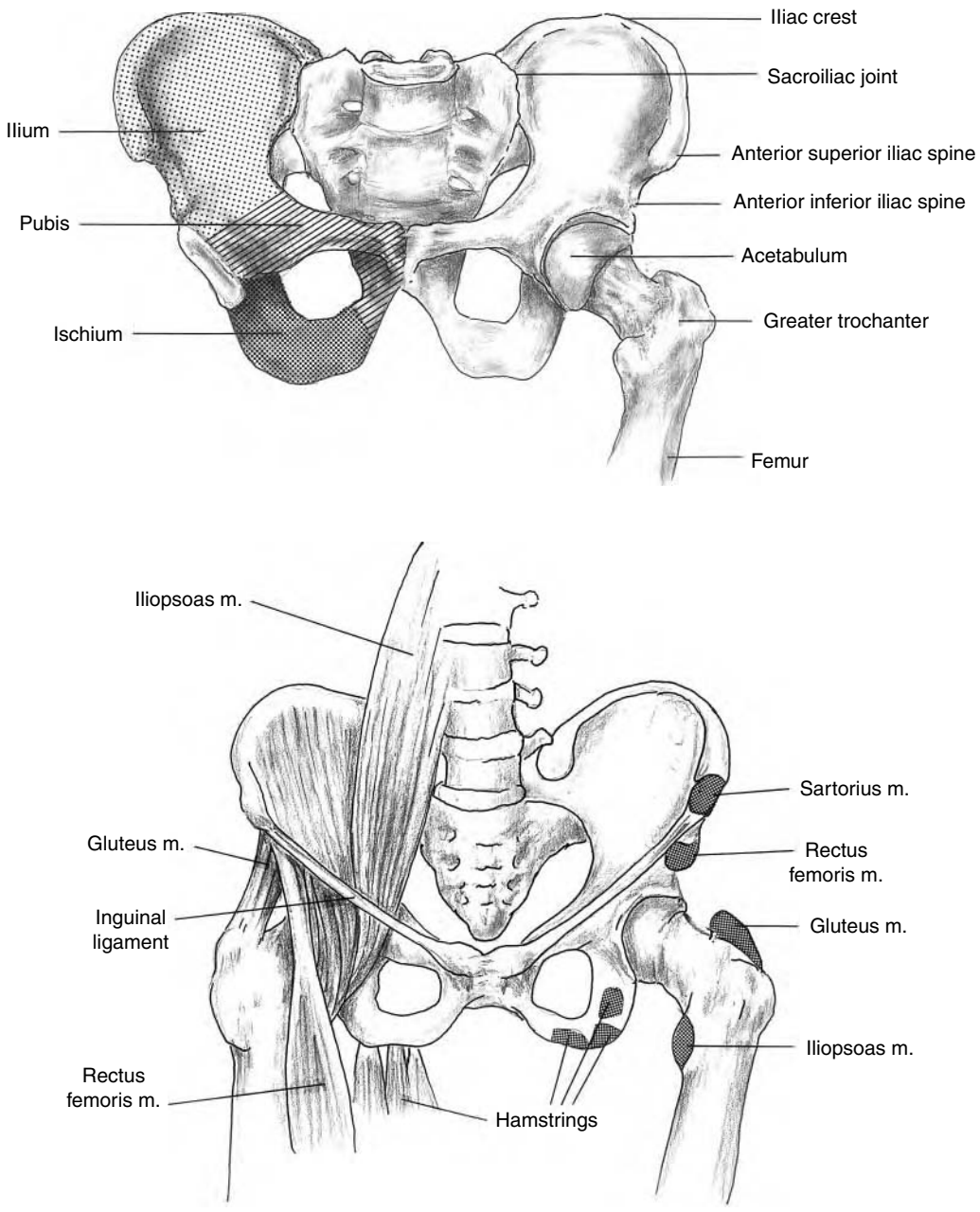

Figure 49.1 Anterior views of the pelvis and hip illustrating bony structures (top) and muscle attachments (bottom).

Inspection

The hip is also inspected for skin changes such as abrasions, pressure sores, discolorations, birthmarks, or abnormal swellings. In the standing position, the patient is examined for asymmetric pelvic height, pelvic tilt or obliquity, and leg length discrepancy. The Trendelenburg test is performed in the standing position and is designed to evaluate the strength of the gluteus medius muscle. When observing the patient who is standing on one leg, the pelvis should be level as the stance leg gluteus muscle contracts normally (a negative Trendelenburg). A positive test would show a sagging hemi-pelvis on the raised-leg side due to a weakened gluteus on the stance leg. The lumbar contour is inspected from the side. An

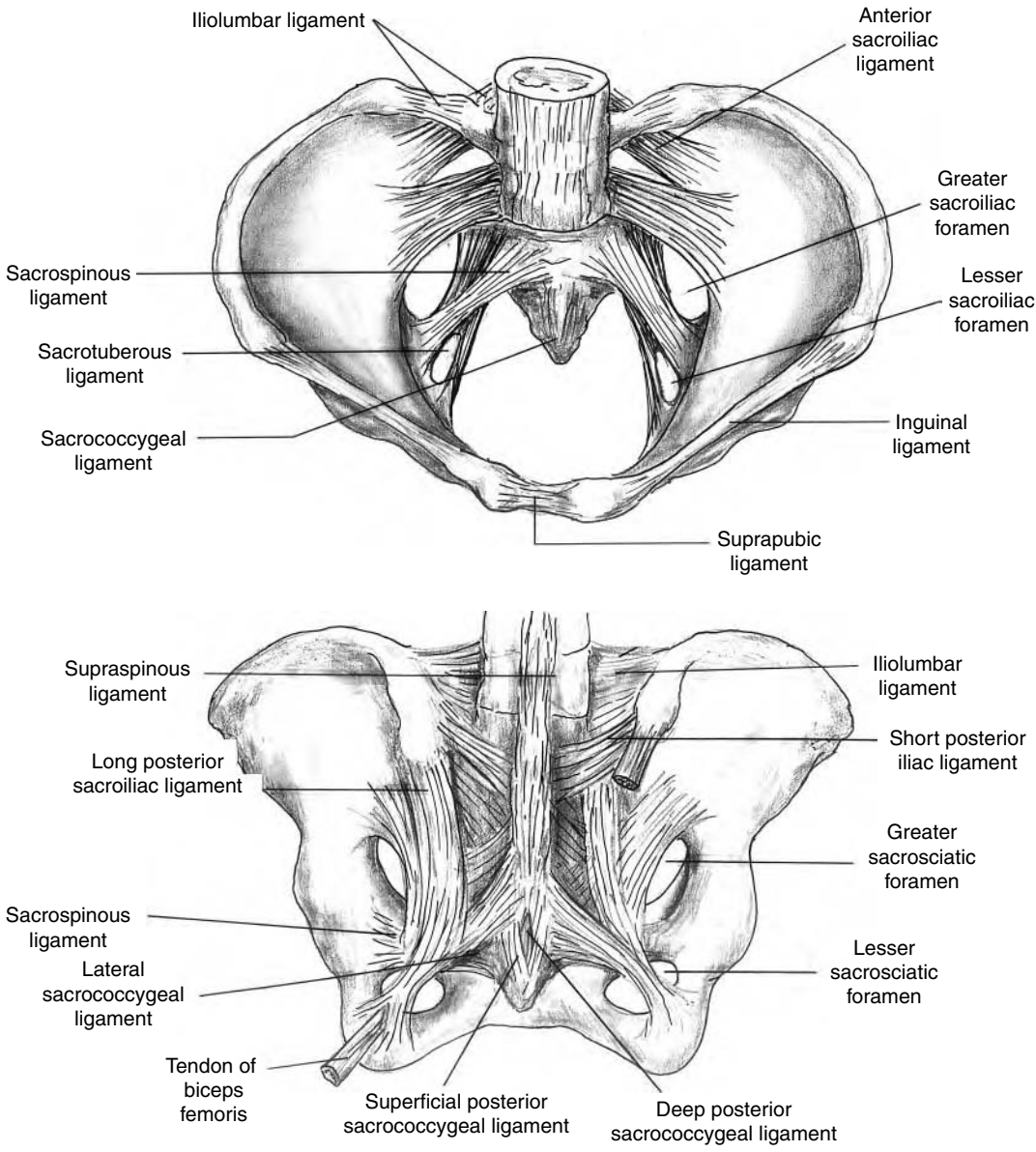

Figure 49.2 Superior (top) and posterior (bottom) views of the pelvis showing ligamentous attachments and bony structures.

absence of normal lumbar lordosis may indicate paravertebral muscle spasms; if exaggerated, it may indicate weak abdominal musculature or a fixed flexion deformity. Posteriorly, the two dimples overlying the superior iliac spine should lie along the same horizontal plane.

Palpation

Examination of the hip requires observation and palpation of bony and soft-tissue landmarks in order to determine the source of pathology. The following structures should be located, identified, and palpated: anterior superior iliac spine (ASIS), anterior inferior iliac spine (AIIS), iliac crest, inguinal crease, posterior superior iliac spine (PSIS), ischial tuberosity, greater trochanter, and pubic symphysis (see Figure 49.1). If percussion of bony landmarks elicits pain, bony discontinuity along the weight-bearing axis is implied. Leg measurements are performed with the patient in the supine position. True leg length is measured from the ASIS to the medial malleolus, while the apparent leg length

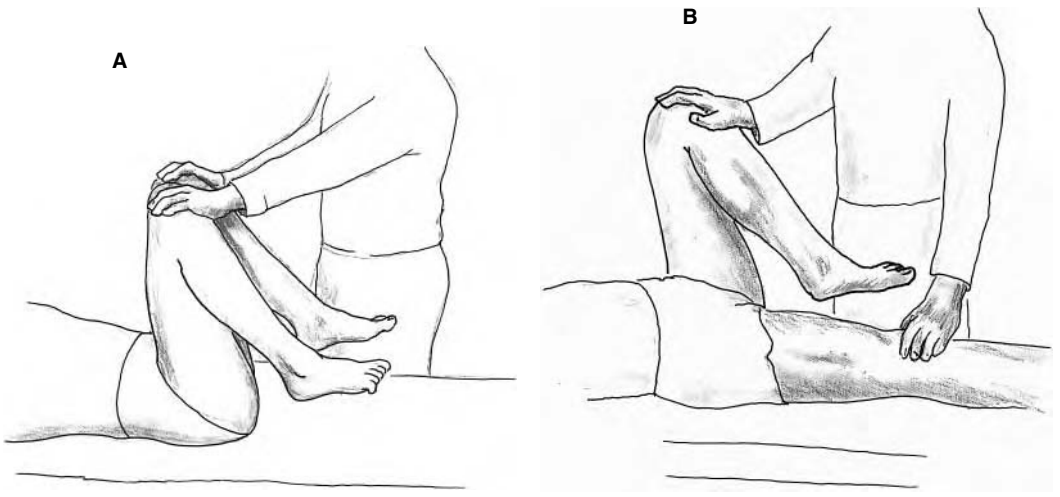

Figure 49.3 Thomas flexion test: (left) flattening of the lordotic curve by hip flexion, and (right) hip extension measures iliopsoas tightness and hip joint flexibility.

is measured from the umbilicus to the medial malleolus. In general, flexion or adduction contractures will produce apparent shortening, while abduction contractures will produce apparent lengthening.

Range of Motion

Range of motion is tested both actively and passively with the patient in the supine position. The hip has a range of 0° to 120–135° of flexion and another 20 to 30° of (hyper)extension. Normally, 45° of abduction and 30° of adduction are observed, while the hip can be internally rotated 40° and externally rotated 45°. Take particular note of any decreased mobility because of pain or mechanical block, such as contractures. Hamstring tightness, for example, may indicate spinal pathology and is a harbinger of strains and patellar problems.

Muscle Testing

Muscle strength is tested comparing both hips, including the abductors, flexors, adductors, and extensors, using the 0 to 5 muscle grading scale. The primary hip flexor is the iliopsoas muscle innervated by the femoral nerve (L1–L3). This is tested by manual resistance as the patient tries to elevate the flexed knee from the sitting position. Take special note of substitution patterns, such as sartorius involvement causing lateral rotation and abduction of the hip and tensor fascia lata causing medial rotation and abduction. Immediate testing of the contralateral hip provides a basis for comparison. The primary extensor is the gluteus maximus muscle innervated by the inferior gluteal nerve (S1). This is tested with the patient in the prone position as he or she tries to raise the tested thigh from the table against resistance applied at the knee. For elderly or weakened patients, this test may be estimated with the patient sidelying. The adductor longus (obturator nerve, L2–L4) can be tested with the patient supine or on the side (avoiding any pelvic rotation causing substitution patterns). Resistance is applied to the adducting leg. In a similar manner, the primary abductor, the gluteus medius (superior gluteal nerve, L5), can be tested with the patient abducting the leg against resistance. The neurovascular exam includes documentation of the pulses, deep tendon reflexes, and any sensory deficits, which can be tested with a pinwheel and outlined on the dermatome chart for ease in recording. Medial thigh pain frequently indicates hip pathology.

Additional Tests

The flexion test (Thomas test) is performed to detect flexion contractures of the hip (Figure 49.3). In a supine position, the patient holds one hip in extreme flexion with the knee against the chest while the other is extended. Inability to place the contralateral leg in full extension indicates flexion contracture of the iliopsoas, which can be due to bursitis or the "snapping hip" syndrome. The Patrick or FABERE (flexion, abduction, external rotation, and extension) test is helpful in evaluating the hip joint (Figure 49.4). The leg is flexed, abducted, and externally rotated by placing the ipsilateral ankle above the opposite

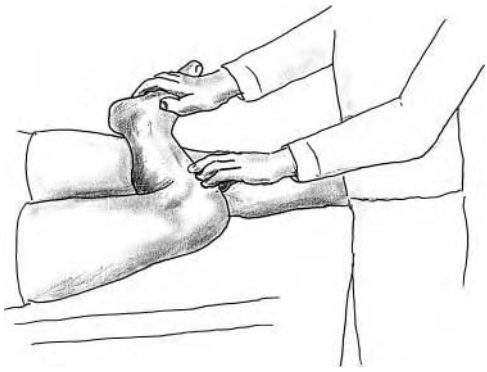

Figure 49.4 Patrick or FABERE (flexion, abduction, external rotation, and extension) test.

knee as the examiner continues to gently externally rotate the leg and extend the hip while stabilizing the opposite hip. Discomfort with this maneuver suggests arthritis of the ipsilateral hip. This maneuver may also indicate SI joint dysfunction.

The hip flexion test passively evaluates the normally smooth in-line motion of hip flexion (see Figure 49.5). When spontaneous external rotation of the hip occurs during this maneuver, the examiner should consider slipped capital femoral epiphysis or joint incongruity. In the adolescent or immature athlete this is viewed as an acute injury, but in the older athlete it is commonly a chronic problem and presages the development of arthritis.

The log-roll test, or passive internal and external rotation of the hip, when positive (increased discomfort) suggests inflammation such as synovitis, infection, or even fracture of the hip.

Ober's test is useful for detecting contractures of the iliotibial band. The patient lies in lateral decubitus position with the side to be tested facing up. The patient's knee is flexed 90°; the hip is abducted to 40° and extended to its limit. The limb is then adducted toward the examining table. The normal hip can be adducted past the midline of the body without pain.

The piriformis test is useful for detecting piriformis tendinitis or piriformis syndrome. The patient is in the lateral decubitus position with the hip to be examined facing up. The patient's hip is flexed 45° with the knee flexed to 90°. The examiner then pushes the flexed knee toward the floor. This maneuver stretches the piriformis muscle and will elicit pain with tendinitis. If pain radiates in distribution similar to sciatica, this is

Figure 49.5 Hip flexion test; spontaneous external rotation occurs with hip flexion in cases of a slipped capital femoral epiphysis (SCFE).

indicative of piriformis syndrome (entrapment of the sciatic nerve within the piriformis). Other maneuvers used to detect piriformis syndrome are Freiberg's and Pace's maneuvers. Freiberg's maneuver involves forceful internal rotation of the extended thigh which produces deep buttock pain.[5] Pace's maneuver involves the patient abducting the legs in the seated position which causes a contraction of the piriformis.[5] An alternative maneuver that distinguishes piriformis pain from hip and back morphology involves the patient lying with the painful side up, the painful leg flexed, and the knee resting on the table. Buttock pain is produced when the patient lifts and holds the knee several inches off the table.[5]

Additional exams that may be necessary include pelvic, rectal, genital, hernia, back, and knee exams as deemed appropriate.

Laboratory and X-Ray Evaluation

X-rays of the pelvis and hip are obtained to show any bone and joint abnormalities or malalignment. Common views to obtain include the anteroposterior (AP), lateral, and abducted AP (frog leg) views. The Judet view and computed tomography (CT) are recommended if a fracture is suspected. Bone scan, CT, and magnetic resonance imaging (MRI) are often useful in differentiating

avascular necrosis from inflammatory or infectious processes. Hypodense areas on CT suggest strains (swelling); hyperdense areas suggest hematomas.[4] MRI can facilitate defining the extent of soft-tissue injury (e.g., strains) as well as stress fractures.

ACUTE SOFT-TISSUE INJURY

Contusion/Hematoma

(See Chapter 33.) Contusions, or bruises, and hematomas are common injuries seen in contact sports such as football and rugby and in any sport with the potential for falls, such as skating. The lateral and posterior aspects of the hip are more commonly injured than the anterior, due to the mechanism of falling or being hit directly. Hip pointers are direct blows to the iliac crest commonly seen in football players and in gymnasts on the uneven parallel bars. An audible "pop" or "snap" may be heard at the time of injury, and the patient may present with a limp. Point tenderness, ecchymosis, swelling, and muscle spasm are common findings. The gluteus muscles (medius, minimus, and maximus) are relatively thick muscles that give additional protection to the pelvis and hip posteriorly and laterally. Nevertheless, large hematomas may form, depending on the extent of injury and the number and caliber of vessels that are disrupted. This may cause compression of adjacent nerves such as the femoral or lateral femoral cutaneous nerves. Additional injuries to the adjacent neurovascular bundles, ligaments, connective tissue, and skin may occur. The differential diagnosis should include avulsion fractures (especially in younger athletes), iliac apophysitis, piriformis syndrome, strains, sprains, and neural injury. Potential avulsions can be ruled out by x-ray (AP pelvis). Treatment consists of rest, ice, compression, analgesics, stretching, and padding.

Strains

(See Chapter 34.) Strains of the hip muscles are seen most commonly among speed athletes, those requiring bursts of speed or rapid acceleration with sudden change of direction, such as in basketball, football, soccer, rugby, and track and field. At risk are athletes who lack flexibility or who demonstrate inadequate warming-up, stretching, and conditioning. Examination may reveal tenderness over the lesser trochanter or ischiopubic ramus and associated edema or ecchymosis. Pain is elicited with resisted hip flexion and adduction. The differential diagnosis includes avulsion and stress fractures, hematoma, nerve stretch injuries, lumbar disc herniation (sciatica), and trochanteric bursitis. It is important to rule out any disorders of the bowel, bladder, and testicles. Treatment consists of analgesics, rest, ice, and stretching. An ultrasound may be indicated if chronic symptoms persist.

CHRONIC SOFT-TISSUE INJURY

Bursitis

(See Chapters 33 and 49.) Bursal sites typically aggravated are the trochanteric bursa, palpated just posterior to the greater trochanter; the iliopsoas, with tenderness over the lesser trochanter; the ischium, with tenderness over the ischial tuberosity; and, infrequently, the iliopectineal and gluteal bursae. Point tenderness over the offending bursa is typical, with radiation to the thigh or buttock pain. Tenderness of the greater trochanteric bursa is best elicited when manually resisting abduction and external rotation while the patient is on his or her side. Pain is also elicited when moving the hip from full extension to flexion. The iliopsoas is identified by medial groin pain which may radiate to the femoral triangle. Pain is elicited with flexion, abduction, and external rotation against resistance. Ischial bursa inflammation is usually the result of direct trauma or a hamstring pull. It is aggravated by sitting and contraction of hamstrings. X-rays are usually unremarkable but are necessary to exclude other possibilities. The differential diagnosis includes stress fractures of the femoral neck and the ischium and underlying bone pathology such as protruding bone tumors (endochondroma, osteomas, osteochondroma). Treatment guidelines, complications, and prevention have been outlined in Chapter 34.

CHRONIC BONY INJURY

Stress Fractures (Hip and Pelvis)

(See Chapter 34.) Stress fractures can occur in the hip and pelvis with chronic overuse and/or an increase in the level or intensity of activity. They are most commonly seen in distance runners, with the majority being female in their second to fifth decade. Amenorrheic athletes are at increased risk. The most common sites include the femoral neck, ischiopubic rami, and iliopubic rami. Pain localizes to the groin, buttock, or thigh; increases during and after activity; and progressively worsens with time. X-rays are typically normal for the first 6 to 8 weeks but are worth repeating to demonstrate a fracture site and/or periosteal reaction. When positive, x-rays may show callus formation, endosteal thickening, or line formation.

The test of choice has in the past been the bone scan using technetium 99-m pertechnetate (Tc99m), which can pick up osteoclastic activity early and accurately; however, MRI is considered more specific. The bone scan is usually positive 2 to 8 days after the development of symptoms. MRI is preferable to bone scan because it yields a more specific diagnosis, offers better spatial resolution, and allows simultaneous evaluation of surrounding soft tissues (Level of Evidence B, review).[6] MRI also detects early changes of osseous stress injury, and it is the preferred modality for evaluating the continuum of osseous manifestations of stress injury (Level of Evidence B, review).[7] The differential diagnosis includes symphysitis, periostitis, and small avulsion fractures of the ilium and ischium.

Evaluation and management should follow the guidelines provided in Chapter 34. Stress fracture of the femoral neck requires consultation for prophylactic pinning, crutches, or protective ambulation. For the ischium and the pubis, crutch walking guided by symptomatology is recommended, with 2 to 3 additional months of active rest (progressive exercises while abstaining from active sport).

Supracondylar femur stress fractures occasionally require cylinder casting to prevent extension of the fracture line, but small fractures may do well with just crutch walking and close observation. The major complication other than outright progression to fracture is avascular necrosis (AVN) of the stressed bone if it is not allowed to heal properly and recurrent fractures if the return to activity is premature. An MRI of the hip may be helpful in order to rule out AVN.

Iliac crest apophysitis represents a microscopic stress fracture within the growth center of mid-adolescent runners. The mechanism of injury is related to repetitive pulling of the abdominal muscles on their insertion to the iliac crest. Pain is described as gradual in onset, typically during activity such as running, and it progressively worsens until it prevents further training. Examination reveals point tenderness to the anterolateral pelvic crest, aggravated with sit-ups. Rest for 4 to 6 weeks is necessary, but other aerobic activities such as swimming or bicycling may be comfortably substituted. Posterior iliac crest apophysitis can be found in cross-country runners and may be related to deceleration forces from running downhill.

ACUTE BONY TRAUMATIC INJURIES

FRACTURES AND DISLOCATIONS OF THE HIP

I. Epidemiology

Generally rare in most contact sports; seen mostly in high-energy trauma (motorcar, motocross, ice dancing, long jumping, rodeo, horse racing, and other equestrian events).

II. Mechanism of Injury

Severe direct trauma; anterior dislocation occurs with hips abducted, and posterior dislocation occurs with blow to knee with hip and knee flexed (90% of dislocations are posterior).

III. Anatomy

Weak points of pelvis: rami, femoral head, and neck.

IV. Symptoms

Severe pain, immediate disability.

V. Signs

Localized tenderness at the area of fracture, crepitus with palpation and/or compression over the bony part (gently perform pelvic rock and pubic compression tests). Patients with posterior dislocation present with the leg flexed, adducted, and internally rotated. The femoral head may be palpable in the buttock. Those with an anterior dislocation present with the leg in external rotation, abduction, and either flexion or extension.

VI. X-Rays/Laboratory

Mandatory films of pelvis and femur, AP and lateral. The pelvic rim is continuous, so if one fracture site is detected, look for another. The plain AP will clearly show the dislocation, but CT may be needed to show acetabular wall and femoral head fractures (Level of Evidence B, review).[8]

VII. Special Studies

Computed tomography is essential after reduction to detect osteochondral fragments and can reliably detect residual subluxation of 2 mm or more at any part of the joint.[8] MRI is not indicated in the early treatment but is needed 6 weeks after reduction to rule out avascular necrosis.

VIII. Diagnosis

Hip.

IX. Differential Diagnosis
Visceral, urinary tract, femoral nerve, or arterial injuries; subluxation.

X. Treatment
Immediate immobilization with traction splint (Hare splint); if none is available, use two crutches medially and laterally or corrugated cardboard gutter splints. Apply pressure dressing and seek emergent consultation with an orthopedic/general surgeon for reduction and stabilization. Remember that blood loss can exceed 6 to 8 units.

XI. Complications
Immediate exsanguination, nerve, and arterial damage; delayed risk for avascular necrosis. Posterior hip dislocations have very high risk for compressive sciatic neuropathy. A dislocated hip is considered ischemic, and the time to relocation strongly influences the frequency of delayed complications. The risk of osteonecrosis of the femoral head is affected by the time it takes to reduce the hip.

XII. Prevention
General safety measures, protective equipment, and proper falling techniques.

HIP SUBLUXATION
I. Epidemiology
A more recently recognized injury that may be surprisingly subtle in its presentation;[9] it is usually seen in sports where athletes stop quickly and pivot over a weight-bearing extremity (football, soccer).

II. Mechanism
Injury occurs when force is applied to a flexed knee with the hip adducted. This forces the head posteriorly onto but not over the acetabular rim. Spontaneous reduction occurs.

III. Anatomy

IV. Symptoms

V. Signs

VI. X-Rays/Laboratory
A fracture of the posterior acetabular rim may be seen on Judet views. MRI will usually demonstrate the fracture as well as a contusion of the femoral head.[9]

VII. Special Studies
Not applicable.

VIII. Diagnosis
Hip subluxation.

IX. Differential Diagnosis
Dislocation, fracture, strain, sprain.

X. Treatment
Rehabilitation protocol of crutches, non-weight-bearing, for 6 weeks, followed by a repeat MRI and then stretching and strengthening if MRI is negative.[9]

XI. Complications
Chondrolysis and AVN.

XII. Prevention
Proper protective equipment and athletic techniques.

AVULSION FRACTURES
I. Epidemiology
Peak incidence is in the second decade when apophyses appear (age 11 to 15). These fractures are seen in sports with sudden acceleration or deceleration forces, such as sprinting or kicking (e.g., football, soccer, basketball) (Figure 49.6).

II. Mechanism of Injury
Acute muscular contraction against a fixed resistance across an open apophysis.

III. Anatomy
An apophysis is a secondary ossification center that contributes to peripheral but not longitudinal skeletal growth and tends to fuse at a later age than epiphyses of the long bones. Avulsions are likely to occur at the following bony landmarks, with their corresponding muscle attachments: the ASIS (sartorius and tensor fascia lata), the AIIS (rectus femoris, which contracts to maintain balance, especially when falling backward after landing from a jump), the ischial tuberosity (hamstrings), the greater trochanter (piriformis), the lesser trochanter (iliopsoas), and the ischiopubic rami (adductors) (see Figure 49.1). The ischial apophysis may not close until the age of 25 years. Avulsion of the apophysis may follow a maximum contraction of the quadriceps against fatigued hamstrings, causing an overload.[10] This is commonly seen in tired athletes who continue to exert themselves.

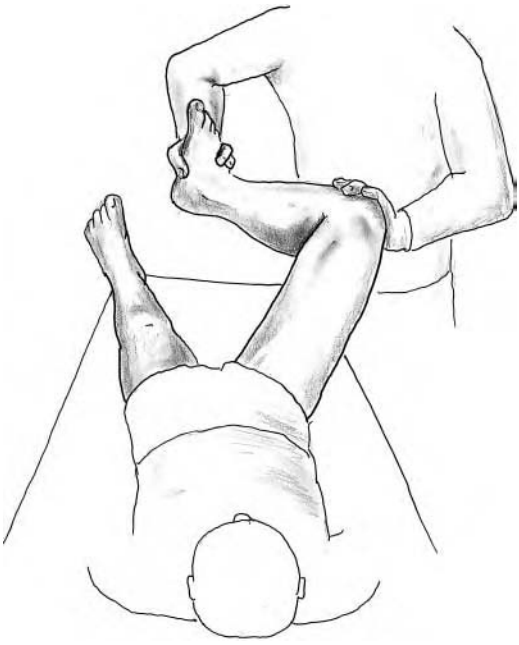

Figure 49.6 Avulsion of the ischial tuberosity during forceful kicking.

IV. Symptoms

Localized immediate pain, with limitation of activity and occasionally pain of longer duration.

V. Signs

Patients will maintain a position that reduces tension on the involved muscle. Swelling, point tenderness, and increased pain with specific muscle testing are observed. Flexion of the hip against resistance increases the pain of ASIS avulsion injuries. A tensor fascia lata avulsion occurs in baseball players usually while swinging the bat. Sartorius avulsions are seen in sprinters. In AIIS avulsion injuries, manual muscle testing of the quadriceps elicits pain. Flexion against resistance, passive or active extension, and rotation of the hip against resistance cause pain to the ilipsoas muscle at its insertion to the lesser trochanter (usually occurs during kicking); abduction and external rotation against resistance will elicit tenderness to the superior greater trochanter at the insertion of the piriformis. With an avulsion of the hamstring muscle from the ischial tuberosity, the patient may experience sudden pain in the buttock with inability to continue activity. It usually occurs when the hip is in flexion and knee extended. A positive straight leg raise may be present.

VI. X-Rays/Laboratory

Anteroposterior and lateral views show avulsion fractures at the anatomic site of injury. Diagnosis is simplified by comparing the radiolucent distance between the apophysis and main bone from the uninjured paired area of bone. Serial x-rays are helpful to ensure adequate healing.

VII. Special Studies

Computed tomography scans are helpful to reveal anatomic detail, bony alignment, and fracture position but cannot be relied on to confirm healing. CT may be helpful in determining whether an ASIS avulsion is secondary to a sartorius versus tensor fascia lata (TFL) contraction. The fragment in sartorius avulsion tends to be small and displaced anteriorly whereas a TFL avulsion is larger and displaced laterally.[11] MRI is excellent for soft-tissue detail. Bone scans are usually not needed.

VIII. Diagnosis

Avulsion fracture, location.

IX. Differential Diagnosis

Strain or "groin pull," periostitis, hip pointer; without a history of antecedent trauma and radiographic changes, consider osteomyelitis, tumors, and Ewing's sarcoma.

X. Treatment

Uncomplicated injuries, as defined by minimal displacement (less than 15 mm), and/or small fracture site can be managed with RICE and crutches with toe-touch weight bearing for 1 to 2 months (guided by symptoms). The muscle should be protected by positioning that does not allow further pull to displace the fragment. At 2 to 3 weeks, when pain is controlled and the inflammatory response has subsided, gradual stretching is introduced. When over 80% of ROM has returned, a gradual strengthening program is introduced, progressing to a graduated return to activity. If complicated (>15 mm displacement) or the displaced fragment is large, surgical fixation may be required to prevent muscular atrophy, chronic pain, or a bony prominence on sitting. Late surgical excision may be necessary.

XI. Complications

Nonunion of fragment and increased ossification, resulting in a bony prominence leading to later

mechanical problems. This occurs increasingly with premature return to activity.

XII. Prevention
Adequate conditioning, stretching, and warm-up.

DEVELOPMENTAL DISORDERS

SLIPPED CAPITAL FEMORAL EPIPHYSIS (SCFE)

I. Epidemiology
Generally a nonsports disease of late childhood to late adolescence. Males are more commonly affected, ages 10 to 17, with the left hip twice as likely to be affected as the right; females have equal predisposition to laterality. Blacks are more likely to have this condition than whites, and it is generally found in obese children. It can occur in younger children with trauma, malnutrition, tuberculosis, or congenital hip disorder. More than one-half of unilateral cases are asymptomatic; 16 to 49% are bilateral.

II. Mechanism of Injury
A. Mechanical factors — acute trauma or repetitive microtraumas with increased shearing stress on the epiphysis, especially with abduction and external rotation
B. Endocrine factors — increased growth-hormone/sex-hormone ratio

III. Anatomy
Acute or chronic slippage of the capital femoral epiphysis, classified as:
A. Pre-slip — subtle widening and rarefaction of the plate without displacement
B. Minimal slip — slippage that is very slight to 1/3 of the epiphyseal diameter
C. Moderate — slippage of 1/3 to 2/3 of the epiphyseal diameter
D. Severe — displacement of >2/3 of the epiphyseal diameter

IV. Symptoms
Antalgic gait with anteromedial groin and thigh pain, often radiating to the knee. Some children present with chronic knee pain. Typically, acute cases have a history of trauma.

V. Signs
Adduction with external rotation deformity of the limb with decreased range of motion, especially with abduction and internal rotation, and tenderness over mid-medial groin; positive flexion rotation test (the leg rotates externally with flexion). Always evaluate the contralateral, "normal" side.

VI. X-Rays/Laboratory
Anteroposterior, frog-leg, or lateral views with comparison views (caution must be used with the frog-leg view as it may exacerbate symptoms). Look for osteopenia of the femoral neck and head, blurred margin of the metaphysis, reduced height of the epiphysis, abnormal "Klein's lines" (a tangent to lateral femoral neck does not intersect the epiphysis). X-ray findings may be subtle or absent.

VII. Special Studies
Computed tomography scan can delineate acetabulo-femoral osseous detail. Ultrasounds may be better than x-rays but their usefulness depends on the experience of the examiner. MRI is usually used for assessments of pre-slips and is more useful for detecting edema in the area of the physis and width of the physis (Level of Evidence B, review).[12] Bone scan using Tc99m can be useful.

VIII. Diagnosis
Slipped capital femoral epiphysis.

IX. Differential Diagnosis
Same as for Legg–Calve–Perthes disease.

X. Treatment
Immediate referral to orthopedic surgeon for percutaneous fixation (send them along with crutches).

XI. Complications
Severe varus deformity, shortening, broadening of femoral neck, osteonecrosis (6 to 15%), chondrolysis (40%), and degenerative joint disease.

XII. Prevention
None, but early diagnosis prevents further complications.

LEGG–CALVE–PERTHES DISEASE

I. Epidemiology
The incidence is around 1:2000, and it is most often seen in males (5:1), usually ages 4 to 10 years. It is bilateral in 10% of males. It is rarely seen in blacks, and 6% of patients have a positive family history. A history of trauma is present in 25% of cases, although the condition can be seen

in normal, vigorous activity. The diagnosis should, therefore, be considered in any child with hip pain.

II. Mechanism of Injury

Unknown; pathogenesis may be due to synovitis of the hip leading to increased intraarticular pressure, resulting in decreased blood flow.

III. Anatomy

Osteonecrosis of the femoral head, secondary to avascularity and ischemia.

IV. Symptoms

Intermittent limping, decreased range of motion with pain localized to the groin with referral to the anteromedial thigh and knee (following the sensory obturator nerve). Symptoms can vary over time. Atrophy and spasm with contracture of adductor muscles may be noted. A history of trauma is found in 25% of cases.

V. Signs

Antalgic gait with limitation of motion, especially abduction and internal rotation; proximal thigh atrophy and limb shortening; flexion–adduction contracture chronically.

VI. X-Rays/Laboratory

Complete blood count, erythrocyte sedimentation rate (ESR), thyroid screen, and a TB test. The first radiographic sign is the reduced size of the femoral ossification nucleus (50% of cases), then fissuring and fracture of the femoral ossific nucleus (seen in the frog-leg view). The third sign is flattening and sclerosis of the femoral ossific nucleus. Soft-tissue swelling may be noted on the lateral side of articulation (i.e., capsular bulging with displacement of fat pad). A residual sign is a radio-dense curvilinear shadow ("sagging rope" sign). Catterall's classification is based on the amount of involvement of the capital femoral epiphysis, with prognosis related to extent of disease (i.e., those in group I have favorable outcomes, but 75% of group IV have poor results):

- Group I — Partial or less than half of head is involved; no metaphyseal reaction, no sequestrum (dense collapsed segment), no subchondral fracture line.
- Group II — More than half of the head is involved; sequestrum present, anterolateral metaphyseal reaction, anterior subchondral fracture line.
- Group III — More than half of the head is involved; large sequestrum, diffuse anterolateral metaphyseal reaction, posterior subchondral fracture line.
- Group IV — The entire epiphysis is involved; diffuse metaphyseal reaction and posterior remodeling.

VII. Special Studies

A bone scan may show areas of "cold" (decreased uptake) or "hot" (increased uptake) in the lateral two thirds of the femoral head. MRI shows the extent of damage, especially early, and is more sensitive than the bone scan; occasional false negatives occur.

VIII. Diagnosis

Legg–Calve–Perthes disease.

IX. Differential Diagnosis

Slipped capital femoral epiphysis, acute irritable hip syndrome, chronic hip syndrome, epiphyseal dysplasia, toxic synovitis, tumors, tuberculosis, juvenile rheumatoid arthritis, osteochondroma, bone cyst, rheumatic fever, hypothyroidism, sickle cell anemia, Gaucher's disease, eosinophilic granuloma, infection.

X. Treatment

Prompt referral to an orthopedic surgeon. Generally, mild, young cases need no specific treatment and are permitted full ambulation and activities of daily living; other groups may require orthotics, braces, and/or surgery. Exercise prescription should be modified to permit nonweight-bearing activities (e.g., swimming) and strengthening of glutei and quadriceps muscles. The only evidence-based factors that are of prognostic importance in the long term are the age of the patient at onset and the shape of the femoral head at skeletal maturity (Level of Evidence A, meta-analysis).[13]

XI. Complications

Complications increase exponentially in children over age 8 years; osteochondritis dissecans can be seen in 2 to 4% of cases (mostly male).

XII. Prevention

None; early diagnosis is important.

NEUROLOGICAL PROBLEMS

LATERAL FEMORAL CUTANEOUS NERVE COMPRESSION (MERALGIA PARESTHETICA)

I. Epidemiology
More common in males than females, predominantly in middle-aged or overweight persons.[7]

II. Mechanism of Injury
Direct blunt trauma; indirect repetitive microtrauma resulting in increased tension (e.g., excessive pelvic tilt, obesity, coughing, tight belt/equipment/undergarment). May be spontaneous (usually compression) or iatrogenic due to orthopedic procedures such as iliac crest bone graft harvesting.

III. Anatomy
The lateral femoral cutaneous nerve supplies the proximal two thirds of the lateral thigh in a purely sensory function. Compression or entrapment of the nerve can occur as it crosses the inguinal ligament at the level of the ASIS.

IV. Symptoms
Paresthesias and hypesthesia along the proximal anterolateral thigh, aggravated by prolonged standing and walking and relieved by rest with the hips flexed.

V. Signs
Blunting of sensation to pinprick and light touch over the anterolateral thigh. No muscle weakness or changes in deep tendon reflexes will be observed.

VI. X-Rays/Laboratory
Not indicated, unless associated with trauma to rule out fracture.

VII. Special Studies
Electromyography and NCV to rule out lumbar root disease. A useful diagnostic test is a nerve blockade with marcaine medial to ASIS. Complete relief of symptoms is diagnostic.

VIII. Diagnosis
Lateral femoral cutaneous nerve entrapment (meralgia paresthetica).

IX. Differential Diagnosis
Avulsion fracture of ASIS, contusion, lumbar root disease (L2 and L3).

X. Treatment
Rest; injection therapy may be of benefit in chronic cases. Occasionally, exploratory surgery with neurolysis or surgical decompression may be necessary for chronic disability. Consultation is necessary for chronic dysfunction.

XI. Complications
Chronic pain.

XII. Prevention
Controlling risk factors such as obesity, strengthening abdominal muscles to decrease pelvic tilt, and better fitting of equipment and undergarments.

FEMORAL NERVE ENTRAPMENT

I. Epidemiology
None available.

II. Mechanism of Injury
Usually associated with inguinal hernia, retroperitoneal hematoma; the most common cause of femoral neuropathy is diabetic amyotrophy.

III. Anatomy
The site of greatest risk of compression is at the inguinal ligament, as the femoral nerve passes beneath the ligament, although entrapment is not common.[7]

IV. Symptoms
Groin pain that is increased with hip extension.

V. Signs
Wasting or weakness of the quadriceps muscle and sensory impairment over the anteromedial thigh.

VI. X-Rays/Laboratory
Anteroposterior, lateral pelvic, and hip radiographs to rule out other pathology.

VII. Special Studies
Electromyograph and NCV studies to document a neurapraxia of the femoral nerve.

VIII. Diagnosis
Compression of the femoral nerve.

IX. Differential Diagnosis
Disuse atrophy, proximal myopathy, inguinal hernia, quadriceps strain.

X. Treatment
Treat underlying cause.

XI. Complications
Chronic disability, weakness of knee/leg extension.

XII. Prevention
None.

MISCELLANEOUS

TRANSIENT/TOXIC SYNOVITIS

I. Epidemiology
This is the most common cause of painful hip in children. It is also known as observation hip, intermittent hydrathosis, and coxitis serosa. Rarely is this condition bilateral. Hip synovitis in adult runners is extremely rare and is probably confused with other disorders of the hip.

II. Mechanism of Injury
It may be related to trauma but without one consistent etiology (viral infection, allergic reaction, trauma).

III. Anatomy
Refer to Figures 49.1 and 49.2.

IV. Symptoms
Half have acute, painful hip with a limp; 50% are insidious.

V. Signs
Positive log roll indicating an irritable hip, muscle spasm around the hip, voluntary limited range of motion (usually resting in flexion, abduction, and external rotation).

VI. X-Rays/Laboratory
Complete blood count and ESR are usually normal. Plain films of the hip help rule out fracture, SCFE, or Legg–Calve–Perthes disease.

VII. Special Studies
Arthrocentesis is used to rule out septic hip. Ultrasound may show hip effusion and capsular distension. Bone scan with Tc99m should not be routinely used unless symptoms persist and do not respond to adequate periods of rest and traction.

VIII. Diagnosis
Transient (toxic) synovitis of the hip.

IX. Differential Diagnosis
Septic arthritis, Legg–Calve–Perthes disease, rheumatoid arthritis, slipped capital femoral epiphysis; in adults, rule out iliopsoas tendinitis, chronic adductor tendinitis, and rectus femoris strain.

X. Treatment
Rest in position of comfort (occasionally may use light Buck's traction with a pillow under the knee); traction for immediate relief.

XI. Complications
Prolonged inflammation may result in coxa magna, osteoarthritis, widening of femoral neck, joint space narrowing, and cystic changes.

XII. Prevention
None.

OSTEITIS PUBIS

I. Epidemiology
Generally seen in distance runners, soccer and hockey players, and race walkers.[8]

II. Mechanism of Injury
Repetitive microtrauma causing fatigue failure within the ligament at its insertion into the pubic rami at the symphysis.[8]

III. Anatomy
Inflammatory condition of pubic symphysis.

IV. Symptoms
Unilateral or bilateral groin or symphysis discomfort increased with activity and relieved with rest; waddling gait from spasm of adductor muscles.

V. Signs
Tenderness of the symphysis pubis with palpation and compression, painful active scissor kick, waddling gait.

VI. X-Rays/Laboratory
Anteroposterior views may show loss of definition of the margins, localized osteopenia, widening of the symphysis, and periosteal reaction. Instability of the pubis may be demonstrated with the flamingo view (or Chamberlain technique); a one-leg stance will show >3-mm displacement of pubic symphysis when compared to the contralateral side. The diagnosis of osteitis pubis can be difficult because radiographic appearances of this entity are commonly identified in the absence of symptoms.[14]

VII. Special Studies
Bone scan has high sensitivity but low specificity. Some physicians have used symphyseal cleft injection of steroid and analgesic as both a diagnostic tool and treatment modality (Level of Evidence B, case controlled).[15] Studies on this technique, however, are limited and are comprised mostly of case-based reviews.

VIII. Diagnosis
Osteitis pubis.

IX. Differential Diagnosis
Inguinal hernia, prostatitis, orchitis, urolithiasis, groin pull, ankylosing spondylitis of the pubic symphysis.

X. Treatment
A. Short-term: Treat with RICE and NSAIDs.
B. Long-term: Physical therapy should emphasize abdominal and adductor exercises and a graduated reconditioning program. Gravid patients need supportive care.

Consultation: Long-standing refractory cases may need possible wedge resection of the symphysis pubis, but this is rare;[16] although research on symphyseal cleft injection may be promising and it may offer an alternative, it is limited.

XI. Complications
Persistent problems refractory to conservative treatment.

XII. Prevention
Flexibility, conditioning, and adequate warm-up.

SNAPPING HIP
I. Epidemiology
Appears most commonly in young female runners or dancers, gymnasts, and hurdlers.

II. Mechanism of Injury
Activities with high repetition that require a twisting motion.

III. Anatomy
An internal snapping is secondary to relative tightness of the iliopsoas muscle as the tendon subluxes over the iliopectineal eminence during flexion and extension.[17] The external snapping hip is attributed to the relative tightness of the iliotibial band or gluteus maximus tendon moving over the greater trochanter.

IV. Symptoms
Dull hip pain associated with an audible snapping of the hip during exercise or pain associated with snapping in the medial groin with flexion, abduction, and external rotation.

V. Signs
Palpable and audible snapping occurring with FABERE maneuver (see Figure 49.4).

VI. X-Rays/Laboratory
Anteroposterior and lateral X-rays of the hip and pelvis are generally normal.

VII. Special Studies
For external snapping hip, dynamic sonography can show real-time images of sudden abnormal displacement of the iliotibial band or gluteus maximus over the greater trochanter (Level of Evidence B, review).[18] Although these reports are based on a small number of patients, the results are encouraging. It is rarely indicated, but iliopsoas bursography with fluoroscopy for definitive demonstration of the snapping tendon may be considered in institutions with that capability. MRI may show an enlarged iliopsoas tendon on the affected side.

VIII. Diagnosis
External snapping hip syndrome (iliotibial band), internal snapping hip syndrome (iliopsoas).

IX. Differential Diagnosis
Iliotibial band or gluteus maximus tendons may snap over the greater trochanter.

X. Treatment
A. Initial: Treat with RICE, NSAIDs, and relative rest.
B. Long-term: Use injection therapy (requires familiarity with anatomy and technique); modify exercise prescription; use hip ROM and PREs.

Consultation: surgical release for refractory cases

XI. Complications
Chronic disability.

XII. Prevention
Slowly progressive training regimen and increased flexibility of the hip with flexion, extension, external and internal rotation, and abduction and adduction exercises.

PIRIFORMIS SYNDROME

I. Epidemiology
Sports that require excessive use of the muscles of the buttock (e.g., ice/roller skaters, cyclists, mountain climbers) are more likely to develop this syndrome. The female to male ratio is 6:1.

II. Mechanism of Injury
One half of cases indicate history of trauma that results in spasm or hypertrophy of the piriformis muscle.[19] The other 50% are idiopathic.

III. Anatomy
The piriformis muscle arises from the pelvic surface of the sacrum and generally fills the sciatic foramen as it passes through and inserts onto the superomedial edge of the greater trochanter. Its action is external rotation of the hip. Typically, the sciatic nerve courses inferiorly beneath the piriformis muscle. In 15% of people, the sciatic nerve penetrates the piriformis muscle and can be strangulated with spasm of the muscle.[19]

IV. Symptoms
Intense aching deep within the buttock, with occasional pain radiating to the back or thigh. Pain worsens with prolonged sitting, such as in a church pew or cycling. The positions that ameliorate the pain include lying prone, floating on water, or the lateral decubitus position. Women may also complain of dyspareunia.

V. Signs
Listing stance and antalgic gait, sciatic notch tenderness, and tenderness and spasm with resisted abduction/external rotation of the hip. Pain increases with passive internal rotation, generally full range of motion (FROM) of the lumbar spine, but may see positive straight leg raise and palpable trigger point proximal to ischial spine on pelvic/rectal exam. Also may see positive Freiberg's and Pace's maneuvers (see Physical Exam section).

VI. X-Rays/Laboratory
Anteroposterior, lateral pelvis, and hip views can rule out other pathologies.

VII. Special Studies
Electromyograph and NCV are helpful to document sciatic nerve involvement in chronic cases. Other diagnostic techniques are emerging, such as the measurement of cauda equine potentials and prolongation of the H-reflex with flexion, adduction, and internal rotation (FAIR) test (Level of Evidence C, opinion).[20]

VIII. Diagnosis
Piriformis syndrome.

IX. Differential Diagnosis
Ischial tuberosity bursitis (weaver's bottom), pelvirectal and ischiorectal abscesses, inflammatory bowel lesions, herniated lumbar discs (L4–L5, L5–S1), myositis ossificans, avulsion fracture, facet syndrome, spinal stenosis.

X. Treatment
A. Initial: Treat with rest, analgesics, NSAIDs, occasional muscle relaxants; treat spasm with ice massage and slow, sustained stretching.
B. Long-term: Use injection therapy, application of transcutaneous electrical nerve stimulator (TENS) units to fatigue the muscle followed by PREs for hip girdle muscles and gluteal stretch exercises (e.g., single knee to chest stretch); modify the exercise prescription to encourage cross-training for one-sport athletes (e.g., cyclists to run or swim). Recent case reports indicate improvement with the use of a CT-guided BOTOX injection as compared to saline or corticosteroid injection.[21]

Consultation: surgical release for sciatic entrapment

XI. Complications
Chronic disability.

XII. Prevention
Protective padding for the cyclist, frequent change of position, and adequate flexibility and warm-up.

ACETABULAR LABRAL TEARS

I. Epidemiology
Initially, they were thought to occur only with posterior dislocations but they may also be related to subluxation and acetabular dysplasia.[22]

II. Mechanism
Acetabular dysplasia results in a lateral uncovering of the femoral head and places stress on the lateral aspect of the labrum. In subluxation, the abnormal motion of the femoral head causes increased stress on the labrum.

III. Anatomy
The acetabular labrum is a horseshoe-shaped fibrocartilaginous structure attached to the periphery of the acetabulum that adds depth to the hip joint.[23] Inferiorly, the labrum continues as the transverse ligament from the anterior margin to the posterior margin of the acetabulum.

IV. Symptoms
Patients tend to report groin pain or an audible or palpable click, along with mild limitation of motion. Pain may be exacerbated when rotating the hip to change from a seated to a standing position. The patient may recall a twisting or slipping injury.

V. Signs
On physical exam, an anterior labral tear can be identified with a sharp, catching pain when the externally rotated, flexed, and abducted hip is extended with internal rotation and adduction.[24] Patients with a posterior labral tear experience sharp pain with passive flexion, internal rotation, and a posterior load.[24] Usually limited ROM and a positive FABERE test are noted.

VI. X-Rays/Laboratory
Conventional MRI has not proven successful in detecting labral injuries because considerable variability in the MR appearance of the labrum exists in asymptomatic patients (Level of Evidence B, review).[25] MRI arthrography has been shown to identify 88% of labral tears while concurrently enabling injection of local anesthetic.[26] The most definitive evidence for labral tear is tracking of contrast into the substance of the labrum. This does account for differentiation between a normal sublabral sulcus vs. injury to the labrum. Clinical correlation and intraarticular anesthetic may be helpful. In some cases, the diagnosis is not made until the time of arthroscopy.[27]

VII. Special Studies
Not applicable.

VIII. Diagnosis
Acetabular labral tear.

IX. Differential Diagnosis

X. Treatment
Partial weight-bearing for 4 weeks. Local anesthetic injection may be therapeutic. Those who do not respond to nonoperative treatment may be offered arthroscopic excision of the torn portion of the labrum.

XI. Complications

XII. Prevention

OSTEOARTHRITIS

I. Epidemiology
The age-old debate as to whether sports participation predisposes a person to osteoarthritis (OA) or actually protects against it has not been clearly solved. The generally held belief is that regular, moderate activity is helpful whereas excessive loading, particularly with an underlying injury or abnormality, accelerates the degenerative process. Elite athletes have been shown to have more radiographic changes of OA than matched controls (Level of Evidence A, randomized controlled trial).[28] In an analysis of patients who underwent total hip arthroplasty, patients with high exposure to sports when combined with jobs that require much physical endurance had a greatly increased relative risk of OA.[29]

II. Mechanism
Osteoarthritis may be primary or secondary. Secondary may be due to trauma, infection, crystal deposition disease, osteonecrosis, or acetabular dysplasia.

III. Anatomy

IV. Symptoms
Pain and stiffness of the affected joint is worsened by activity. Slow and insidious onset; morning stiffness of less than 30 minutes' duration.

V. Signs
There may be swelling. Passive and active range of motion is reduced. Crepitus may be elicited with joint movement.

VI. X-Rays/Laboratory
Initial plain radiograph findings follow a continuum. Initially, nonuniform joint space narrowing from cartilage loss is noted, then, as joint space loss progresses, reactive bone formation (subchondral

sclerosis and osteophytosis) occurs.[30] Osteophytes and subchondral cysts may be noted in later stages. CT may be useful for detecting loose bodies. MRI has had poor success evaluating cartilage thickness and demonstrating cartilage abnormalities of the femoral head and acetabulum.[31]

VII. Special Studies
Not applicable.

VIII. Diagnosis
Osteoarthritis of the hip.

IX. Differential Diagnosis

X. Treatment
Range of motion exercises, NSAIDS, non-weight-bearing activity (e.g., swimming). Surgical consultation for progressive pain and disability.

XI. Complications
Progressive disability; loss of ADLs.

XII. Prevention
Protection from micro/macrotraumas.

HERNIA

An inguinal hernia in the competitive athlete may mimic other pelvic pathology, with twinges of pain during exertion and a dull ache in the groin after exercise. Football and weightlifting are two of many sports in which increased intraabdominal pressures with exertion over time can weaken or tear the musculofascial layer of the inguinal canal. Diagnosis is made from clinical suspicion and physical findings consistent with scrotal or inguinal mass that increases with intraabdominal pressure. Occasionally herniography is employed to show leakage within the peritoneal cavity to make this diagnosis. Important differential diagnoses include ilioinguinal or genitofemoral nerve neuralgia, tenoperiostitis, symphysitis, and prostatitis. Treatment requires referral for herniorrhaphy, with precautions against premature return to play, which may increase the risk of recurrence.

SUMMARY

Although the pelvis is not the most frequently injured site on the body, it is important to understand the form and function of the many muscles and nerves that comprise this focal point of upper and lower extremity balance. By having a baseline knowledge of the pelvis anatomy and the muscle attachments and function, along with taking a systematic approach to history-taking and examination, the examining physician will be able to make an accurate diagnosis, provide a specific and progressive program of rehabilitation, and allow the athlete to return to active play in a timely and safe manner. Along with the systematic approach, it is helpful to view specific diagnoses within the outline suggested here: acute vs. chronic vs. developmental; soft tissue vs. bony; and other, noncategorized problems. This outline aids in confidently making the diagnosis while exploring all the possibilities.

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50 THIGH INJURIES*

Richard Levandowski and Philip Cohen

INTRODUCTION

Injuries to the thigh often do not have the same dramatic qualities as other sports injuries. Yet, many competitive athletes have experienced the disability of a thigh strain, bursitis, or contusion during their careers. Unfortunately, the diagnosis and treatment of these injuries may not be as aggressive as with other athletic conditions. As a result, long-lasting limitations of function and prolonged recovery periods can occur. Therefore, it is critical that the physician evaluating an athlete with a thigh injury fully understand the relevant anatomy, the injuries that occur in the region, and the appropriate diagnostic and therapeutic regimen.

TROCHANTERIC BURSITIS

I. Epidemiology

Trochanteric bursitis is a common pain syndrome of the thigh. It comprises approximately 2 to 3% of all injuries in runners[1,2] and is associated with poor flexibility, leg length discrepancy, and low percentage of body fat.

II. Mechanism

The injury often develops due to a tendinitis of the abductors secondary to overuse in runners and dancers. The injury can also occur due to a direct blow over the lateral aspect of the greater trochanter.[3]

III. Anatomy

The trochanteric bursa lies between the iliotibial tract and the greater trochanter, and it acts to reduce friction between these two surfaces; however, when the bursa becomes inflamed, the space surrounding the bursa becomes filled with bursal fluid, resulting in both swelling and pain (Figure 50.1).

IV. Symptoms

The patient may complain that the hip hurts. Pain may radiate to the level of the knee, resulting in limping and difficulty in standing.

V. Signs

Tenderness to direct palpation is noted over the superolateral aspect of the greater trochanter and associated tendons; passive range of motion may be intact; pain can be elicited through several methods:

- Ober's test, in which the patient lies on his side with the injured leg toward the ceiling; the thigh is then abducted with the knee flexed at 90° and the hip slightly extended
- Stretching of the tendons associated with the greater trochanter by having the patient lie on his back and bringing the ipsilateral knee to the opposite shoulder

The feet, ankles, and knees should also be examined for malalignment, leg length discrepancy, and hyperpronation.

* In the second edition, this chapter was authored by Richard Levandowski and John P. Difiori.

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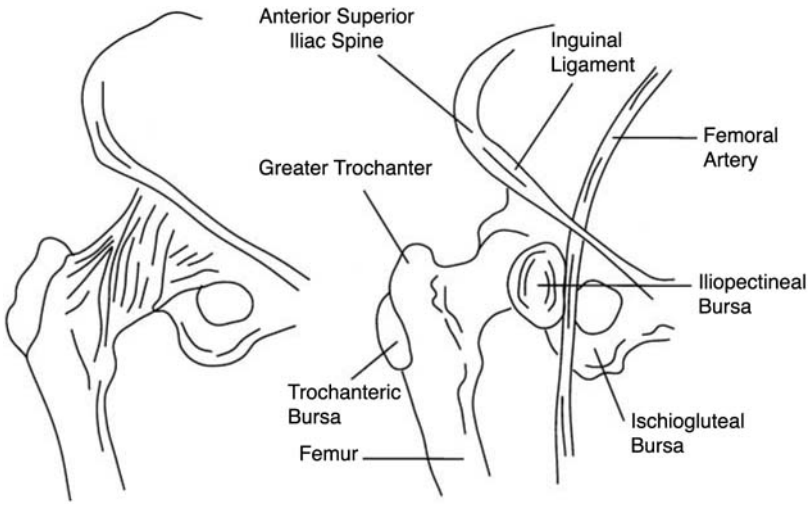

Figure 50.1 Anterior aspect of the hip, illustrating the trochanteric, iliopectineal, and ischiogluteal bursae.

VI. X-Rays
X-rays usually are not necessary. Anteroposterior (AP) and frog-leg lateral hip films may demonstrate soft-tissue edema and calcification; avulsion fractures may also be detected.

VII. Special Studies
Additional studies usually are not indicated; consider bone scan or magnetic resonance imaging (MRI) if patient is not responding or atypical features suggest stress fracture, hip joint pathology, or neoplasm.

VIII. Diagnosis
Trochanteric bursitis.

IX. Differential diagnosis
Avulsion fracture, sciatica, fibromyalgia, inflammation of the gluteus medius, leg length discrepancy, hip joint pathology, neoplasm.

X. Therapy
A. Initially: Treat with RICE (rest, ice, compression, elevation), nonsteroidal anti-inflammatory drugs (NSAIDs), and analgesics, as needed. A cane used in the opposite hand may be useful in patients with a limp.
B. Long-term: Physical therapy should include range of motion (ROM) and progressive resistance exercise (PREs) aimed at hip abductors and external rotators. If the patient fails to improve within 3 to 4 days, and/or continues to limp, phonophoresis and possibly corticosteroid injection in skeletally mature patients may be helpful (see Chapter 28).

Consultation: chronic bursitis or bursal calcification

XI. Complications
See Chapters 27 and 34.

XII. Prevention
Increased flexibility and strength of all the muscles of the hip and thigh, correction of leg length discrepancy, control of abnormal foot motion with an appropriate orthotic device, appropriate padding in contact/collision sports.

THIGH CONTUSIONS
I. Epidemiology
Thigh contusions are probably the most common thigh injury and are found in all contact/collision sports, particularly football, basketball, soccer, and ice hockey.[4–7]

II. Mechanism of Injury
Blunt trauma.

TABLE 50.1
Thigh Contusions

Degree of Flexion	Severity of Injury
>90°	Grade I
>45°, <90°	Grade II
<45°	Grade III

Source: Jackson, D.W. and Feagin, J.A., *J. Bone Joint Surg. (Am.)*, 55A(1), 96, 1973. With permission.

III. Anatomy
The quadriceps muscles are most commonly injured, although hip abductors and the iliotibial tract/band are frequently involved.

IV. Symptoms
Pain localized to the anterior thigh (or other area of contusion) and often limping.

V. Signs
As described in Chapter 27; knee flexion is limited and is a predictor of injury severity (Table 50.1).

VI. X-Rays
See Chapters 27 and 34.

VII. Special Studies
None indicated initially; x-rays and/or MRI in chronic cases to evaluate for myositis ossificans.

VIII. Diagnosis
Thigh contusion (charley horse).

IX. Differential Diagnosis
Osteogenic sarcoma, thigh strain.

X. Therapy
If the physician is present at the time of the injury, the leg should be immediately flexed and iced while in flexion. This will help to prevent muscle shortening and may decrease hematoma size. The leg may then be bound in flexion.

Gradual return to weight-bearing, with a gentle, progressive physical therapy regimen is recommended. Aggressive passive range of motion or returning to activity too early may hamper healing and increase the likelihood of complications, such as myositis ossificans. Despite some reports of positive effects, the literature does not conclusively prove NSAIDS, corticosteroids, or other agents are beneficial in these settings. The physician must balance the need for analgesia and promotion of normal function with the need to avoid further bleeding and allow a certain amount of inflammatory response which is felt to be necessary for normal healing. The use of protective padding may prevent recurrent injury but must not be seen as a short-cut to return to play before the athlete is truly ready.

XI. Complications
Myositis ossificans, ischemic necrosis.

XII. Prevention
See Chapters 27 and 34.

THIGH STRAIN
I. Epidemiology
These injuries are common in running, skating, and jumping sports (7 to 12% of injuries) and are associated with dynamic muscle imbalance due to poor strength and flexibility, inadequate warm-up, and muscle fatigue.[1,2,4,5,8–10] Hamstring strains are twice as common as quadriceps strains.

II. Mechanism
See Chapters 27 and 34.

III. Anatomy
Anterior thigh strain ("quad" strain) involves the quadriceps muscles: the rectus femoris, vastus medialis, vastus lateralis, or vastus intermedius (Figure 50.2). Posterior thigh strain (hamstring strain) involves the three major posterior thigh muscles: biceps femoris, semitendinosus, and semimembranosus (Figure 50.3). These muscles function as knee flexors and are thus opposed by the quadriceps. The quadriceps-to-hamstring strength ratio has been found to be approximately 1.5:1. Athletes with a dynamic imbalance (that is, a quadriceps to hamstring strength ratio greater than 1.5:1) are more susceptible to hamstring pulls.[10] The medial compartment muscles, including adductor longus, adductor brevis, and adductor magnus, may also be involved in thigh strains.

IV. to X. Symptoms, Signs, X-Rays, Special Studies, Diagnosis, Differential Diagnosis, Treatment
See Chapters 27 and 34.

XI. Complications
Avulsion fractures, myositis ossificans, recurrent injury, and complete rupture; consultation necessary for third-degree strain, suspected tendon rupture, or if hematoma evacuation is needed.

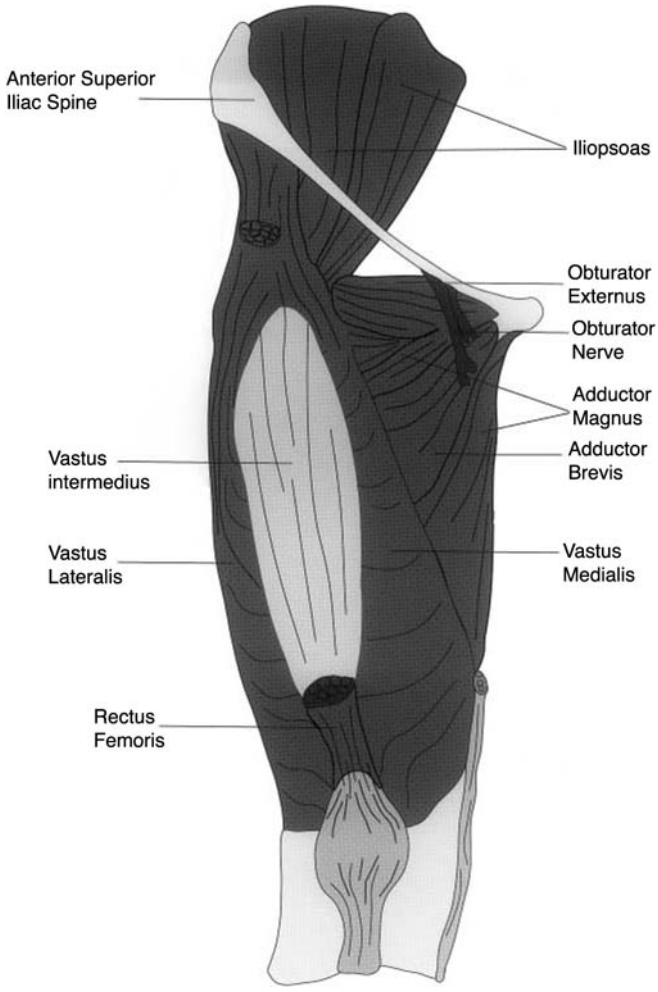

Figure 50.2 Anterior and anteromedial thigh muscles (right thigh). Part of the rectus femoris and the pectineus, sartorius, gracilis, adductor longus, and tensor lata not shown.

XII. Prevention

Correction of dynamic muscle imbalance, especially in hamstring pulls, by stretching and strengthening of anterior and posterior muscle groups (quad extension and hamstring curls).

FEMORAL STRESS FRACTURE

I. Epidemiology

An overuse injury that often is due to excessive downhill or mountain running and is more common in jumping sports. Studies show that 10 to 25% of stress fractures involve the femur.[11,12]

II. Mechanism

Repetitive stress; compression, where the fracture line is more horizontal to the trabeculae; distraction, where the fracture line is perpendicular to the trabeculae of the femoral neck.

III. Anatomy

Femoral stress fractures can occur in the femoral neck or shaft. The femoral neck was the most common site in one study.[12]

IV. Symptoms

Progressive anterior hip/groin pain (femoral neck fractures) or thigh pain beginning as early as 2 weeks after initiating or changing a conditioning program; night pain is present in 20% of patients.

V. Signs

Pain with passive range of motion (especially internal rotation) for femoral neck stress fractures; pain

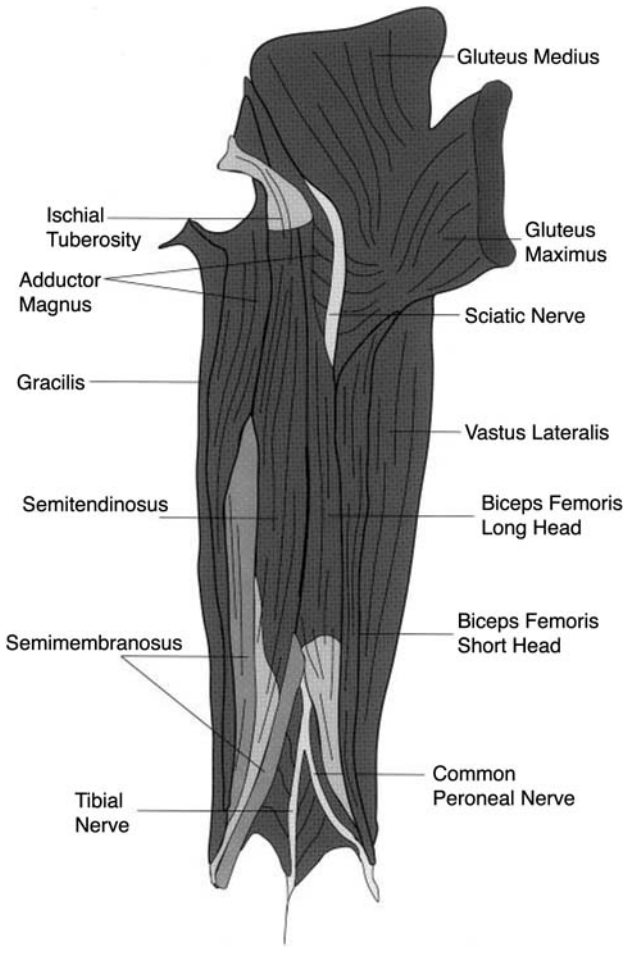

Figure 50.3 Posterior thigh muscles (right thigh).

with impact activity; tenderness to palpation variable; pain with heel percussion or passage of an ultrasound device over the injury site; shortening and external rotation of the hip with a displaced fracture.

VI. X-Rays

Routine films (AP and lateral) may be negative until 2 to 6 weeks after injury.

VII. Special Studies

Triple-phase bone scan (most sensitive) will be positive 1 week after injury but it will be 2 or more weeks before changes are present on plain films. MRI is more specific than bone scan but is also more costly (Level of Evidence, review).[13]

VIII. Diagnosis

Femoral stress fracture.

IX. Differential Diagnosis

Osteogenic sarcoma, osteoid osteoma, groin/quadriceps strain, athletic pubalgia/sportsman's hernia, avascular necrosis, osteitis pubis, patellofemoral pain syndrome (in supracondylar fractures).

X. Treatment

A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; non-weight-bearing.
B. Long-term: Further evaluation and treatment for femoral neck stress fractures:
   Nondisplaced fracture — (1) *Compression side* (inferior medial cortex) fractures may be treated conservatively with close follow-up (Level of Evidence, review).[14] Initial treatment is strict rest until patient is pain free, then non-weight-bearing ambulation with

crutches. Serial radiographs are recommended every 2 to 3 days for the first week, followed by weekly radiographs until healing is documented. Operative intervention is necessary if the fracture widens, both cortices develop a defect, or if the patient is uncooperative or unreliable. Allow 6 months for recovery (Level of Evidence, review).[11,15,16] (2) *Tension side* (superior lateral cortex) fractures require *urgent* referral for consideration of operative intervention.

Displaced fracture — Refer for immediate open reduction internal fixation (ORIF); high rate of complications, including AVN and nonunion (Level of Evidence B, longitudinal non-controlled case series).[14,17]

Consultation: Advisable early in all but the simplest fractures

C. Further evaluation and treatment for other femoral stress fractures: These injuries may occur along the femoral shaft or even at the supracondylar region of the distal femur (where they can mimic patellofemoral disorders) (Level of Evidence B, case series and review).[18] Treatment is usually conservative, with protected weight-bearing casting or bracing and physical therapy. Specific regimens vary depending upon the location and severity of the fracture.

XI. Complications

Displacement, prolonged disability secondary to pain, nonunion, and avascular necrosis. These fractures may bring about a sudden end to an athlete's career. They may also be a clue to syndromes such as the female athlete triad and underlying metabolic disorders. Appropriate screening and workup are required.

XII. Prevention

Decrease training errors by avoiding sudden increases in training intensity or frequency and avoiding running on hard surfaces. Wear proper footwear; footwear should prevent hyperpronation and provide adequate support and cushioning. Screen for athletes at high risk (e.g., abnormal biomechanics, large increase in training volume and/or intensity, female athlete triad, chronic use of systemic corticosteroids, osteoporosis) with a thorough history and physical.

SUMMARY

Little attention has been given to the rather common injuries involving the thigh. Their potentially devastating properties have not been widely recognized. The attitude that most thigh injuries should be handled by "running it off" may have serious consequences. Accurate diagnosis, aggressive treatment, and appropriate emphasis on rehabilitation will not only allow a higher level of participation at the time of return to activity but may also diminish the probability of recurrent injury.

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51 KNEE INJURIES

Richard Levandowski and Philip Cohen

INTRODUCTION

One of the most mobile joints in the body, the knee is the focal point for sports that involve running, jumping, kicking, and changing direction. It is also essential to such everyday activities as standing, walking, and climbing stairs. Injuries to the knee occur in contact and non-contact sports and are the leading cause of long-term disability from athletics.[1]

Epidemiology

One third of the sports injuries presented to outpatient sports clinics are knee injuries. The average age of athletes with problems is 21 years.[2] In

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skiing, the relative frequency of knee injuries continues to increase despite an overall reduction in the number of skiing injuries.[1] The knee accounts for 20% of the injuries in football and 13% of those in long-distance running.[2] A slight increase in knee injuries has occurred since the introduction of artificial turf. Football and other contact sports commonly produce traumatic injuries to the ligaments (9%) and menisci (7%), while the majority of knee injuries seen by the primary care physician fall into the overuse category — tendinitis (20%), apophysitis (10%), and chondropathy (10%).[2] Neurovascular injuries are rare. When they do occur, it is often the more superficial peroneal nerve on the lateral aspect of the knee near the fibular head that is injured.

Anatomy

The knee consists of four bones (femur, tibia, fibula, and patella) held together by strong ligaments, a capsule, and the muscle groups that transverse the joint. The tibia and fibula have articular cartilage at their ends. Similarly, the patella has articular cartilage on the inner border, which tracks in the femoral groove. Between the tibial and femoral articular surfaces are the lateral and medial menisci, which are biconcave, C-shaped fibrocartilaginous disks (Figure 51.1). Multiple interdigitating components make up their multilaminated structure. The blood supply comes from the periphery and only partially penetrates to the menisci. As a result, the inner portions of the menisci do not repair when injured. The menisci are attached to the joint plateau by small coronary ligaments. They function in load bearing and absorb approximately 65% of the force transmitted across the knee. They also facilitate joint stability by maintaining the relative position of the femur on the tibia. An inability to absorb shock is linked to meniscal injury and degenerative joint disease. It has also been hypothesized that nutrition to the articular cartilage itself and joint lubrication may be aided by the menisci.

Support is given by the strong capsular ligaments, most notably the medial (tibial) collateral ligament, lateral (fibular) collateral ligament, and oblique (posterior and popliteal) and arcuate ligaments (see Figure 51.1). The medial collateral ligament connects the femur and the tibia on the medial side of the knee and is intimately involved with the medial meniscus. The lateral collateral ligament supports the knee laterally from the femur to the fibula. The proximal tibial–fibular ligament joins the tibia and fibula at the tibial plateau.

Internal stability is gained by the addition of the crisscrossing anterior and posterior cruciate ligaments (see Figure 51.1). Because of their role in maintaining the stability of the knee, the cruciate ligaments, particularly the anterior cruciate ligament (ACL), are often thought of as protectors of the meniscus. A high rate of meniscal injury is associated with tears of the ACL.

Anteriorly, the quadriceps muscles (vastus medialis, vastus lateralis, vastus intermedius, and rectus femoris) join to encase the patella and then become the infrapatellar tendon (Figure 51.2). Posterior stability is enhanced by the hamstrings and the gastrocnemius/soleus/plantaris complex (Figure 51.3). Medially, the adductors and the gracilis, sartorius, and semitendinosus (pes anserine muscles) give support. The popliteus, iliotibial tract, and biceps femoris aid in maintaining lateral stability of the knee (Figure 51.4).

Mechanisms of Injury

Acute injuries to the knee may occur frequently in activities that require acceleration, deceleration, twisting, pivoting, cutting, and jumping (such as football, soccer, basketball, skiing, and racquetball). In football, for example, linemen, linebackers, and tight ends are likely to incur knee injuries. The most common knee injuries in contact sports such as football are ligament sprains, which occur when the foot is planted. If force is applied against the knee while the foot cannot be moved from its fixed position, ligament injuries are likely.

A block or tackle to the outside of the knee when the foot is fixed (valgus force) may result in the "unhappy triad": tearing of the medial stabilizing complex, the anterior cruciate ligament, and the medial meniscus (Figure 51.5). A force applied to the medial side of the knee is less common. This varus force may tear the lateral stabilizing complex, the anterior cruciate ligament, and the lateral meniscus. Force that is directed anteriorly with the knee bent may result in a tear of the anterior cruciate ligament; force that is directed posteriorly with the knee bent may result in a tear of the posterior cruciate ligament. Jumping and landing with a straight leg (hyperextension force) may tear the anterior cruciate ligament. If the knee is forced far enough posteriorly, this force may also tear the posterior cruciate ligament. An internal rotation force (common in skiing) may also result in a tear of the anterior cruciate ligament. If both cruciates are torn, along with the medial and/or lateral stabilizers, a knee dislocation may result. This is an orthopedic emergency, which is often associated with concomitant injury to the popliteal vasculature.

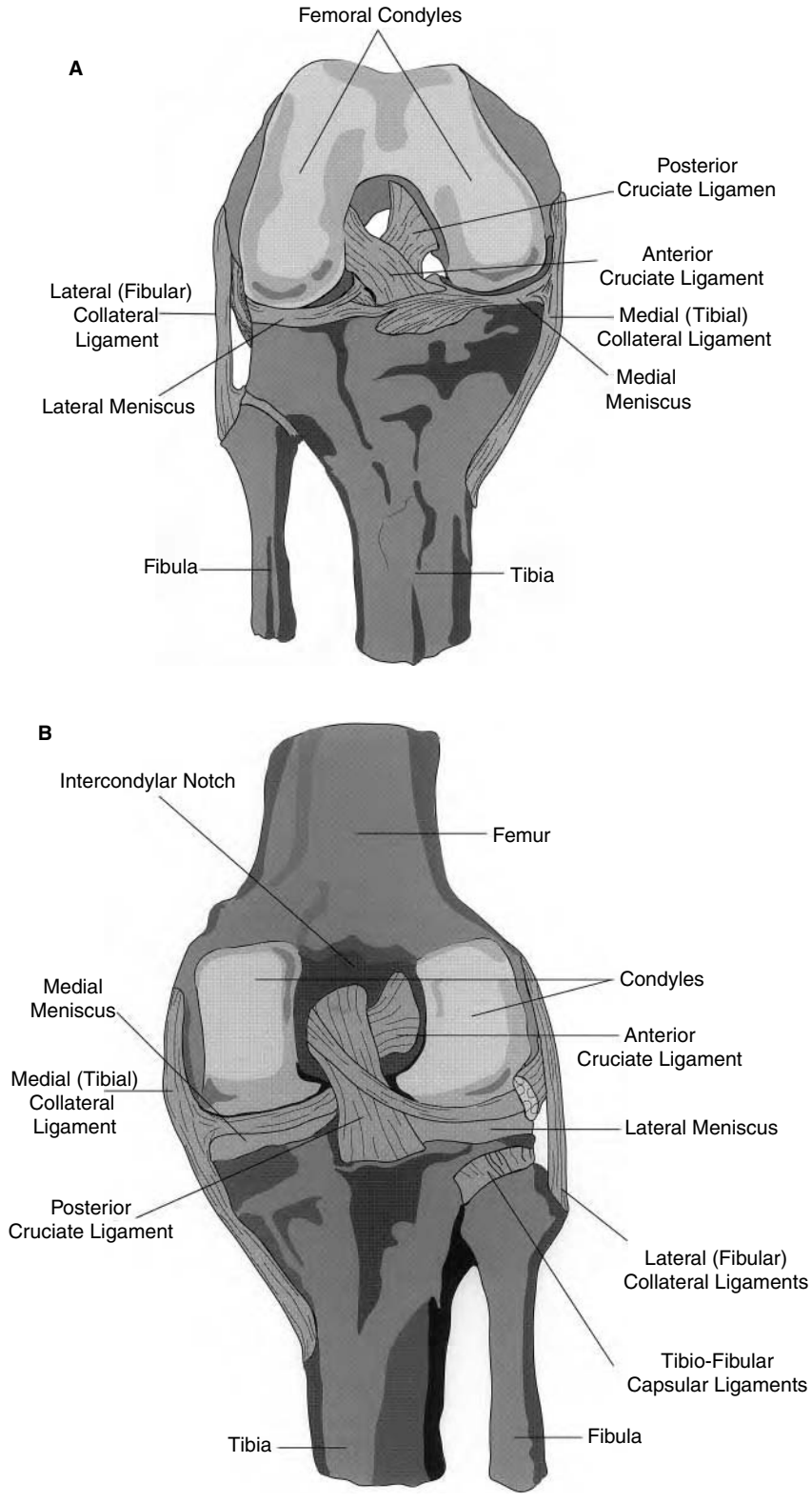

Figure 51.1 (Top) Anterior view of right knee; (bottom) posterior view of right knee.

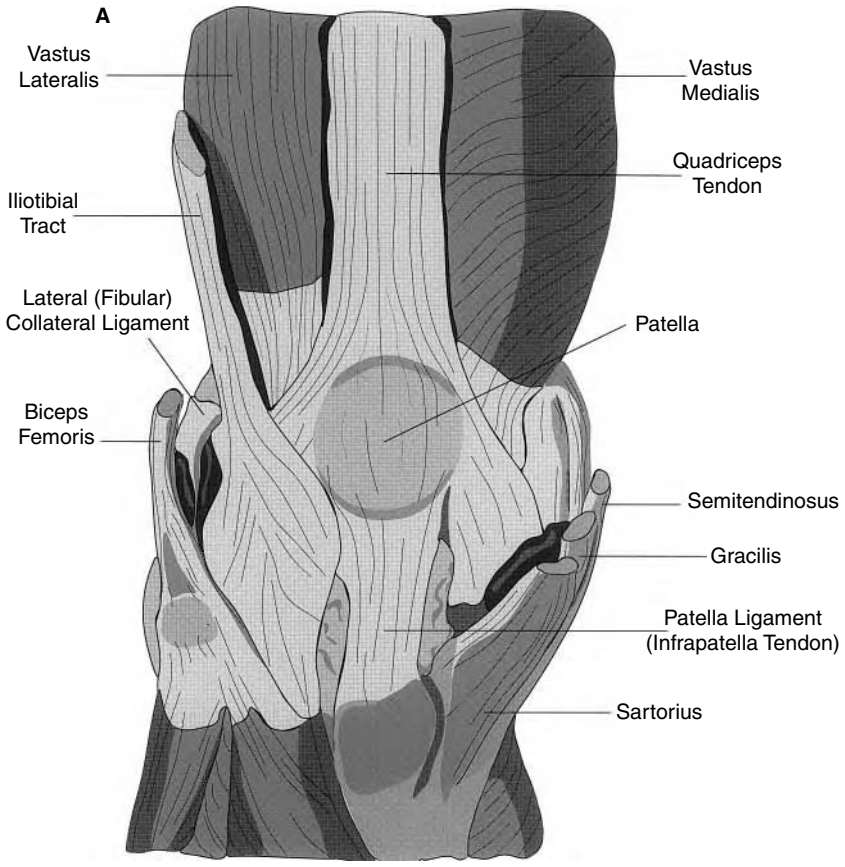

Figure 51.2 Anterior view of the knee; (facing page) lateral view of the knee.

Clinical Evaluation

History

It is crucial to perform a detailed history when evaluating a patient with a suspected knee injury. One of the most important questions to ask is if the athlete recalls a specific, acute injury. If so, the athlete should be asked to fully describe the incident, including the body positions and forces involved, the directions from which the forces came, and what happened next. Appropriate questions include:

1. Was the injury contact or non-contact?
2. What was the position of the foot of the injured limb? Was it firmly planted? Was the athlete's full or partial weight on the injured limb?
3. What was the position of the knee? Did it become hyperextended, hyperflexed, or rotated? Was a valgus or varus stress involved?
4. Did it feel like the kneecap came out of place?
5. Did it feel as though the knee itself twisted?
6. Was the athlete cutting or changing direction?
7. Was any equipment involved (such as catching the edge of a ski)?
8. Was a pop felt or heard?
9. Was the athlete able to continue playing? If not, why?
10. Could the athlete bear weight? If not, why?
11. Did the knee become swollen? If so, how much did it swell and how rapidly?
12. Has the athlete noted locking, catching, or buckling? If so, under what circumstances?

Note that true locking means the loss of knee extension due to a mechanical obstruction within

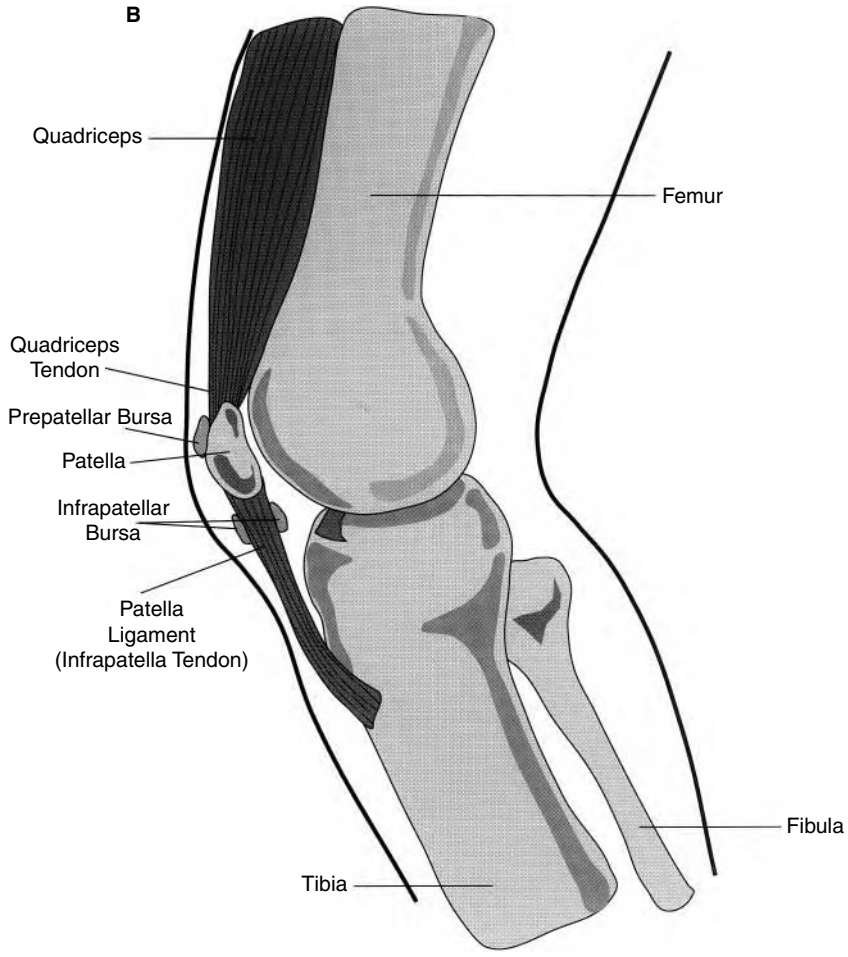

Figure 51.2 (Continued)

the joint, whereas pseudo-locking involves decreased extension caused by painful hamstring spasm. Similarly, it is important to differentiate stiffness from locking and catching, the sensation that a loose body is intermittently stuck in the joint. Finally, one must realize that true buckling occurs when the knee suddenly gives out, due to ligamentous instability or interposition of an intraarticular loose body. True buckling may be precipitated by an attempt to cut or change positions but is usually not preceded by pain. In contrast, pseudo-buckling occurs when sudden, severe pain causes reflex inhibition of the quadriceps, leading to the sensation that the knee gives out.

If the injury appears to be a chronic, rather than acute process, asking the following questions from the "S list" can be helpful:

1. What *sport*, position, or event does the athlete participate in?
2. What *shape* is the athlete in? Determine the current level of conditioning and note the duration and frequency of workouts.
3. How *serious* is the athlete — competitive or recreational?
4. What is the athlete's training and racing *speed* (pace)?
5. On what *surfaces* does the athlete participate — roads, trails, fields, beaches, or tracks?
6. Does the training surface have a *slope* (e.g., banked track)?
7. What *shoes* does the athlete wear? Inquire about the manufacturer and model and assess the wear pattern.
8. Have the athlete's *stretching* and *strength* training been adequate?

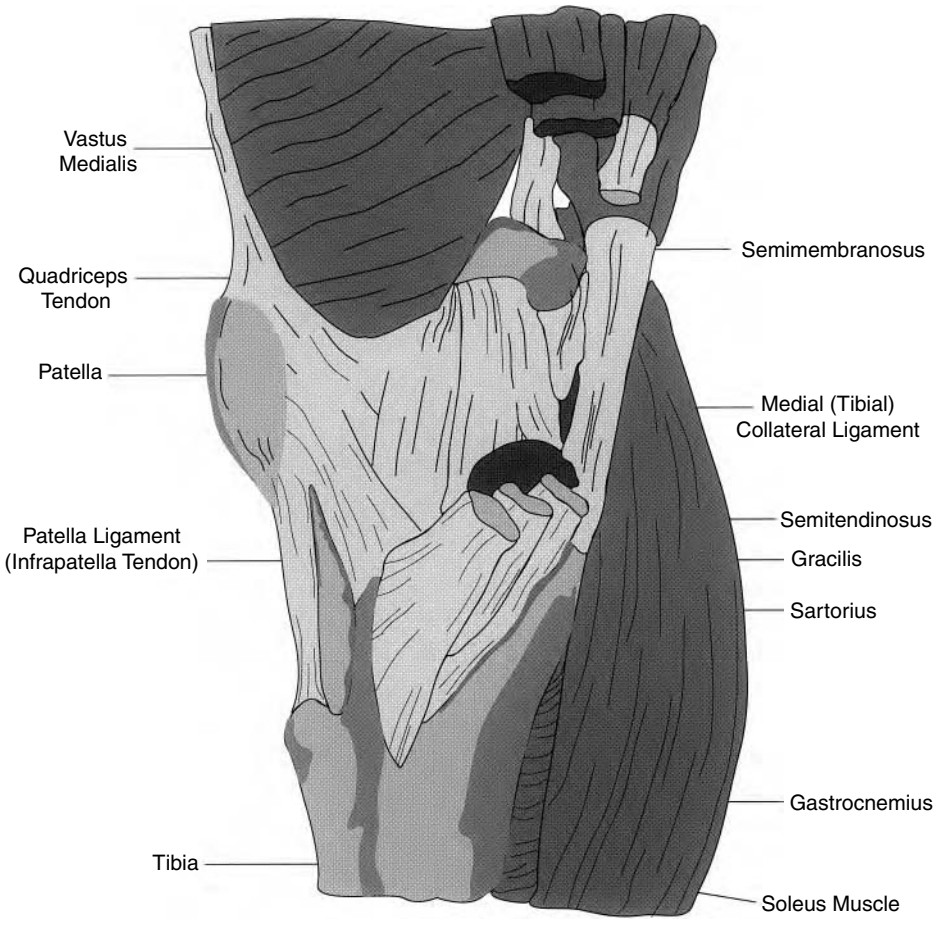

Figure 51.3 Medial view of the knee.

9. Does the athlete use any *supplements* to enhance performance (e.g., vitamins, minerals, amino acids, anabolic steroids, medications)?
10. Does the athlete use any *supports* (e.g., taping, arch supports, orthotics)?
11. Does the athlete *substitute* multiple activities (cross-training) for total body fitness, or is he or she engaged in one sport to the exclusion of others?

Regardless of the nature of the injury, it is also necessary to ask:

1. What are the current symptoms/signs that prompted the visit?
2. How long has the athlete had these complaints?
3. Does the athlete notice abnormal joint sounds, such as popping, cracking, grating, or grinding?
4. Does the athlete have any significant past medical history, including prior injury to the knee, or surgery/arthroscopy?
5. What exacerbates or alleviates the complaints?
6. Do the symptoms worsen with going up or down stairs, deep squatting, or sitting in cramped spaces with little leg room (theater sign)?
7. What has the athlete done to try to improve the situation (e.g., rested, wore a brace, used NSAIDS)?
8. Are the complaints associated only with one sport or also with other activities?
9. How does the problem impact the athlete's current training, ADLs, work, etc?

A differential diagnosis of knee pain is presented in Table 51.1.

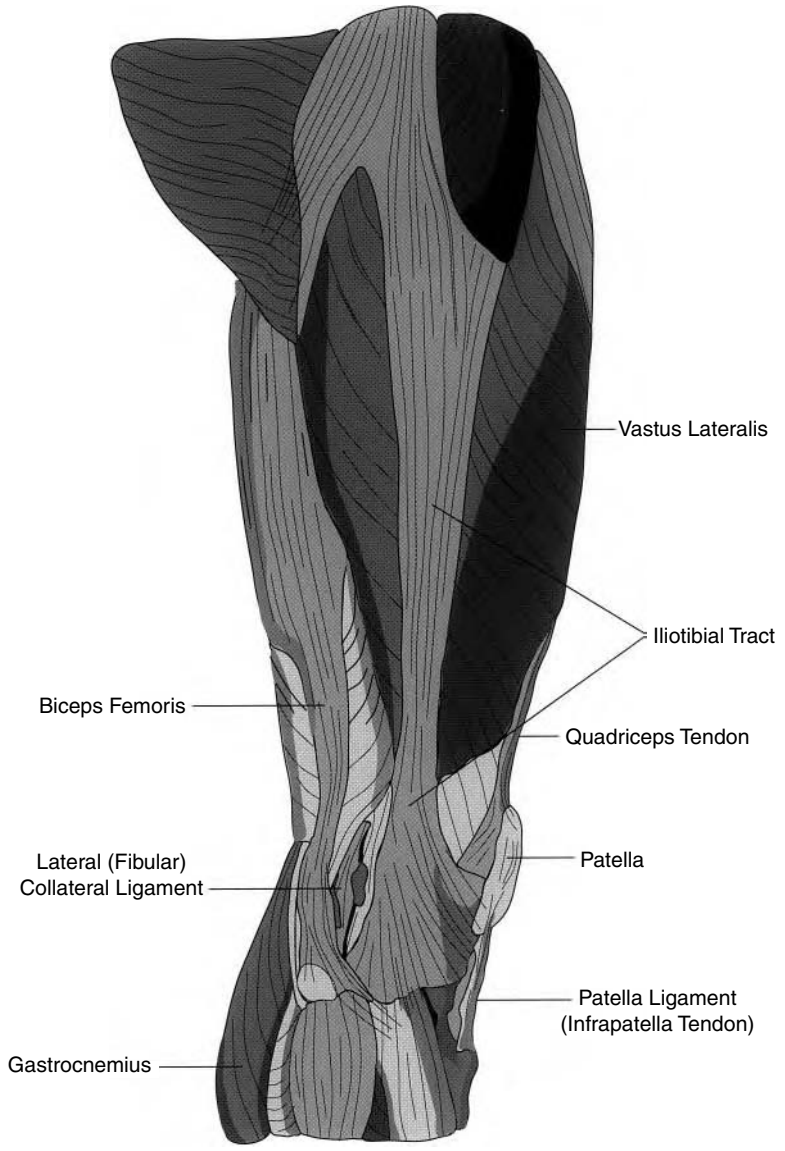

Figure 51.4 Lateral view of the knee and thigh.

Physical Examination[5]

Before examining the injured knee, check the equality, alignment, and angulation of the hips (e.g., retroversion, anteversion, and rotation), the tibia (e.g., internal or external torsion), and the leg, heel, and foot in standing, sitting, and lying positions. Examine the back for scoliosis, rotation, and flexibility. Always look carefully at the uninjured knee first to establish alignment, clinical stability, and musculature. When examining the injured knee, inspect for varus, valgus, or recurvatum deformity. Assess muscle tone and size, including the vastus medialis obliquus (VMO) and vastus lateralis (VL), and look for swelling, redness, or ecchymosis. On palpation, the patella should feel cooler than the surrounding thigh. Is patellar tenderness or tenderness over the joint line noted? Palpate for muscle tone, including the gastrocnemius–soleus, quadriceps, and hamstrings. Manually assess the quad–hamstring ratio for strength and flexibility. Manually measure and compare all muscle groups. Assess for crepitation and effusion. A true joint effusion is easily movable; extraarticular edema is boggy, thick, and not movable.

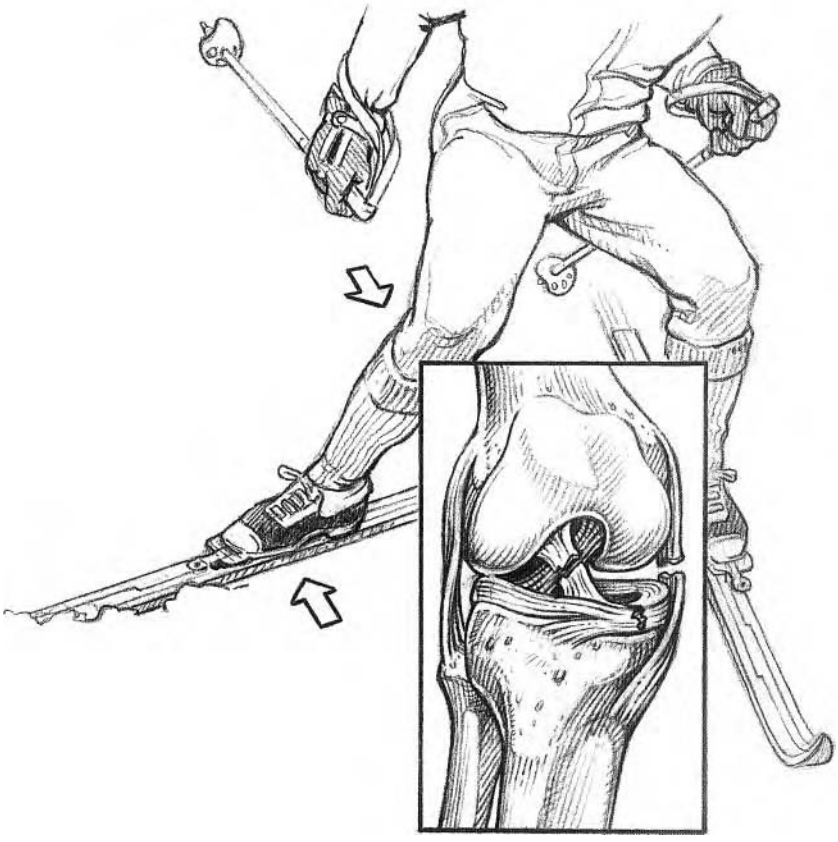

Figure 51.5 The "unhappy triad" — medial meniscus, medial collateral, and anterior cruciate tears.

Observe any limitations of normal knee flexion (135°), extension (0 to –10°), or internal (10°) and external (10°) rotation. To observe knee flexion, have the patient squat in a deep knee bend and flex both knees symmetrically. If the patient is able to stand straight with the knees in full extension, the arc of motion from flexion to extension will be smooth. Difficulty extending the knee through the last 10° of motion (extension lag) may indicate quadriceps weakness. To assess the knee's range of rotation, have the patient rotate the foot medially and laterally. Loss of lateral rotation ("screw home motion") suggests a torn meniscus. Observe patellar position and tracking during active flexion and extension. Watch for extensive displacement. Complete the exam with walking, running, and cutting maneuvers, looking for asymmetry, limping, malalignment, etc.

In determining specific injuries, the following tests are often useful (see Table 51.2). Remember always to compare the results with the normal (uninjured) side. While the office is the usual setting for these maneuvers, an examination under anesthesia affords greater accuracy, particularly in patients with pain and muscle spasm associated with an acute injury.

- *Abduction (valgus) stress test:* (See Figure 51.6.) This test assesses the integrity of the medial ligament complex. The patient must lie supine with the muscles relaxed. Swing the leg to the side, with the knee flexed to 30°. Place one hand on the lateral knee. Grasp the forefoot and pull it away from the midline with the other hand. Repeat the procedure with the knee in full extension (0°) to test the posterior cruciate ligament. In a positive test, the knee will open up on the medial side; this excessive joint motion, or laxity, is indicative of a tear in the medial complex as well as in the posterior cruciate ligament (PCL).[1] If the test is negative at 0° but positive at 30° of flexion and in full

TABLE 51.1
An Anatomic Approach to Knee Pain

Anterior Knee Pain

1. Patellofemoral syndrome
2. Infrapatellar tendinitis/quadriceps tendinitis
3. Quadriceps strain or tendinitis
4. Subluxing or dislocating patella
5. Osgood–Schlatter's disease
6. Prepatellar bursitis
7. Plica

Lateral Knee Pain

1. Iliotibial band syndrome
2. Popliteus tendinitis
3. Biceps femoris tendinitis/bursitis
4. Lateral meniscal injury
5. Lateral collateral ligament (LCL) injury

Medial Knee Pain

1. Pes anserine tendinitis/bursitis
2. Medial tibial stress syndrome
3. Medial plateau stress fracture
4. Voschell's bursitis
5. Medial meniscus injury
6. Medial collateral ligament (MCL) injury

Posterior Knee Pain

1. Popliteal (Baker's) cyst
2. Meniscus (posterior horn)
3. Posterior capsule (posterior oblique, oblique popliteal, and arcuate ligament) injury
4. Gastrocnemius or hamstring injury or tendinitis

Diffuse Knee Pain

1. Meniscal injury
2. Degenerative joint disease (osteoarthritis)
3. Inflammatory arthritis
4. Osteochondral fracture (acute)
5. Osteochondritis dissecans
6. Malignancy

Source: Feagin, J.A., *Clin. Sports Med.*, 8(3), 453–460, 1989. With permission.

extension, a tear in the medial complex is indicated.

- *Adduction (varus) stress test:* (See Figure 51.7.) This test assesses the integrity of the lateral ligament complex. Have the patient lie supine, as in the abduction stress test. Reverse the positions of your hands. With one hand on the medial side of the patient's knee, hold the forefoot with the other hand and pull it toward the midline. Perform this test at 30° flexion and at full extension (0°). In a positive test, the knee will open up on the lateral side. Excessive laxity with the knee at 30° flexion indicates a tear in the lateral compartment and possibly also the PCL; at 0°, injury to both the lateral collateral and PCL is indicated.
- *Lachman test:* (See Figure 51.8.) Have the patient relax and lie in a supine position. With the knee flexed at approximately 15 to 30°, place one hand around the distal thigh and the other about the proximal tibia with the thumb on the tibial crest. Attempt anterior translation. A positive test occurs when a firm endpoint is not detected or excessive anterior translation occurs. This is the most sensitive test for acute ACL insufficiency.
- *Anterior drawer test:* (See Figure 51.9.) This test assesses the ACL as well as the secondary restraints against excessive tibial translation and rotation (PCL, MCL, capsule). With the patient lying supine with the head down, sit lightly on the patient' s forefoot with the hip flexed 45° and the knee 90°. Exert pressure on the posterior proximal tibia to attempt to translate the tibia forward. With the foot in a neutral position, forward movement of the tibia on the femur suggests an anterior cruciate ligament injury. With the foot in external rotation, additional medial collateral involvement is suggested if the medial tibial plateau advances anteriorly, rotating from the medial to the lateral side. This suggests ACL and medial capsular injury. With the foot in internal rotation, the lateral tibial plateau moves anteriorly and rotates medially from the lateral side when there is an injury to the anterior and posterior cruciate ligaments, as well as the posterolateral capsule. Note: Make certain that all drawer tests begin at the neutral starting point; otherwise, the test may be erroneously interpreted. Because other structures influence the drawer test, do not rely on this test alone for the diagnosis of an ACL sprain.

TABLE 51.2
Summary of Maneuvers

Test	Structure					
	ACL	PCL	MCL	LCL	Menisci	Patella
Abduction stress at 0°		+	±			
Abduction stress at 30°		±	+			
Adduction stress at 0°		+		±		
Adduction stress at 30°		±		+		
Lachman	+					
Anterior drawer, neutral	+				±	
Anterior drawer, internal rotation	+		±		±	
Anterior drawer, external rotation	+			±	±	
Posterior drawer		+				
Posterolateral drawer				+		
Posterior sag/gravity		+				
Jerk	+					
Pivot shift	+					
Reverse pivot shift				+		
External rotation recurvatum				+		
McMurray's					+	
Apley grind/compression					+	
Hypermobility/apprehension						+
Patellar inhibition						+
Patellar compression						+

Note: The ± symbol indicates that the test is inconclusive because the maneuver may be positive or negative with pathology.

- *Posterior drawer test:* (See Figure 51.10.) From a position of neutral rotation, push the tibia posteriorly to assess the posterior cruciate ligament. Excessive posterior translation occurs in athletes who have torn the posterior cruciate ligament.
- *Posterolateral drawer test:* Position the patient as in the anterior drawer test with external rotation of the tibia. Push posteriorly on the proximal tibia. Excessive posterior rotation of the lateral tibial condyle is positive in cases of posterolateral corner injury.
- *Posterior sag (Gravity) test:* (See Figure 51.11.) Have the patient lie supine with the hips flexed to 45° and the knees to 90°, with the feet flat on the table. Observe from the lateral side for posterior displacement or sagging of one tibial tuberosity compared with the other. To perform the quadriceps active test, the patient contracts the quadriceps while the examiner stabilizes the ipsilateral foot on the table. If the PCL is torn and the tibia has sagged posteriorly, quadriceps contraction will now pull the tibia visibly forward into a reduced position. Next, have the patient flex the hips to 90°; support both legs by cupping the heels in your palms or holding the great toes. With the patient's quadriceps relaxed, observe from the lateral side for posterior displacement of one tibial tuberosity compared with the other.
- *Jerk test:* With the patient relaxed and supine and the knee flexed to 90°, internally rotate the foot and leg. The knee is progressively extended while valgus force is applied. At 20 to 30° of flexion, the lateral tibial condyle subluxes anteriorly; with further extension, a sudden reduction occurs, and an ACL injury is indicated.

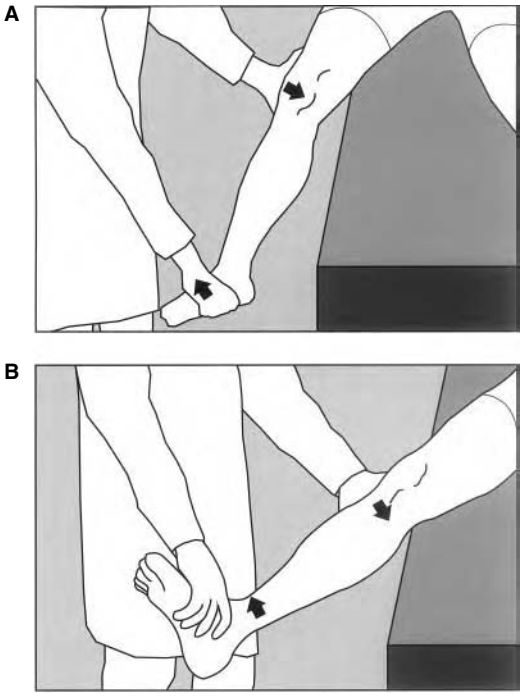

Figure 51.6 Abduction (valgus) stress test. (A) Medial ligament injury is assessed by testing at 30° of flexion. (B) Acute posterior cruciate injury and the medial capsule are assessed by testing in full extension.

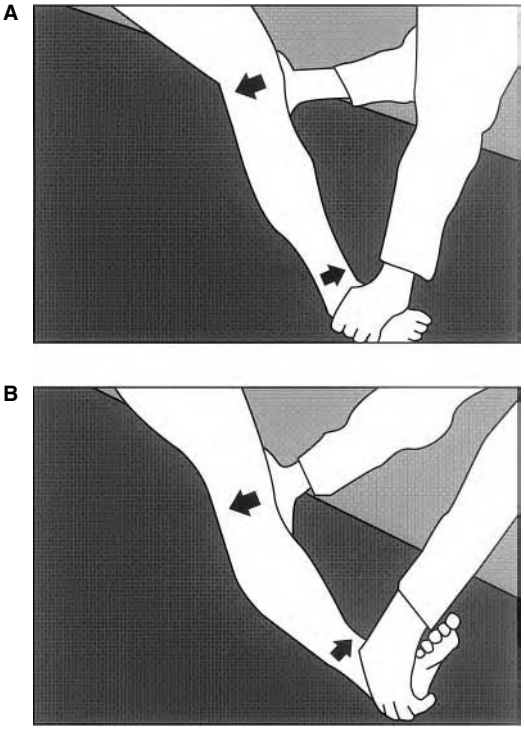

Figure 51.7 Adduction (varus) stress test for injury to the lateral compartment. (A) LCL injury is assessed by testing at 30° of flexion. Most normal knees will have mild laxity with a firm endpoint. (B) Full extension, which also assesses the integrity of the posterior cruciate ligament.

- *Pivot shift test:* (See Figure 51.12.) This test is used to diagnose anterolateral rotatory instability. Begin with the tibia and foot internally rotated and the knee hyperextended. As the knee reaches 20 to 30° of flexion while valgus force is applied, the patient will feel a sudden shift as the lateral tibial condyle rotates anteriorly on the femur. This positive result indicates an injury to the ACL.
- *Reverse pivot shift test:* Perform the pivot shift test with the tibia in external, not internal, rotation. With 90° of flexion, the lateral tibial condyle is subluxed posteriorly, producing a palpable "clunk" during knee extension. A positive test result is common in chronic lateral ligament injuries.
- *External rotation recurvatum test:* (See Figure 51.13.) Lift the leg by the patient's great toe. Varus, external rotation, and hyperextension will be produced if an injury to the posterolateral structures has occurred.
- *McMurray's test:* (See Figure 51.14.) This test is useful for evaluating the posterior menisci. Have the patient lie supine, muscles relaxed and the knee maximally flexed. With valgus stress, slowly extend the knee with the tibia externally rotated (medial meniscus) or internally rotated (lateral meniscus) and your second and third fingers on the medial or lateral joint line, respectively. The test is positive if painful clicking occurs at the medial joint line (medial meniscus) or lateral joint line (lateral meniscus).
- *Apley's meniscal/compression test (Apley grind test):* (See Figure 51.15.) Have the patient lie prone with the knee flexed to 90°. Apply pressure by pushing straight down on the foot while rotating the tibia and partially flexing and extending the knee. A painful pop over the medial joint line during external tibial rotation indicates an injury to the medial meniscus; a

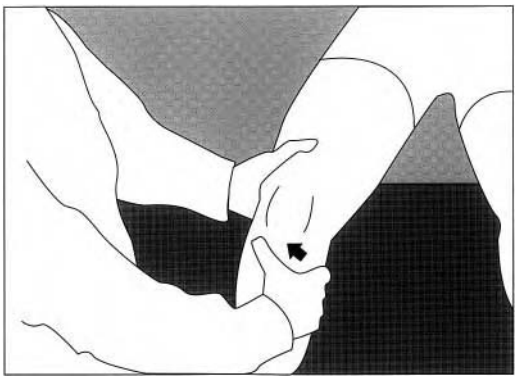

Figure 51.8 Lachman test for ACL sprain/disruption. With the knee flexed at 30°, the thigh is stabilized. The lower hand is placed with the thumb on the tibial crest and fingers purchasing as much of the calf as possible. Increased AP slide with a firm endpoint suggests attenuation of the ACL. If no endpoint resistance is met, it is likely that the ACL is torn completely.

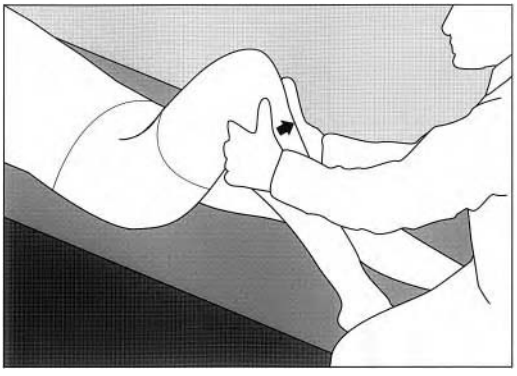

Figure 51.9 Anterior drawer sign.

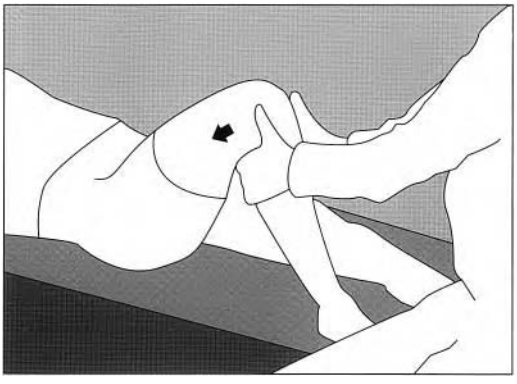

Figure 51.10 Posterior drawer sign.

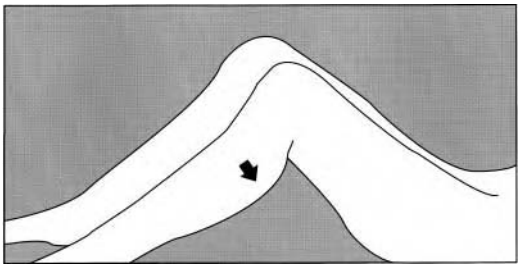

Figure 51.11 Positive gravity (sag) test of the left tibia.

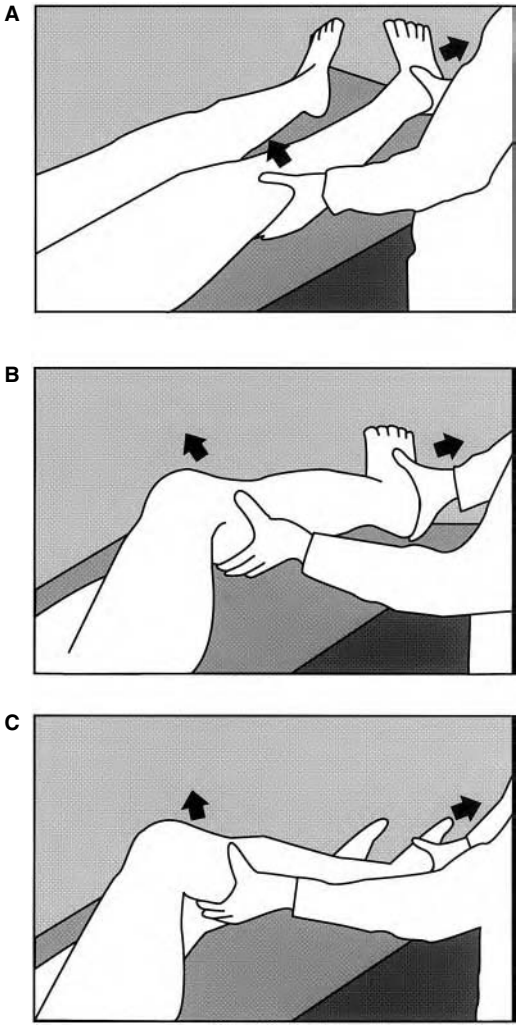

Figure 51.12 Pivot shift. (A) Anterior force is applied to the fibular head while the knee is in valgus. (B) Anterior subluxation of the lateral tibial condyle occurs in a positive test. (C) Reduction of the proximal tibia. *Note:* The jerk test is a reverse pivot shift, starting at 90° of flexion and ending in full extension.

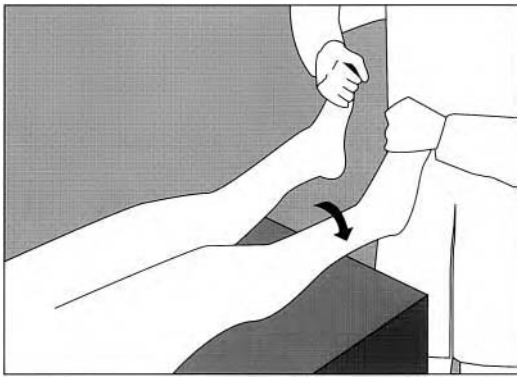

Figure 51.13 External rotation recurvatum test. Posterolateral corner injury is suggested by excessive recurvatum, proximal external tibial rotation, and apparent varus when both lower limbs are lifted by the great toes.

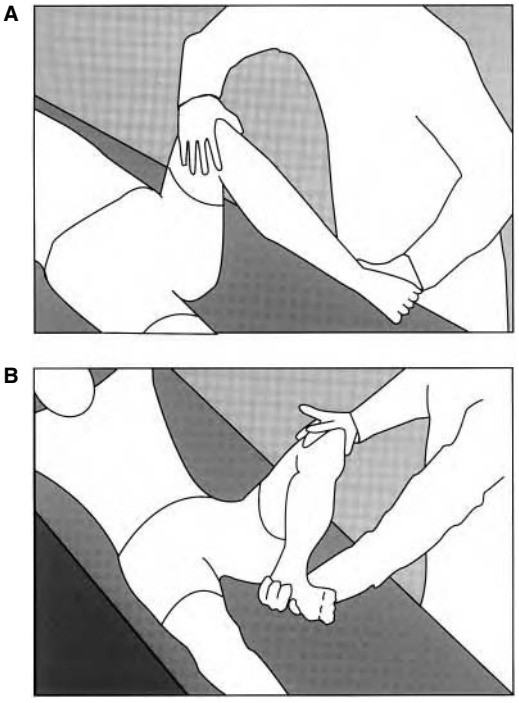

Figure 51.14 McMurray test. (A) To assess the posterolateral meniscus, with thumbs (or fingers) palpating the lateral joint line and valgus stress applied, the foot is internally rotated and the knee is brought from full flexion into extension. (B) To assess the posteromedial meniscus, with fingers palpating the medial joint line and the foot in external rotation, the knee is brought from full flexion into extension. A positive test is a painful click in the appropriate joint line.

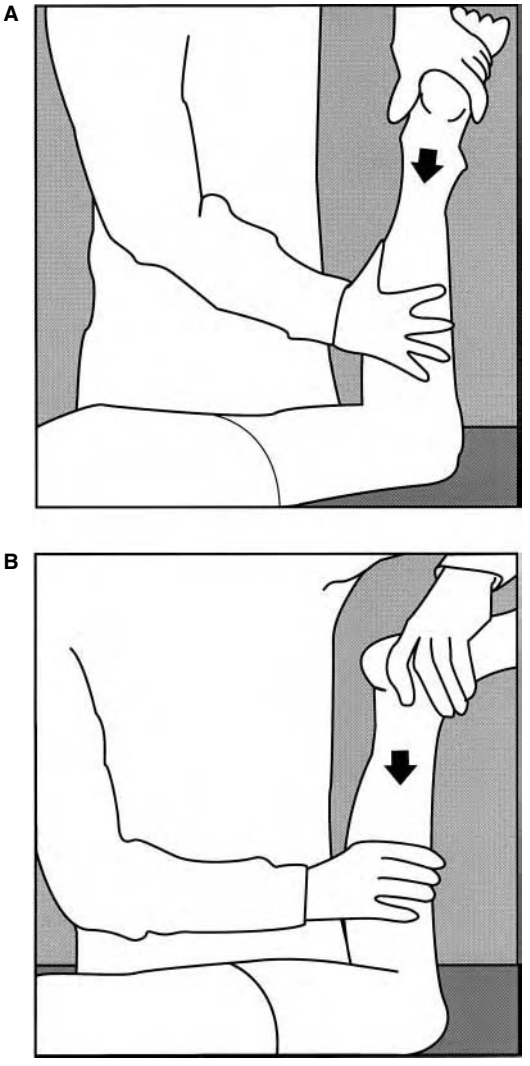

Figure 51.15 Apley compression (grind) test. (A) External rotation for medial meniscus position. (B) Internal rotation for lateral meniscus position.

painful pop over the lateral joint line during internal rotation indicates an injury to the lateral meniscus.

- *Patella apprehension or hypermobility test:* (See Figure 51.16.) Have the patient lie supine and relax the quadriceps. Press against the medial border of the patella with your thumb with the knee flexed 30 to 45°. If the patella begins to sublux, the patient will show apprehension and will ask you to stop. This positive result is indicative of patellar instability.

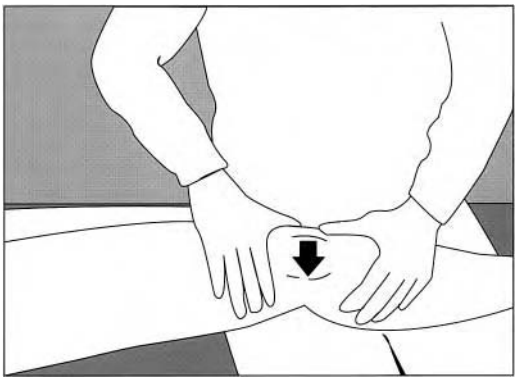

Figure 51.16 Patellar hypermobility/apprehension test. Lateral displacement of the patella may produce sensation of impending subluxation or dislocation.

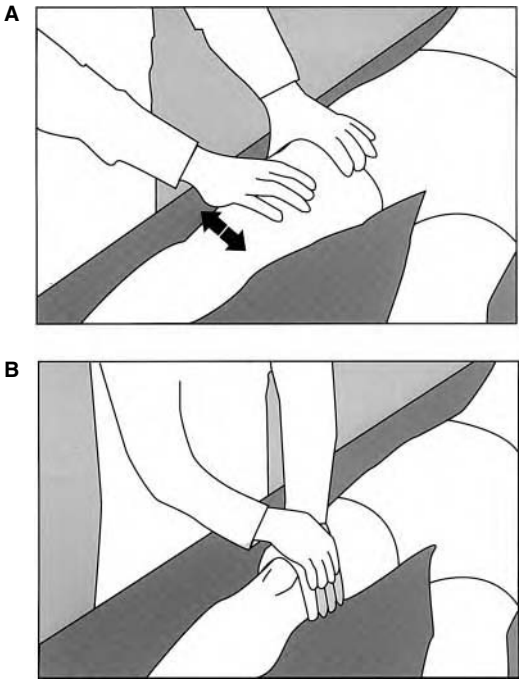

Figure 51.17 Patellar crepitation. (A) Patellar compression test; transverse compression of patella, checking for tenderness and/or crepitation. (B) Patellar inhibition test; longitudinal compression of the patellofemoral joint. Trap the patella distally and then have the patient maximally contract the quadriceps. Check for tenderness, crepitation, and unwillingness (inhibition) to maximally contract the quadriceps against resistance.

- *Patellofemoral compression test:* (See Figure 51.17A.) Compression of the patella against the femur longitudinally and/or transversely produces pain and crepitation.
- P*atella inhibition test:* (See Figure 51.17B.) Apply resistance to the superior pole of the patella. Ask the patient to contract the quadriceps forcefully. In a positive test, the patient will be reluctant to do so or will have a painful crunching under the patella.
- *Malacrea's test:* This test is helpful in diagnosing iliotibial band syndrome. With the patient lying on the uninvolved side, the injured leg should be abducted at the hip with the knee in extension. Apply resistance to the abducted leg while the patient fully flexes and extends at the knee. A positive test occurs when the patient's pain is reproduced in the area of the lateral femoral epicondyle.
- *Quadriceps (Q) angle measurement:* (See Figure 51.18.) Make a line from the anterior superior iliac spine (ASIS) down the thigh to the midpoint of the patella, and a second line from there to the tibial tubercle. A normal Q angle for males should measure less than 10°; for females, less than 15°. A Q angle of more than 20° is generally abnormal.[6]

Laboratory/Radiology Examination

In cases of effusion where the diagnosis is in doubt, infection or crystalline arthropathy is suspected, or when a tense effusion is causing significant symptoms, needle aspiration (under sterile technique) is indicated (see Chapters 27 and 34). The approach can be medial or lateral, with the knee in slight flexion. Acute hemarthrosis is highly suggestive of an ACL tear, although this may also be seen with an osteochondral injury, intraarticular fracture (usually producing a lipohemarthrosis with visible fat droplets in the aspirate), peripheral meniscal tear, and patellar subluxation or dislocation.

Multiple radiographic projections (anteroposterior, lateral, axial patellar, tunnel, and oblique) are necessary to evaluate the knee completely. The axial patellar view (sunrise, Houston, or Merchant projection) is useful for evaluating patellar pathology (subluxation, fracture, and degenerative arthritis). The sunrise or skyline view is taken with the knee flexed to 90°. The tunnel or infrapatellar projection, taken at 30 to 45° of knee

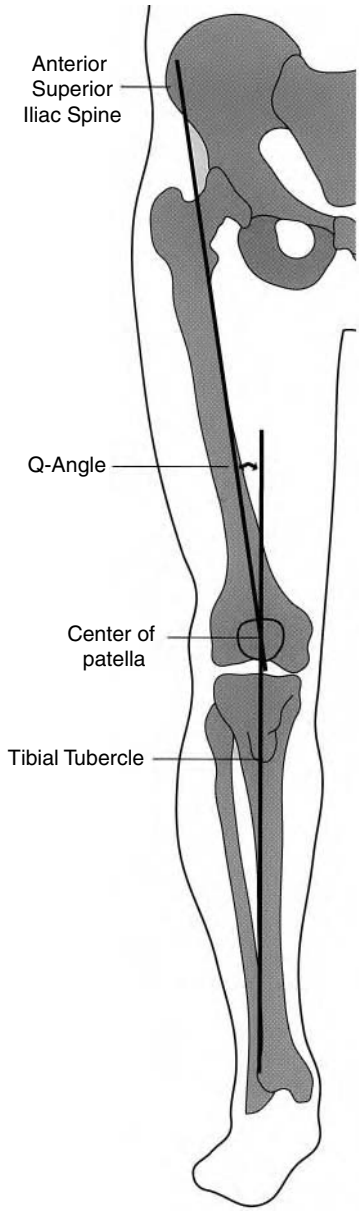

Figure 51.18 Q-angle.

flexion, images the intercondylar notch (e.g., loose bodies) and the subchondral surfaces of the proximal tibia and distal femur. Oblique projections are essential whenever a knee joint fracture is suspected. Stress films can identify growth plate fractures in youth but have limited utility in the assessment of ligament injury severity. Guidelines for obtaining knee radiographs are listed in Table 51.3.

TABLE 51.3
High Yield Criteria for Knee Radiography

Ottawa Knee Rules
Age >55 years
Isolated patella tenderness
Tenderness at the head of the fibula
Inability to flex the knee to 90°
Inability to walk four weight-bearing steps immediately after the injury and in the emergency department
Sensitivity, 97%
Specificity, 27%

Note: These rules do not identify all pediatric patients with knee fractures.

Pittsburgh Decision Rules
Age <12 years or older than 50
Inability to walk four weight-bearing steps in the emergency department
Sensitivity, 99%
Specificity, 60%

Source: Tandeter, H.B. et al., *Am. Family Phys.*, 60(12), 2599–2608, 1999. With permission.

Magnetic resonance imaging (MRI) is very helpful in evaluating the menisci and collateral and cruciate ligaments, as well as in diagnosing osteochondral fractures and osteochondritis dissecans. It is the procedure of choice for assessing acute ligamentous and soft tissue injuries of the knee when the diagnosis is in question or more information is required, as it has excellent sensitivity.[7] However, not all knee injuries require MRI evaluation, and one must be careful not to overread the findings one obtains, as is sometimes the case with degenerative meniscal changes which are misread as acute tears. MRI/magnetic resonance arthroscopy (MRA) or Doppler ultrasonography can help differentiate between a popliteal aneurysm and a popliteal cyst (Baker's cyst). Arthroscopy is the most important advance in the diagnosis of knee joint disorders. The diagnosis and treatment of most intraarticular conditions are now possible with this technique. Specific procedures include loose body removal, cruciate reconstruction, synovectomy, menisectomy and meniscal repair, internal fixation of osteochondral fractures, and resection of intraarticular osteophytes.

LIGAMENTOUS INJURIES

MEDIAL LIGAMENT INJURIES

I. Epidemiology

Injuries to the medial collateral ligament and/or medial capsular ligament can occur in football from a tackle or block against the lateral aspect of the knee if the foot is planted. A skier can incur this injury if one ski becomes trapped in the snow and momentum carries the skier onward (see Figure 51.5). A swimmer can injure the medial ligament(s) when performing the whip kick with the breaststroke.

II. Mechanism of Injury

Contact or noncontact in fixed-foot rotational injuries; valgus force with external tibial rotation.

III. Anatomy

Tearing of medial collateral ligament; may also involve peripheral detachment of medial meniscus and anterior cruciate ("unhappy triad") or injury to medial capsular ligament, tibial collateral ligament, and posterior oblique ligament. For grades, see Chapter 34.

IV. Symptoms

- Grade 1 — mild medial pain; perhaps swelling and limping
- Grade 2 — moderate medial pain, swelling, limping
- Grade 3 — severe medial pain; swelling; knee gives way into valgus

V. Signs

- Grade 1 — medial edema; tenderness; positive abduction stress test
- Grade 2 — medial edema; tenderness; positive McMurray's (if meniscus is involved) and abduction stress tests
- Grade 3 — marked medial edema and tenderness

With stress testing: at 0°, some valgus opening; at 30°, marked opening of joint; at 90°, negative neutral anterior and posterior drawer tests unless cruciates are involved; anterior drawer test with external tibial rotation is often positive.

VI. X-Rays

Routine films (AP, lateral) are negative.

VII. Special Studies

Use abduction stress film to distinguish ligament injury from epiphyseal fracture in skeletally immature patients. Fracture opens at growth plate; ligament tear opens at joint line. MRI has high sensitivity and specificity for medial collateral ligament (MCL) tears; results cannot distinguish grade 1 and 2 injuries but can separate them from grade 3 (complete) tears.

VIII. Diagnosis

Sprain ("breaststroker's knee"); location: MCL; grade 1, 2, or 3.

IX. Differential Diagnosis

Epiphyseal fracture of distal femur or proximal tibia (in young patients), patellar dislocation with medial retinacular injury, medial meniscus tear.

X. Treatment

A. Initially: Treat with RICE (rest, ice, compression, elevation), nonsteroidal anti-inflammatory drugs (NSAIDs), and analgesics as needed; posterior splint should be followed by crutches with weight bearing.

B. Long-term: For grades 1 and 2, treat symptoms. For a grade 3 injury (isolated injury only), use a brace (cast or hinged) that allows motion but prevents excess valgus stress, physical therapy (range of motion and progressive resistance exercises).[8,9] Surgery is recommended for complicated cases (e.g., anterior cruciate ligament and medial collateral ligament tears).[1]

Consultation: recurrent disability, fractures, grade 3 injuries, large effusion, abnormal x-rays

XI. Complications

Degenerative joint disease, Pellegrini–Stieda disease (calcification of MCL).

XII. Prevention

Use of multistudded shoes instead of long cleats, avoiding drills that force athletes into dangerous positions and techniques that purposely injure opponents' knees, weight training.

LATERAL LIGAMENT INJURIES

I. Epidemiology

The lateral stabilizing complex is not as vulnerable to injury as the medial ligaments and is therefore injured less frequently. Injuries to these ligaments do occur in contact (e.g., wrestling,

soccer) and non-contact (e.g., racquet sports, running) injuries.

II. Mechanism of Injury
Varus force with internal tibial rotation; anterotibial trauma with hyperextension injures posterolateral components.

III. Anatomy
Tearing of lateral (fibular) collateral ligaments; biceps tendon, iliotibial band, peroneal nerve, popliteal tendon, and posterolateral corner of the lateral complex may be injured in severe cases. Cruciate ligaments and meniscus are rarely involved. For grades, see Chapter 34.

IV. Symptoms
For grades 1 and 2, pain over lateral ligament complex. For grade 3, pain; giving way of knee when twisting, cutting, and pivoting or with standing, walking, and backwards running (with posterolateral injury).

V. Signs
In acute cases, possible increased adduction stress test at 30° flexion and positive posterolateral drawer test. For chronic cases, positive reverse pivot shift test and external rotation recurvatum test; external rotation recurvatum may also be seen on standing.

VI. X-Rays
Routine films (AP and lateral) of proximal lateral tibia are usually negative; may see avulsion of midportion of lateral capsular ligament with small fragment of proximal lateral tibia (lateral capsular sign) or small avulsion fragment of proximal fibula with posterolateral ligament complex (arcuate sign).

VII. Special Studies
See Medial Ligament Injuries section, above.

VIII. Diagnosis
Sprain; location: LCL; grade 1, 2, or 3.

IX. Differential Diagnosis
Degenerative arthritis (medial compartment), anterior or posterior cruciate injury, lateral meniscus tear.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; immobilization.
B. Long-term: For grades 1 and 2, treat with bracing, crutches with weight bearing, and physical therapy (ROM and PREs).[8,9] For grade 3, surgical repair is recommended.

Consultation: recurrent disability, fractures, grade 3 injuries, large effusion, abnormal x-rays

XI. Complications
Degenerative joint disease.

XII. Prevention
Use of multistudded shoes instead of long cleats, avoiding drills that force athletes into dangerous positions and techniques that purposely injure opponents' knees, weight training.

ANTERIOR CRUCIATE LIGAMENT INJURIES

I. Epidemiology
One of the most common knee injuries in sports, a tear of the anterior cruciate ligament has traditionally been diagnosed as a "knee sprain." Eighty percent of ski injuries involve the ACL. An estimated 100,000 ACL injuries per year result from skiing in the United States.[1] Despite an 80% return to participation in sports, 50 to 65% are reinjured in 1 to 2 years. After 5 years, only 35% still participate.[10]

II. Mechanism of Injury
The ACL is usually injured as part of a more complex injury through hyperextension, varus/internal rotation, extremes of valgus and external rotation, deceleration (usually non-contact), or the application of force that drives the tibia in an anterior direction when the knee is flexed at a right angle. High-top ski boots, steep slopes, and rapid speeds are associated with ACL injury.

III. Anatomy
The ACL may be torn from the femur or tibia or, more commonly, in its midportion. The injury usually involves damage to other knee structures, such as avulsion of the tibial spine in youngsters, damage to the middle third of the lateral capsular ligament or to the menisci, or often vertical longitudinal (circumferential) tears (so-called

"bucket-handle tears"). In 75 to 85% of knees with acute hemarthrosis, the injury is a tear of the ACL.[1]

IV. Symptoms

Acutely, a loud pop is heard followed by dizziness, sweating, faintness, nausea, and severe swelling within the next several hours. Patient feels as though the knee is unstable, particularly in rotation. Full extension is difficult. Chronically, the patient reports often feeling a sensation of the knee "giving way" and has a history of a "knee sprain" or "trick knee," especially during pivoting and cutting.

V. Signs[11]

In acute cases, large hemarthrosis and positive Lachman test are observed. Chronic cases have a positive Lachman test and positive pivot shift or jerk test. Anterior drawer sign is unreliable. Findings are of functional instability.

VI. X-Rays

Routine films (AP and lateral) may reveal a lateral capsular sign (i.e., avulsion of midportion of lateral capsular ligament from the lateral tibial condyle) and avulsion of tibial spine in young patients.

VII. Special Studies

Magnetic resonance imaging is sensitive and specific, especially with difficult cases. Arthroscopy is valuable in diagnosing associated intraarticular injuries (e.g., torn menisci or chondral fractures).[1,6] Arthrography has been supplanted by above techniques.

VIII. Diagnosis

Sprain; location: ACL; grade 1, 2, or 3.

IX. Differential Diagnosis

Acute: osteochondral fracture, patellar dislocation, peripheral meniscus tear; chronic: ligamentous laxity and/or meniscal tears.

X. Treatment

A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; use of crutches with limited motion brace or splint. Diagnostic or therapeutic aspiration is occasionally indicated.

B. Long-term: For grades 1 and 2, use orthotics/bracing/taping to prevent full extension during participation in contact sports; physical therapy (ROM and PREs) should concentrate on both quadriceps and especially hamstrings; resistive exercises between 0 and 45° of flexion during the first year following injury or reconstruction should be included (Level of Evidence A, randomized controlled trial).[8,9] Relative effectiveness of modalities remains to be established. A knee brace may be useful because it improves proprioception of the knee. For Grade 3, conservative therapy offers a prognosis of 33% improvement with minimal symptoms, 33% functional though still symptomatic, and 33% progressive functional disability requiring surgical reconstruction. Autografts have the highest success rate and lowest incidence of complications. A poor prognosis is associated with (1) age less than 30, (2) jumping and pivoting sports, (3) torn menisci, (4) marked anterior subluxation, (5) generalized joint laxity.

Consultation: grade 3 sprains in active athletes and recurrent functional instability in persons who do not participate in sports and who have had a full course of physical therapy; avulsion injuries or unclear associated intraarticular injuries[10,11]

XI. Complications

Degenerative joint disease, meniscal degeneration or injury ("bucket-handle" tear of meniscus).[1]

XII. Prevention

Use of multistudded shoes instead of long cleats, avoiding drills that force athletes into dangerous positions and techniques that purposely injure opponents' knees, functional knee brace/stabilizer,[12] weight training.

POSTERIOR CRUCIATE LIGAMENT INJURIES

I. Epidemiology

A posterior cruciate ligament injury occurs in sports when a player lands on the tibial tubercle with the knee flexed. This injury may occur on artificial turf in football or when skating into the boards in hockey. It usually occurs in conjunction with a lateral or medial ligament injury.

II. Mechanism of Injury
Valgus/varus in full extension, rotation; rapid deceleration (e.g., hitting the dashboard in a motor vehicle accident); hyperextension, direct blow to anterior proximal tibia, fall on a flexed knee.

III. Anatomy
Tear of part or all of two major bundles (posteromedial and anterolateral) of PCL.

IV. Symptoms
Swelling (less than in injury of ACL); in chronic cases, feeling of femur sliding off the tibia, especially when decelerating or descending stairs or slopes. Many patients are asymptomatic. Secondary patellofemoral pain symptoms may develop.

V. Signs
In acute cases, the abduction or adduction stress test is positive with full extension in cases caused by varus or valgus trauma; the posterior drawer test is positive if injury was caused by blow to the anterior tibia. In chronic cases, the posterior drawer test and gravity test are positive. Findings are of functional instability.

VI. X-Rays
Routine films (AP and lateral) may show sag in tibia and bony avulsion with tibial attachment of PCL.

VII. Special Studies
Stress films assess posterior drawer sign while taking a cross-table lateral view. Computed tomography (CT) is good for cases of bony involvement. MRI shows the PCL clearly and is effective in difficult diagnoses.

VIII. Diagnosis
Sprain; location: PCL; grade 1, 2, or 3.

IX. Differential Diagnosis
Posterolateral corner injury (posterior drawer test and posterolateral drawer test may appear the same) can coexist with PCL injury. Consultation and MRI can be used to distinguish the two.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: For grades 1 and 2, treat with orthotics/bracing and physical therapy (ROM and PREs).[8,9] Many continue to recommend conservative treatment for isolated grade 3 PCL injuries. Surgical repair/reconstruction using arthroscopy with autografts, allografts, or prosthetic ligaments has been attempted but totally satisfactory surgical treatment has not yet been developed.[1]

Consultation: grade 3 injuries and recurrent instability

XI. Complications
Degenerative joint disease, meniscus tears.

XII. Prevention
Using multistudded shoes instead of long cleats; avoiding drills that force athletes into dangerous positions and techniques that purposely injure opponents' knees; wearing seat belts; wearing a functional knee brace/stabilizer (see Chapter 32);[12] weight training.

MENISCAL INJURIES
MEDIAL MENISCUS INJURIES
I. Epidemiology
The meniscus is torn less frequently than the knee is sprained, but most vertical longitudinal tears of the menisci occur in knees in which the ACL is torn.[1] Many meniscal injuries occur in soccer (rather than football). Meniscal injuries are rare in preadolescents and often are not diagnosed in adolescents. Many of these injuries occur at the time of medial collateral or ACL sprains but go unrecognized because of lack of symptoms and disability. The incidence of medial meniscal tears increases over time in ACL-deficient knees.[13]

II. Mechanism of Injury
A fixed-foot rotation injury while weight bearing with the knee flexed, resulting in a combination of compression and rotational forces being exerted on the meniscus. The tibia rotates externally with respect to the femur.

III. Anatomy
Disruption of medial semilunar cartilage of knee; circumferential (bucket-handle) tears are commonly associated with ACL injury.

IV. Symptoms
Mild swelling, joint-line pain; popping, slipping, or catching over joint line; recurrent locking in chronic setting; buckling.

V. Signs
Generally poor sensitivity and specificity for individual maneuvers which may vary from exam to exam (Level of Evidence B, prospective cohort),[14] (Level of Evidence A, quantitative systematic meta-analysis):[15] positive McMurray's test, positive Apley's test, joint-line tenderness, quadriceps atrophy, loud "clunk" during anterior drawer testing, inability to squat and duck walk.

VI. X-Rays
Routine films usually normal; if tear has been present for a long time, possible joint line spurring, narrowing of joint line, and calcification of cartilage may be observed.

VII. Special Studies
Magnetic resonance imaging is the procedure of choice and offers high sensitivity, specificity, and accuracy.[16] Arthroscopy has the additional advantage of being treatment but has low sensitivity to tears in the posterior horn.

VIII. Diagnosis
Medial meniscus tear.

IX. Differential Diagnosis
Sprain, patellar problems, pathogenic synovial plica, loose bodies, medial joint arthritis.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Physical therapy (ROM and PREs) for a stable tear without ligamentous instability.[8] Surgery/arthroscopy should be considered in cases of persistent mechanical symptoms, continued pain and swelling, or injuries involving competitive athletes. During surgery, as much of the meniscus as possible should be saved to minimize the development of arthritic degeneration. Many tears, especially peripheral ones, can be repaired.

Consultation: "locked" joint (urgent referral), consideration of meniscal repair, unclear injury extent, failed conservative therapy, or for competitive athletes

XI. Complications
Degenerative joint disease, cystic degeneration of meniscus.

XII. Prevention
Use of multistudded shoes instead of long cleats, avoiding drills that force athletes into dangerous positions and techniques that purposely injure opponents' knees, wearing a functional knee brace/stabilizer (see Chapter 32),[12] weight training.

LATERAL MENISCUS INJURIES

I. Epidemiology
The lateral meniscus has greater mobility than its medial counterpart due to differences in tibial plateau topography, the interposition of the popliteus tendon between the lateral collateral ligament and the periphery of the meniscus, and its more narrowly spaced horns. As such, it is less likely to be torn over time in a chronically ACL-deficient knee or suffer an isolated tear; however, it is still susceptible to tears, especially during injuries causing acute ACL disruption. In fact, lateral meniscus tears are the most frequent injury associated with an acute ACL tear (Level of Evidence B, Prospective Cohort).[13,17] An increased incidence of lateral meniscus injuries may also be noted in wrestling because of the frequently fixed attitude of the knee with the foot in external rotation. Injury may result from a single traumatic episode, degenerative processes, or a combination and may also be associated with abnormalities such as a congenital discoid meniscus.

II. Mechanism of Injury
A fixed-foot rotation injury in weight bearing with the knee flexed results in a combination of compression and rotational forces being exerted on the meniscus. Rotational movements taking place within the meniscus, instead of between the condyle and the meniscus, may result in a lateral meniscus tear.

III. Anatomy
Disruption of lateral semilunar cartilage of knee.

IV. Symptoms
Mild swelling, joint line pain; slipping or catching over joint line; recurrent locking in chronic setting; buckling.

V. Signs
Generally poor sensitivity and specificity for individual maneuvers, which may vary from exam to exam:[14,15] positive McMurray's test, positive Apley's test, localized puffiness or distinct cystic lesion directly over lateral joint line, quadriceps

atrophy, loud "clunk" during anterior drawer testing, inability to squat and duck walk.

VI. X-Rays
Routine films usually normal; if tear has been present for a long time, possible joint line spurring, narrowing of joint line, or calcification of cartilage may be noted; widening of joint space occurs with congenital discoid lateral meniscus.

VII. Special Studies
Magnetic resonance imaging is the procedure of choice as it has high sensitivity, specificity, and accuracy.[16] Arthroscopy is diagnostic and/or therapeutic.

VIII. Diagnosis
Lateral meniscus tear.

IX. Differential Diagnosis
Sprain, loose bodies, degenerative arthritis of lateral compartment, popliteus tendinitis, iliotibial band friction syndrome, discoid meniscus.

X. Treatment
See Medial Meniscus Injuries section, above; a child with discoid meniscus requires consultation.

XI. Complications
Degenerative joint disease, cystic degeneration of meniscus.

XII. Prevention
See Medial Meniscus Injuries section, above.

PATELLAR INJURIES

PATELLOFEMORAL SYNDROME/DYSFUNCTION (RUNNER'S KNEE, CHONDROMALACIA)

I. Epidemiology
Patellofemoral syndrome is an imprecise term for a condition characterized by anterior knee pain as a major symptom.[6,18] Predisposing factors include increased running mileage, especially with downhill running in individuals who have recently gone through a growth spurt or who are overweight. Perhaps accounting for 40% of all knee pain presenting to the primary care physician, this condition is often incorrectly termed chondromalacia patella, which is a diagnosis that should be reserved for articular cartilage damage diagnosed by the arthroscopist or pathologist.

II. Mechanism of Injury
Repetitive microtrauma due to abnormal tracking of the patella within the femoral groove or direct blow to the patella;[14,18] weakness of the hip girdle may also contribute.

III. Anatomy
Inflammation and degeneration of patellar cartilage; also shallow femoral groove. A high-riding patella with a dynamic imbalance of the stronger lateralis vs. weaker medialis, with a possible deficiency of the vastus medialis oblique fibers, is often seen. Also observed are lower extremity malalignment ("miserable malalignment") with varying patterns of femoral anteversion, medial squinting of the patellae, external tibial torsion, tibial varum, genu valgum, pes planus, and excessive foot pronation.[10,19]

IV. Symptoms
Retropatellar and peripatellar pain with grating, grinding, or clicking. Pain is often worse when walking down stairs or slope. Weakness, swelling (usually mild if present at all), snapping, popping, pseudo-locking, or giving way around the patella. When sitting in a cramped position, patient will need to flex and extend the knee (theater sign). Pain with squatting, especially while compressing the patella during the squat.

V. Signs
Malalignment[10] with increased Q angle, dysplastic vastus medialis oblique muscle, abnormal patellar alignment and tracking, midfoot pronation, tenderness under the medial facets, crepitation with patellar movement, positive patellofemoral compression and inhibition tests, and swelling of infrapatellar fat pad. A large effusion should make the examiner suspicious of other pathology (e.g., meniscal tear, chondral lesion, cruciate tear, Lyme disease). Hip girdle weakness may also be noted. The patellar apprehension test is usually negative. The patellar inhibition test is positive.

VI. X-Rays
X-rays are not necessary for classic cases but may be helpful if the diagnosis is in question or patient is not progressing as expected. Films are usually normal but may demonstrate patella alta. Tunnel view may show loose body/osteochondritis dissecans (OCD); sunrise view may demonstrate lateral tilt and/or subluxation.

VII. Special Studies
Magnetic resonance imaging utilizes newer machines with improved sensitivity for detecting chondral lesions but findings are unremarkable in most cases. Arthroscopy is the gold standard if symptoms persist despite appropriate treatment.

VIII. Diagnosis
Patellofemoral syndrome.

IX. Differential Diagnosis
Osteochondritis dissecans of femur or patella, synovial plica irritation, Hoffa's syndrome (fat pad inflammation), proximal infrapatellar tendinopathy, osteoarthritis, neoplasm, Legg–Calve–Perthes disease, or slipped capital femoral epiphysis in youngsters.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; patella cutout brace with inferior horseshoe.
B. Long-term: Physical therapy (ROM and PREs);[8,19] functional patellar bracing, orthotics, or arch supports for foot malalignment (Level of Evidence A, randomized controlled trial).[12] Optimal physical therapy regimen is controversial but focuses on improving patellar tracking, flexibility of lateral tissues, VMO function, and addressing other potential weak links in kinetic chain. Short-term effects of therapy are superior to placebo, but long-term outcomes require more study (Level of Evidence, randomized controlled trial).[20,21] Surgical treatment is recommended for lateral release or extensor mechanism reconstruction for failed conservative therapy (rarely required).

Consultation: recurrent disability

XI. Complications
Worsening chondral changes.

XII. Prevention
Avoid training errors; improve biomechanics; pay attention to the "S" list (see above); control pronation with appropriate shoes and orthotics as needed; provide shock absorption with cushioned arch supports; wear bracing with a patella cutout brace with inferior horseshoe (see Chapter 32), with or without an infrapatellar strap (Level of Evidence A, randomized controlled trial);[12] strengthen medialis muscles; run on soft, flat surfaces; minimize downhill running.

PATELLAR SUBLUXATION/DISLOCATION

I. Epidemiology
Predisposing factors include: (1) a dynamic imbalance with the lateral musculature overpowering the medial muscles, (2) hypoplastic patella, (3) patella alta, and (4) shallow femoral groove. Patellar dislocation commonly occurs as a noncontact injury, resulting from the force of contraction in the quadriceps combined with genu valgus. The injury can also occur in athletes with relatively normal lower extremity alignment and quadriceps mechanisms. Patellar subluxation may occur with less severe force than dislocation, or in normal, everyday activity.

II. Mechanism of Injury
Valgus and/or twisting with strong quadriceps contraction.

III. Anatomy
Partial or complete lateral displacement of patella from femoral trochlea.

IV. Symptoms
Possible previous complaints of instability or pain; peripatellar and retropatellar pain; feeling of unstable kneecap, deformity ("something coming out" medially or "something going back into place"); theater sign; swelling within 2 hours of injury; rarely, locking and buckling.

V. Signs
Dependent on whether patella is reduced or still dislocated: If reduced, signs include medial retinacular tenderness, lateral squinting of patella, patella alta, increased Q angle, positive hypermobility and apprehension tests, swelling (mild to marked). If the patella is still dislocated, signs include a deformity over the lateral femoral condyle with a prominence of uncovered medial femoral condyle; possible medial ligamentous instability.

VI. X-Rays
Routine films (AP and lateral) are usually normal as spontaneous relocation is typical. Infrapatellar view may show avulsion of medial edge of patella or large osteochondral fracture, lateral

tilt, or subluxation. Lateral view may demonstrate patella alta.

VII. Special Studies
None indicated usually, but consider MRI to evaluate for associated injury in complex cases.

VIII. Diagnosis
Patellar subluxation/dislocation.

IX. Differential Diagnosis
Sprain, meniscus tear, chronic knee ligament instability.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, analgesics as needed. Relocation is performed by knee flexion, then extension and gentle pressure along lateral patellar edge; aspiration may be useful for diagnosis of associated fracture (fat in blood) and treatment.
B. Long-term: For first dislocation, immobilize in extension with foam pad over vastus medialis obliquus and lateral buttress for 4 weeks, followed by physical therapy (ROM and PREs) and functional patellar bracing that prevents subluxation but allows some flexion and extension.[8] For recurrent dislocation, temporary immobilization and crutches should be followed by physical therapy and bracing. Surgery is recommended for chronic cases or acute cases associated with a loose fracture fragment in the joint or grade 3 strain of the VMO.

Consultation: recurrent cases or acute cases involving a VMO tear or fracture.

XI. Complications
Chronic subluxation or recurrent dislocations, articular cartilage damage, avulsion fracture, osteochondral fracture, patellar fracture, chondromalacia patella.

XII. Prevention
Patellar stabilizing braces (see Chapter 32); strengthening exercises (VMO and medialis) may help.

TENDINITIS/BURSITIS

INFRAPATELLAR TENDINITIS ("JUMPER'S KNEE")

I. Epidemiology
Infrapatellar tendinitis, or tendinitis at the inferior pole of the patella, is most commonly seen in athletes involved in jumping activities (e.g., volleyball, basketball, track and field) and in those doing squats (e.g., powerlifting).[19] A skeletally immature athlete with pain in this area likely has Sinding–Larsen–Johansson syndrome, an apophysitis secondary to excessive traction at the inferior patellar pole.[2]

II. Mechanism of Injury
Repetitive microtrauma due to excessive jumping or other high-patellofemoral-stress activity.

III. Anatomy
Inflammation of patellar tendon, usually at attachment to inferior pole of patella.

IV. Symptoms
Infrapatellar pain, first after exercise then later during exercise and at rest.

V. Signs
Tenderness at inferior pole of patella; some tenderness over body of patellar tendon; extensor mechanism malalignment variably present (see Patellofemoral Syndrome section, above); tightness of hamstring, heelcord, and/or quadriceps muscles; ankle dorsiflexor weakness.

VI. X-Rays
Routine films are usually normal; occasionally may see irregularity at inferior pole of patella or signs of malalignment (see Patellofemoral Syndrome section, above).

VII. Special Studies
Magnetic resonance imaging may demonstrate degenerative changes in tendon.

VIII. Diagnosis
Infrapatellar tendinitis ("jumper's knee").

IX. Differential Diagnosis
Patellofemoral syndrome, Hoffa's syndrome, other soft-tissue lesion of patellar tendon such as tumor.

X. Treatment

A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Physical therapy (ROM and PREs) should concentrate on hamstring, ankle dorsiflexors, heelcord, and quadriceps flexibility and strength; counterforce brace;[8,19] phonophoresis, iontophoresis. Various needling procedures have been utilized with anecdotal success but more research must be done in this area. Steroid injection is very risky as it may promote rupture.

Consultation: rarely necessary but appropriate with recurrent or persistent disability

XI. Complications

Rare rupture of infrapatellar tendon (IPT).

XII. Prevention

Use a patella cutout brace with inferior horseshoe; stretching; counterforce bracing may be helpful.

QUADRICEPS TENDINITIS

I. Epidemiology

Quadriceps tendinitis occurs as a result of repetitive jumping (e.g., basketball, volleyball) with acceleration or deceleration. It is usually seen during a forceful contraction of the quads. This injury has become more common as the length of playing seasons and the frequency of daily participation have increased.[19]

II. Mechanism of Injury

Repetitive trauma (e.g., excessive jumping, squatting with heavy weights).

III. Anatomy

Inflammation of quadriceps tendon at superior pole attachment of patella; may involve only vastus lateralis insertion into superolateral pole of patella or vastus medialis obliquus insertion into superomedial pole of patella.

IV. Symptoms

Suprapatellar pain, especially with squatting and jumping.

V. Signs

Tenderness or swelling at superior pole of patella; hamstring, heelcord, and quadriceps muscle tightness; signs of malalignment (see Patellofemoral Syndrome section, above).

VI. X-Rays

Routine films are normal.

VII. Special Studies

None indicated.

VIII. Diagnosis

Quadriceps tendinitis.

IX. Differential Diagnosis

Suprapatellar pain from synovial plica, bone tumor of distal femur.

X. Treatment

See Infrapatellar Tendinitis section, above.

XI. Complications

None.

XII. Prevention

Exercises, stretching; patella cutout brace may be helpful (see Chapter 32).

ILIOTIBIAL BAND (ITB) SYNDROME

I. Epidemiology

Iliotibial band syndrome is the most common cause of lateral knee pain. Predisposing factors include increasing running mileage to 20 to 40 miles per week and recent changes in the "S list" in training, especially hilly terrain.[22] Hard-surface running with increased speed is a common factor. ITB syndrome often occurs in athletes with neutral or varus knee alignment, particularly in the downside leg when the athlete is running on a sloping road. Men develop this condition more commonly than women because of lower body-fat percentages, alignment differences, and variations in bony morphology. If ITB syndrome occurs in a woman, it may be indicative of an eating disorder (e.g., anorexia nervosa).

II. Mechanism of Injury

Repetitive microtrauma.

III. Anatomy

The iliotibial band is pulled anteriorly by the tensor fascia lata in flexion and posteriorly by the gluteus maximus in extension. The iliotibial band may become inflamed with excessive flexion and extension of the knee as it rubs back and forth over the lateral femoral epicondyle.

IV. Symptoms
Lateral knee pain in activity, usually occurring at a fixed distance at a given pace; stinging during deceleration when foot contacts the ground (footstrike); inflexibility; occasional popping.

V. Signs
Tenderness to palpation is noted at distal ITB over lateral femoral epicondyle, usually about 3 cm proximal to the lateral joint line. Malacrea's test will produce pain and tenderness will be noted in the area of the lateral femoral epicondyle of the knee. Renne's creak sign may be positive (lateral knee creaking sound when flexing or extending knee). Intraarticular findings are negative.

VI. X-Rays
X-rays are not helpful.

VII. Special Studies
None indicated.

VIII. Diagnosis
Iliotibial band syndrome.

IX. Differential Diagnosis
Popliteus tendinitis; vastus lateralis tendinitis; lateral meniscus disorders.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; transverse friction and ice massage.
B. Long-term: Cross-training, phonophoresis/iontophoresis, injection therapy; Physical therapy (ROM and PREs) should include stretching of hamstrings, gluteals, abductors, and iliotibial tract; lateral heel or sole wedge or orthotics should be worn in shoes.

Consultation: recurrent disability may rarely require surgical release

XI. Complications
Chronicity causing pain and disability.

XII. Prevention
Stretching of the iliotibial band; control of abnormal foot motion or leg length discrepancy; run on softer surfaces; minimize downhill or sloped-road running.

OTHER TYPES OF TENDINITIS
Tendinitis, generally the result of overuse, is characterized by inflammation and/or pain at a given tendon and nearby soft tissues. *Popliteus tendinitis* occurs especially in athletes who run on banked surfaces or downhill. It causes lateral knee pain that can be reproduced by resisted internal rotation of the tibia with the knee flexed. The symptoms may also mimic those of a lateral meniscus injury or iliotibial band syndrome. *Biceps femoris tendinitis* results from ballistic stretch or chronic irritation. *Pes anserine tendinitis* may occur from overuse or a direct blow. The patient will experience initial pain on stretching the affected tendon and later pain on active contraction of the tendon, as well as tight hamstrings. X-rays are usually not helpful, but MRI can be used. *Hamstring tendinitis* must be differentiated from sciatica; *semimembranosus tendinitis* may resemble medial meniscus disorders. Stretching and strengthening exercises are essential in prevention. It is important to note that many cases of "tendinitis" actually show no evidence of acute inflammation and are more appropriately termed "tendinosis," reflecting a pathophysiology primarily involving degenerative changes and scarring. This difference is important in that the treatment of tendinosis requires physical therapy, relative rest, cross-training, and correction of training errors but may not respond to or require NSAIDS.

BURSITIS
Bursitis occurs as either an acute or a chronic irritation to the bursa. Injury occurs as a result of overuse or a direct blow. Inflammation, with bleeding into the bursa, may occur. Swelling and pain may occur in the prepatellar region (*prepatellar bursitis*), over the medial joint line (tibial collateral ligament or *Voschell's bursitis*), in the distal patellar tendon region (*deep infrapatellar bursitis*), and on the proximal medial tibia (*pes anserinus bursitis*) (see Figure 51.2B). X-rays may show soft-tissue swelling or, in chronic cases, calcification. MRI may be used. The differential diagnoses should include medial meniscus tear (tibial collateral ligament bursitis); pes anserinus tendinitis, tumors, and other causes of proximal medial tibial pain (pes anserinus bursitis); and other causes of patellar tendon pain (deep infrapatellar bursitis). In acute cases of bursitis, treatment should be with ice, compression, aspiration, and padding. Care must be taken to rule out an infectious etiology, especially with the superficial bursae. Most cases respond well to conservative

management, but chronic cases may require injection therapy or even bursa excision.

MISCELLANEOUS CONDITIONS

SYNOVIAL PLICA

I. Epidemiology
A synovial plica is a congenital redundancy of synovial tissue that may manifest itself during overuse or after a direct blow (e.g., falling on turf or a dashboard injury). Various forms of synovial plica are present in 20 to 60% of the population.[2]

II. Mechanism of Injury
Microtrauma from repetitive flexion and extension; direct blow to medial patellofemoral joint.

III. Anatomy
Thickening of synovium, which rubs against bones during flexion; structurally, a remnant of embryonic walls that divide the knee into medial, lateral, and suprapatellar pouches. It appears as a fold of synovium attached to the periphery of the joint and to the underside of quadriceps tendon (suprapatellar plica); possibly a free edge along medial patellofemoral joint (medial plica) with variable protrusion into the joint space.

IV. Symptoms
Anterior knee pain, swelling; pain over suprapatellar or medial patellofemoral regions with long periods of knee flexion; distinct snap or pop after knee is extended; painful pseudo-locking over the medial knee joint.

V. Signs
Sometimes able to palpate tender plica medially by passively flexing and extending the internally rotated tibia; hamstring and heelcord tightness; other extensor mechanism malalignment signs present.

VI. X-Rays
Not helpful.

VII. Special Studies
Magnetic resonance imaging demonstrates plica in most cases but is rarely necessary.

VIII. Diagnosis
Synovial plica.

IX. Differential Diagnosis
Other painful patellofemoral conditions; medial meniscus injury or loose body.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Simple external patellar support is indicated; physical therapy should include ROM and PREs if VMO is dysplastic; phonophoresis to plica area (injection may be helpful if recalcitrant); arthroscopic removal of plica for cases of chronic inflammation and fibrosis. Prognosis is good.

Consultation: persistent symptoms

XI. Complications
Degenerative articular changes.

XII. Prevention
None.

OSGOOD–SCHLATTER DISEASE (TIBIAL TUBERCLE APOPHYSITIS)

I. Epidemiology
Osgood–Schlatter apophysitis is seen in children 10 to 15 years old.[2] It is usually seen in boys who have recently experienced a growth spurt.

II. Mechanism of Injury
Repetitive microtrauma; rarely, acute onset of pain over tibial tuberosity.

III. Anatomy
Inflammation of apophysis of tibial tubercle due to relative inflexibility and shortening of infrapatellar tendon.

IV. Symptoms
Painful enlargement of tibial tuberosity.

V. Signs
Enlarged, tender tibial tuberosity; signs of malalignment (see Patellofemoral Syndrome section, above); tightness in hamstrings, heelcords, and quadriceps.

VI. X-Rays
Most are normal initially but then may show enlarged, irregular tibial tuberosity, loose ossicle separated from tuberosity, and patella alta.

VII. Special Studies
None indicated.

VIII. Diagnosis
Osgood–Schlatter apophysitis.

IX. Differential Diagnosis
Infrapatellar tendinitis, tumors, and avulsion fracture of tibial tuberosity.

X. Treatment
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Modify activity prescription; physical therapy (ROM and PREs) should use exercises to stretch hamstrings, heelcords, and quadriceps and strengthen VMO; use a simple patella cutout brace with inferior horseshoe; rarely, immobilization, casting, and surgery are necessary.

Consultation: prolonged disability not responding to conservative therapy

XI. Complications
Large exostosis if persistent.

XII. Prevention
Stretching; bracing; minimizing impact activity on hard surfaces.

OSTEOCHONDRITIS DISSECANS (OCD)

I. Epidemiology
Osteochondritis dissecans is a disorder in which a segment of bone and the overlying articular cartilage become separated from the rest of the bone. In patients under 15 years of age, it is generally not related to trauma. Patients over 15 are usually males who have experienced a traumatic incident (possibly a compression fracture). Other related factors are impairment of the blood supply to the affected area of the femur (avascular necrosis) and heredity.

II. Mechanism of Injury
May result from dislocation of the patella, ACL tear, or other trauma to the joint surface (e.g., twisting or a direct blow).

III. Anatomy
The majority of cases occur at the femoral condyles, but OCD may occur at the retropatellar surface as well. The lesion may be stable (still firmly attached) or unstable (only partially attached) or may be a loose body within the joint ("joint mouse").

IV. Symptoms
Nonspecific knee pain, stiffness, swelling; symptoms usually worse with activity (especially impact) and better with rest. May develop catching, locking, and buckling if loose bodies develop.

V. Signs
External tibial rotation when walking, effusion, quadriceps atrophy, tenderness.

VI. X-Rays
Cartilaginous fragments and very small, bony, loose bodies are not visible. Large lesions and significant bone fragments are usually visible.

VII. Special Studies
Magnetic resonance imaging is excellent for defining stage of lesion and planning treatment.

VIII. Diagnosis
Osteochondritis dissecans.

IX. Differential Diagnosis
Meniscal tear.

X. Treatment
For smaller or stable lesions, consider trial of rest from pain-producing activities (possible enforced rest in an immobilizer); arthroscopic removal of loose bodies may also be considered. Surgical debridement, internal fixation, or prolonged protected weight bearing may be necessary for large or unstable lesions. Defects may be treated in numerous ways. Recently, techniques involving osteochondral transplant (mosaicplasty) have shown good results for small femoral condyle lesions, whereas microfracture techniques may be better for larger lesions (although the resultant fibrocartilage does not perform as well as the native hyaline cartilage). Autologous chondrocyte transplantation is another evolving technology that holds much promise for the future; however, as with all of these techniques, the results are less than perfect and much more work remains to be done in this area.[23]

XI. Complications
Degenerative joint disease.

XII. Prevention
None.

CYSTS

A cyst is not a specific injury, but a fluid-filled lesion arising as a synovial outpouching, either into a normal bursal structure or into the soft tissue surrounding the knee. The patient will experience swelling in the medial popliteal space or over the meniscus, as well as discomfort when running full speed. Examination will reveal cystic swelling in the medial popliteal space or over the mid-joint line. X-rays may show soft-tissue swelling with a large popliteal cyst. An arthrogram may show dye extension into the cyst; MRI and ultrasonography can also be used. A popliteal (Baker's) cyst is almost always associated with an internal derangement of the knee. Other diagnoses include popliteal ganglion and meniscus cyst. Cysts may resemble other tumorous lesions. Treatment methods are aspiration and injection with a corticosteroid (rarely curative), surgical excision (usually curative), and treatment of underlying disorders (e.g., meniscal tear).

OVERUSE SYNOVITIS

At times of dramatic increase in activity, a patient may suffer a self-limiting synovitic reaction causing swelling and tightness in the knee. Previously unrecognized intraarticular injuries (meniscal, osteochondral, arthritic) predispose to synovitis. Apparently, even youngsters and "normal" adults can overdo activities such as running (marathons) and develop synovitis. Ice, compression, elevation, decreased activity, and judicious use of anti-inflammatory agents are the bases of treatment. If unresolved within 1 week, the definitive diagnosis should be sought.

SUMMARY

The knee is subject to a wide variety and large number of sports injuries. Both acute and overuse injuries are common. The complex structure of the knee often makes the diagnosis and treatment less than perfectly straightforward. Serious injuries such as cruciate ligament tears are often overlooked. For many acute knee injuries, especially meniscal tears and grade 3 sprains, the best treatment is and will remain a matter of clinical judgment and personal preference, and, therefore, of controversy. The diagnosis and management of chronic knee problems such as instability and disability due to pain or locking depend on a clear understanding of the functional anatomy of the joint and the mechanism of injury, as well as a careful physical examination. An aggressive therapeutic plan must include adequate rehabilitation and a preventive strategy stressing the correction of faulty biomechanics and a modification of the exercise prescription.

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52 LEG PAIN*

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INTRODUCTION

Leg injuries make up 7 to 12% of injuries presenting in the outpatient setting.[1,2] Among track and field athletes, a prospective study found that the leg was the most common site of injury, accounting for 28% of all injuries.[3] Among military personnel, 26 to 36% of overuse injuries occur in the leg.[4,5] This chapter focuses on the diagnosis and treatment of common causes of leg pain in the athlete. The differential diagnosis of leg pain is broad (see Table 52.1). Pain that is of gradual onset may be caused by other etiologies such as referred pain from lumbar disc disease, tumors, connective tissue disorders, and peripheral arterial disease. Active patients may attribute such symptoms to physical activity.

MEDIAL TIBIAL STRESS SYNDROME

Pain along the posteromedial border of the tibia has frequently been referred to as shin splints. The American Medical Association's *Standard Nomenclature of Athletic Injuries* (1966) defined shins splints as pain in the leg from repetitive running on hard surfaces involving forcible use of the foot dorsiflexors, excluding fractures and ischemic disorders.[6] Because of this rather imprecise definition, other terms have since been proposed, including soleus syndrome, medial tibial syndrome, and medial tibial stress syndrome.[7–9] This chapter utilizes the term *medial tibial stress syndrome* (MTSS), originally proposed by Drez,[8] to describe an overuse syndrome of the leg that produces pain of the posteromedial border of the distal tibia. Because of the varying terminology used, the prevalence of MTSS is not well described. In a study of track and field athletes, when stress fractures were excluded, shin pain was responsible for about 10% of all injuries.[3]

Etiology

The precise etiology of MTSS is not known. It appears to be due to a stress reaction along the distal two thirds of the posteromedial border of the tibia that involves the fascia, periosteum, or bone or a combination of these structures. The site of injury has been most consistently associated with the insertions of the soleus and the deep fascia along the posteromedial border of the distal two thirds of the tibia. The flexor digitorum longus and the tibialis posterior may also be involved (Level of Evidence B).[7,10–15] Early reports suggested that the tibialis posterior was the primary structure involved.[10,11] Studies using radionuclide bone scans and anatomic dissection provided evidence that the soleus and its fascia play a direct role in MTSS.[7,12] A dissection study concluded that the tibialis posterior arises from the distal third of the tibia, consistent with the location of MTSS symptoms.[13] A more recent anatomic study confirmed that the fibers of the soleus, the flexor digitorum longus, and the deep crural fascia attach consistently along the postermedial border of the tibia, where symptoms, exam findings, and bone scans have localized the injury.[14] The fibers of the tibialis posterior were found to be more laterally located. Finally, a study of runners with tibial pain who underwent magnetic resonance imaging (MRI) found that of those with medial pain consistent with MTSS had edema at the insertions of the soleus, flexor digitorum longus, and tibialis posterior.[15]

Symptoms

Medial tibial stress syndrome usually presents as dull, aching pain of gradual onset. The pain is located along the distal two thirds of the medial aspect of the leg. The symptoms may be bilateral. The pain develops with the onset of the activity but often dissipates as the exercise continues. Because of this, individuals with MTSS are often able to complete training sessions despite the

TABLE 52.1
Causes of Leg Pain in the Athlete

Bone
Medial tibial stress syndrome
Stress fractures of the tibia and fibula
Acute fractures
Contusions
Musculotendinous
Gastrocnemius strain (tennis leg)
Muscle cramping
Delayed onset muscle soreness
Contusions
Myopathies
Vascular
Popliteal artery entrapment syndrome
External iliac artery fibrosis
Adductor canal syndrome
Distal superficial femoral artery fibrosis
Deep venous thrombosis
Effort induced thrombosis
Peripheral vascular disease
Neurologic
Referred pain form the lumbar spine
Superficial peroneal nerve entrapment
Common peroneal nerve entrapment
Common peroneal nerve contusion
Compartment Syndromes
Chronic exertional compartment syndrome
Acute exertional compartment syndrome
Acute post-traumatic compartment syndrome
Other
Tumors
Connective tissue disorders (sarcoidosis, erythema nodosum, vasculitides)
Metabolic bone diseases

TABLE 52.2
Potential Risk Factors for Overuse Injuries

Intrinsic Factors
Previous injury
Menstrual dysfunction
Growth-related factors in children (increased susceptibility of growth cartilage to repetitive stress)
Inadequate conditioning
Psychological factors
Poor flexibility
Muscle weakness or imbalance
Anatomic malalignment
Joint laxity
Extrinsic Factors
Training progression (too rapid and/or inadequate rest)
Inappropriate equipment/footwear
Incorrect sport technique
Peer and/or adult influences
Uneven or hard surfaces

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injury. The symptoms typically return after exercise and then slowly improve with rest. With continued loading of the lower extremity, the symptoms become easier to provoke and may affect performance and daily activities. Neurologic and vascular symptoms are not associated with MTSS. Their presence should raise suspicion for an alternative diagnosis. Symptoms may occur in relation to abrupt increases in training frequency, intensity, or duration. Excessively worn footwear may also contribute (see Table 52.2). Because previous injuries to the lower extremity may increase the risk for overuse injuries such as MTSS, a history of such injuries and their treatment should be sought. As in other overuse injuries, determining when the pain occurs in relation to the onset of activity helps to estimate the severity of the injury (see Table 52.3).

Physical Examination

Tenderness along the posteromedial border of the tibia is present over a span of several centimeters, beginning above the medial malleolus and extending proximally. Slight swelling may also be seen. Resisted plantarflexion, dorsiflexion, inversion, and eversion does not usually provoke pain. Striking of the patient's heel with the examiner's hand (indirect percussion) is pain free, but hopping on the leg may cause pain. Those with MTSS demonstrate an increased maximum pronation velocity and maximum pronation, varus alignment of the hindfoot and/or forefoot, and a standing foot angle of less than 140°.[16,17] Footwear should be examined for signs of excessive wear and for their suitability for the sport.

TABLE 52.3
Clinical Assessment of Overuse Injury

Symptom Pattern	Severity	Reduction of Loading to Injured Site (%)
After activity only, or at onset, but not persisting	Low	≤25
During activity, late onset		>25–50
During activity, early onset		>50–75
Limits quality and/or quantity of training		>75
Prevents training	High	Complete rest

Source: Adapted from DiFiori, J.P. and Hosey, R., *Clin. Atlas Office Proc.*, 1(2), 227, 1998. With permission.

Imaging Studies

Radiographs of the leg are usually normal. A three-phase bone scan will demonstrate diffuse uptake along the posteromedial border of the tibia that is present only in the delayed phase.[18] (Bone scans of tibial stress fractures are abnormal in all three phases.) Magnetic resonance imaging may demonstrate periosteal edema with or without bone marrow edema (see Table 52.4).[15] In some cases, it may difficult to rule out a deep posterior compartment syndrome. If a stress fracture has been excluded by a bone scan or MRI and the symptoms do not improve with a program of rest and rehabilitation, compartmental pressure measurements should be considered. In patients with MTSS, compartmental pressures are normal.[8,19]

Treatment

In most cases treatment can be initiated without the need for imaging. Reduction of impact loading to the leg is the cornerstone of treatment. Complete rest may not be necessary (see Table 52.3). In most situations, only impact activities need to be curtailed (jumping, running). Non-impact activities such as cycling, swimming, or pool running should be used to maintain conditioning. Ice can provide local pain relief. Iontophoresis and ultrasound have also been used but without definite evidence of benefit. Soft-tissue treatment (e.g., digital pressure, transverse friction, and myofascial release) may be helpful. For patients who have pain with daily activities, a long pneumatic splint is helpful in reducing symptoms. Acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs) can help to alleviate pain. Given the lack of consistent evidence of an inflammatory reaction in MTSS, the use of NSAIDs for the purpose of reducing inflammation is controversial.[8,20,21] Developing strength and flexibility of the entire lower extremity should be emphasized, including the heel cord and dorsiflexors. Dietary calcium intake should be reviewed for women and adjusted to age-appropriate levels. Evaluation and management of menstrual dysfunction should be initiated if appropriate. If an eating disorder is suspected, consultation should be obtained.

Should patients with MTSS be prescribed orthotic devices to correct malalignment of the foot and ankle? Limited prospective evidence suggests that lower extremity malalignment plays a causal role in the development of MTSS. Modification of malalignment with orthoses should be

TABLE 52.4
Magnetic Resonance Imaging Grading of Tibial Stress Injuries

	Magnetic Resonance Findings			
Grade	Periosteal Edema	Marrow Edema	Fracture Line	Clinical Correlation
1	Mild to moderate on T2-weighted images	None	None	Stress reaction
2	Moderate to severe on T2-weighted images	Seen on T2-weighted images	None	Stress reaction
3	Moderate to severe on T2-weighted images	Seen on T1- and T2-weighted images	None	Stress fracture
4	Moderate to severe on T2-weighted images	Seen on T1- and T2-weighted images	Visible	Stress fracture

Source: Adapted from Fredericson, M. et al., *Am. J. Sports Med.*, 23(4), 472–481, 1995. With permission.

considered on an individual basis (Level of Evidence B, nonrandomized clinical trial, nonquantitative systematic review).[16,17,22–29]

Biomechanical features such as foot pronation, pronation velocity, standing Achilles tendon angle measures, arch index, and other forefoot and hindfoot abnormalities have been found to be associated with MTSS.[16,17,22,23] Prospective evidence is quite limited. Of the available prospective studies, most have studied alignment in relation to lower extremity overuse injury in general. Individually, they have reported a number of alignment measures to be risks for injury, including arch height, severe knee valgus, pes cavus, pes planus, and quadriceps angle greater than 15°;[24–27] however, no measure of alignment has been consistently found to increase risk of injury. Furthermore, variations in study methodology make it difficult to draw any conclusions.[28]

Few prospective studies have specifically reported findings for the relationship between alignment and MTSS. Wen and colleagues[29] found a greater knee tubercle sulcus angle and knee varus to be risks for shin injury. When a cause for MTSS injury can be readily identified (such as a rapid increase in weekly running mileage), correction of malalignment may not be a necessary component of treatment. In other cases, especially those in which rehabilitation measures have failed, the injury is recurrent, or the malalignment is severe, over-the-counter or prescription orthoses may be beneficial.

Return to Play

Return to impact-loading activities occurs gradually. Training should begin on an alternate day schedule at low intensity levels, preferably on softer surfaces. Training progresses using symptoms and physical findings as a guide. Intensity of training is adjusted as more volume is tolerated. The return to unrestricted activity is variable. Typically, a 6-week time period is required; however, the overall recovery can be more prolonged in some cases. A pneumatic stirrup splint can decrease recovery time.[30]

Prevention

Review the factors that led to the injury and develop a specific plan to mitigate those factors. This should include a review of the training program, along with an ongoing conditioning program. Footwear should be appropriate for the activity and replaced at regular periods to prevent excessive wear that may cause changes in lower extremity loading.

Surgical Consultation

Consider orthopedic consultation in cases that fail to improve with rest from impact activity and a thorough trial of rehabilitation. Posteromedial fasciotomy with release of the medial soleus fascial bridge may be indicated in such instances.[7]

STRESS FRACTURES OF THE TIBIA AND FIBULA

Epidemiology

The tibia and fibula are common sites for stress fractures. The true incidence is not known. In studies in which stress fractures were confirmed by imaging modalities, the tibia was the site for 19 to 55% of these injuries, while the fibula accounted for up to 30%.[31] Of tibial stress fractures, the most common location is at the level of either the proximal or distal one third of the shaft. Of fibular stress fractures, the distal shaft is the most common site.

Etiology

Bone is a dynamic tissue that remodels in response to loading. Stress injuries occur as a result of the inability of bone to effectively remodel in response to repetitive stress. The etiology is multifactorial. Contributing factors include external forces such as ground reaction forces, repeated muscle contractions across the bone, and muscle fatigue. Strain rate, rather than the absolute magnitude of strain placed upon bone, may be a critical factor for stress fracture development.[52] These external factors influence the many intrinsic activities that control the balance of bone resorption and formation. Finally, individual susceptibilities that affect bone resorption and formation (such as menstrual dysfunction in female athletes) also play a role.

Symptoms

Symptoms include a gradual onset of aching pain that is often well localized. In some cases, the pain onset can be sudden. Initially, the pain occurs only with exercise and is relieved by rest. If untreated, symptoms increase and begin to limit the activity. When severe, the pain can be present with walking and during routine daily activities. Nighttime symptoms can also occur. Inquire about risk factors for overuse injury:

- Does the athlete have a history of previous stress fractures?
- Does the female athlete have a history of menstrual dysfunction?
- Are features of disordered eating present?

- Did the athlete change exercise intensity, frequency, duration, or mode in the weeks preceding the onset of symptoms? Was the change made in a gradual fashion?
- Has the athlete been running on a different surface than usual?
- Is the footwear excessively worn? Did the athlete recently change footwear?

Physical Examination

Begin by inspecting the entire lower extremity and compare it to the opposite side. Note any features of malalignment that may affect force distribution. The presence of muscle imbalance, weakness, restricted range of motion, and poor flexibility should also be noted. Carefully palpate the involved area. In most cases, the tenderness is well localized. Swelling may be present. Periosteal thickening may be palpable when symptoms have been present for several weeks. Hopping on one foot often reproduces symptoms. Indirect percussion (percussing at a distant point on the tibia or fibula or striking the heel) may also produce pain. Resisted manual muscle testing usually does not produce symptoms. Application of a vibrating tuning fork or ultrasound to the site in an attempt to reproduce symptoms has generally *not* been helpful.[33,34] Inspect the athlete's footwear for signs of excessive or uneven wear.

Imaging Studies

Imaging studies are routinely performed to exclude other causes of bony leg pain such as tumors and to confirm the diagnosis. Exercise-related leg pain accompanied by well-localized bony tenderness on exam is very suggestive of a stress fracture. In such cases, the exam findings have been shown to correlate well with MR imaging.[15]

Radiographs

Radiographs have a relatively low sensitivity for the diagnosis of stress fractures, particularly if the symptoms have been present for less than 2 to 3 weeks. When present, findings include a localized periosteal reaction, sclerosis, callus formation, or a lucent fracture line. Repeat radiographs may ultimately demonstrate the stress fracture, but it may be weeks or months before this occurs. In some cases, radiographs will remain unremarkable.[18]

Bone Scans

Three-phase radionuclide imaging is highly sensitive. All three phases of the study are abnormal in stress fractures. The delayed phase demonstrates a focal uptake pattern. While bone scans are very sensitive, they can be non-specific. Scans frequently show multiple areas of abnormal uptake that are not in the area of clinical concern.

Magnetic Resonance Imaging

Magnetic resonance imaging has emerged as the imaging study of choice in some centers.[18] It is of comparable sensitivity to the three-phase bone scan. MRI also depicts the surrounding soft tissues and provides greater specificity for bony injury. Grading criteria for tibial stress fractures have been described based upon the presence or absence of periosteal edema, marrow edema, and a visible fracture line.[15] Preliminary data suggest that the MRI findings are predictive of recovery time.[35]

TREATMENT

The mainstay of treatment for all stress fractures is rest from activities that stress or load the injured site. All activities that produce pain should be discontinued. If daily activities are painful, a long pneumatic splint is helpful.[34] A walking boot may be used in cases in which a pneumatic splint is not effective. Non-weight-bearing or partial weight-bearing with crutches can be used if such devices are not sufficient to eliminate pain. Acetaminophen may be used for pain relief, if needed. The use of NSAIDs, including COX-2 selective agents, has been shown to impede fracture healing.[36,37] Although the effects of NSAIDs on the healing of stress fractures is not known, the use of alternative analgesics should be considered. Other activities that do not load the lower extremity should be implemented to maintain fitness levels. Cycling, swimming, and pool running are examples. An elliptical trainer may also be used on an individualized basis. Flexibility training and progressive resistance exercises can be performed throughout the rehabilitation process if they do not cause pain. A gradual return to loading activities is begun once pain with daily activities and focal tenderness have resolved. If pain recurs as activities are reintroduced, the progression should be halted until the patient has been without pain for 2 to 3 days. When functional skills can be performed without pain, sport-specific training may begin.

Return to Play

Return to play is allowed when the patient demonstrates the ability to train in an unrestricted fashion without symptom recurrence. Return to play can be considered when this is achieved even if a splint or brace is being used during training (and will therefore be used for competition). Matheson and colleagues[38] reported that the

average time to recovery for tibial stress fractures was approximately 12 weeks, and for fibular stress fractures 8 weeks.

Is splinting or bracing effective in speeding up recovery time for stress fractures of the tibia and fibula? The use of a pneumatic stirrup splint can accelerate the return to activity (Level of Evidence A, randomized controlled trial, nonrandomized clinical trial).[30,34,39] Two uncontrolled studies using a pneumatic stirrup splint at the time of diagnosis of either a tibial or fibular stress fracture reported a quicker return to activity. In one study of 42 patients (17 with stress fractures and 25 with MTSS), the average time for return to competition was 5.3 weeks.[30] In another study of 13 female athletes with stress fractures (10 of the tibia, 3 of the fibula), all returned to play immediately after application of the stirrup splint. Two continued to have some pain while wearing the splint; the others were asymptomatic. All had complete symptom resolution within 1 month.[39] These studies both found a much sooner return to activity than previous reports.[38] Finally, a single randomized, prospective study of 18 tibial stress fractures found that those using the stirrup splint became pain free sooner (14 days vs. 45 days) and returned to play significantly faster (21 days vs. 77days) than those not using a splint.[34]

Prevention

Review any risk factors for overuse injury identified during the evaluation. A specific plan to address the factors that may have led to the stress fracture should be developed. Training progression, scheduled rest periods, and a program to maintain strength and flexibility should be discussed. Evaluate and treat menstrual disturbances and disordered eating behavior, if such concerns are present. Calcium supplementation should be encouraged for all women.

Can stress fractures be prevented by the use of custom orthoses? Evidence is insufficient to recommend the use of custom orthoses for stress fracture prevention in running athletes (Level of Evidence B, randomized controlled trial, non-randomized clinical trial).[40–43] Studies of the effectiveness of shock-absorbing shoe inserts and biomechanical orthoses in reducing the incidence of stress fractures have been conducted in military populations. Two earlier prospective studies of military recruits using shock-absorbing shoe inserts in military boots did not find a reduction in stress fracture incidence.[40,41] A prospective randomized trial evaluating custom biomechanical shoe orthoses in military boots found a 50% reduction in stress fracture incidence among infantry recruits.[42] A recent *in vivo* study of tibial strain and tibial strain rates with the use of custom biomechanical shoe orthoses among military recruits found a favorable effect when the devices were used while walking in military boots;[43] however, no beneficial effect was found when the orthoses were used while running either with military boots or running shoes.

STRESS FRACTURES OF SPECIAL CONCERN

Anterior Cortex of the Tibia

These stress fractures often develop complications such as delayed union, nonunion, and complete fracture. Radiographs demonstrate disruption of the anterior cortex (the dreaded black line), thickening of the anterior tibial cortex, and narrowing of the medullary canal. Stress fractures occurring at this site should generally be referred to an orthopedic surgeon. Conservative treatment involves a period of prolonged rest from impact loading with or without a cast or modified immobilization. Return to activity has been reported to take from 8 to 12 months.[45,46] Because of the difficulty healing these fractures with conservative treatment, some authors recommend surgical treatment as the treatment of choice.[44] Operative treatment options include intramedullary nailing and bone grafting.[47,48]

Longitudinal Stress Fractures of the Tibia

These stress fracture should be considered in patients presenting with poorly localized distal leg pain. Fewer than 50 cases have been reported in the literature.[49] Radiographs are often negative. A bone scan is abnormal, but the appearance is variable. CT or MRI is usually needed to make the diagnosis. Management is similar to that for stress fractures of the proximal or distal tibia occurring at the posteromedial tibial border.

Proximal Fibula

The proximal fibula is a rare location for fibular stress fractures; just nine cases have been reported in athletes.[50] The most frequent reports involve military personnel. Clinically, two distinct types of proximal fibular stress fractures have been identified. In one type, the symptom onset is abrupt, and radiographs demonstrate a frank fracture. In the second type, the onset is typical of other stress fractures with a gradual onset and variable radiographic findings. Both types of proximal fibular stress fractures heal uneventfully. Because the location is unusual, other causes

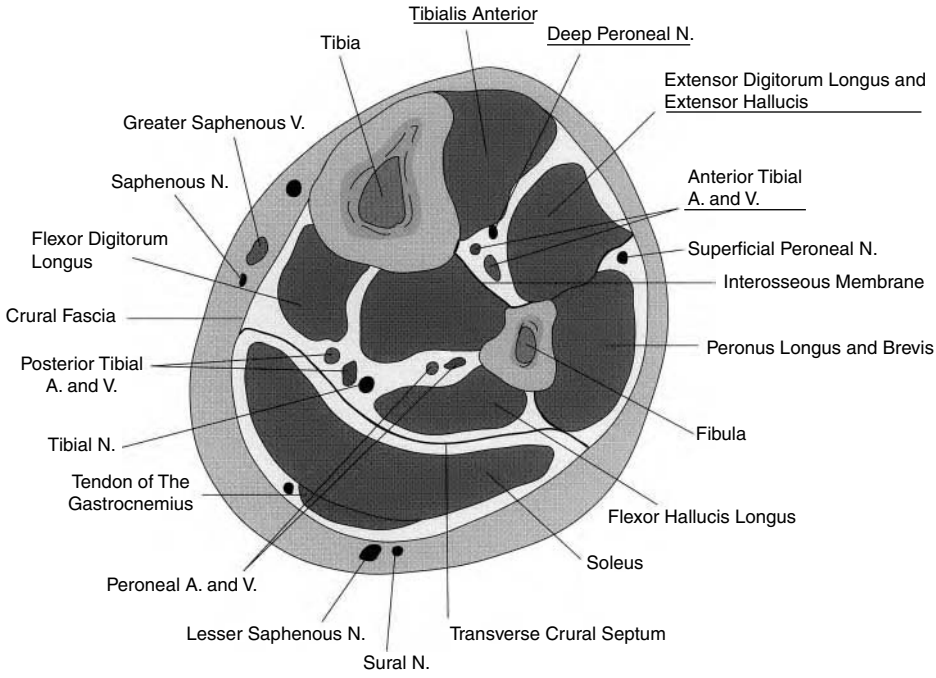

Figure 52.1 Cross section of the lower right leg, illustrating the four fascial compartments and their components. (Anterior compartment structures are underlined.)

(e.g., tumors) should be excluded with the use of imaging studies.

EXERTIONAL COMPARTMENT SYNDROMES

Introduction

Compartment syndromes are a result of an increase in tissue pressure within a closed fascial space. This is believed to cause a decrease in perfusion, damaging the intracompartmental structures.[51] An acute compartment syndrome is a surgical emergency. Most occur after trauma, such as limb fractures. Acute compartment syndromes due to trauma are uncommon in athletes but may occur with injuries such as tibial fractures and muscle ruptures. Acute exertional compartment syndromes (AECSs) occur in the absence of blunt trauma and are also rare. The most common type of compartment syndromes in athletes are chronic exertional compartment syndromes (CECSs). In these cases, recurrent leg pain occurs with activity and resolves with rest. Acute and chronic exertional compartment syndromes are injuries typically associated with athletic activities and are discussed in this section.

Chronic Exertional Compartment Syndrome

Anatomy

The leg contains four fascial compartments: anterior, lateral, deep posterior, and superficial posterior (Figure 52.1). The structures within each compartment are as follow:

- Anterior compartment — tibialis anterior, extensor digitorum longus, extensor hallucis longus, deep peroneal nerve, anterior tibial artery, and anterior tibial vein
- Lateral compartment — peroneous longus and brevis, superficial peroneal nerve
- Deep posterior compartment — popliteus, tibialis posterior, flexor digitorum longus, flexor hallucis longus
- Superficial posterior compartment — gastrocnemius, soleus, plantaris

The tibial nerve, posterior tibial artery, and peroneal artery supply both posterior compartments.

Epidemiology

The prevalence of CECS of the leg is not known. In one study of patients referred specifically for

evaluation of suspected CECS, 26 of 98 had the diagnosis confirmed by intramuscular pressure measurements.[52] Of over 2400 patient visits to a sports medicine center, 3.5% were diagnosed with compartment syndromes.[1] The anterior and deep posterior compartments are the most common sites for CECS.[51]

Etiology

Chronic exertional compartment syndromes involve an increase in intracompartmental pressure during exercise that resolves with rest.[51] The mechanism is not well understood. It has been thought that the increase in intracompartmental pressure, due in part to the increase in muscle volume that occurs with exercise and an increase in extracellular fluid, results in relative muscle ischemia;[53] however, two studies have disputed the role of ischemia in CECS.[54,55]

Symptoms

The characteristic symptom is leg pain over the involved compartment that occurs only with exercise. The pain is described as cramping, aching, or a tightness. Bilateral involvement is common. Transient symptoms of numbness or weakness related to the involved nerve can also develop (e.g., paresthesias of the first web space and dorsiflexor weakness in an anterior compartment syndrome). Typically, the symptoms progress during exercise, limiting the ability to continue training. Usually the pain resolves once the activity is stopped. In some cases, the symptoms may persist for several hours. The symptoms are often described as being very predictable in onset, returning after a specific amount of exercise has been performed.

Physical Examination

The physical examination is typically normal. Having the patient exercise to provoke the symptoms may yield neurologic findings and may produce a palpable tightness or tenderness over the involved compartment. The neurologic findings are specific to the involved compartment: Anterior compartment CECS can cause dorsiflexor weakness and paresthesias of the first web space. Lateral compartment CECS can cause anterolateral paresthesias and paresthesias of the dorsum of the foot; weakness may be seen in eversion and dorsiflexion. Deep compartment CECS affects the tibial nerve, producing paresthesias of the arch of the foot; passive stretching may increase the symptoms. The pulses are normal. If present, a palpable fascial hernia is highly suggestive of CECS.

TABLE 52.5
Diagnosis of Chronic Exertional Compartment Syndrome (CECS)

Time of Measurement	Intramuscular Pressure (mmHg)
Before exercise	≥15
1 minute after exercise	≥30
5 minutes after exercise	≥20

Note: One or more of the above readings is considered diagnostic of CECS.

Source: From Pedowitz, R.A. et al., *Am. J. Sports Med.*, 18, 35–40, 1990. With permission.

Special Studies

Radiographs are normal but assist in excluding stress fractures and bone tumors. The role of bone scans and MRI in the evaluation of CECS is not clear. They are probably most useful in ruling out other causes of leg pain (e.g., stress fractures, MTSS, soft tissue and bony tumors).[55,56] A controlled study of MRI and CECS, however, reported an increase in T2-weighted signal intensity in the involved compartment.[56] Nerve conduction studies may assist in excluding nerve entrapment syndromes. The standard of diagnosis is elevated intracompartmental pressures that occur in association with leg pain (see Table 52.5).[57]

Treatment

Decreasing exercise relieves symptoms; however, symptoms typically recur with attempts at resuming full activities. Little evidence suggests that conservative measures such as flexibility training and physical therapy are effective in allowing a return to the previous activity level. A pilot study of flexibility training and massage therapy showed limited benefit in symptom improvement and did not alter post-exercise compartment pressure measures.[53] Referral for surgical decompression of the involved compartment(s) is frequently necessary. Success rates of nearly 90% have been reported with fasciotomy for those with CECS of the anterior compartment.[51] The surgical results for patients with deep posterior compartment syndromes, however, are less favorable.[51] In one retrospective study of 62 patients treated for CECS, 81% (26 of 32) of those with anterior or lateral compartment involvement had significant improvement compared to 50% of those with deep compartment involvement (3 of 6).[59] Some authors prefer fasciectomy, either alone or in

combination with fasciotomy, as the primary procedure. Recently, endoscopic fasciotomy techniques have been described.[61] The recurrence of symptoms after fasciotomy may indicate insufficient fascial release, excessive post-operative scarring, or inaccurate initial diagnosis.[53]

Acute Exertional Compartment Syndrome

Etiology

Prolonged and irreversible tissue pressure elevation occurs within the fascial compartment.[62] Without rapid decompression, nerve and muscle capillary perfusion is compromised, leading to muscle necrosis with or without permanent neurologic damage.

Symptoms

Severe pain over the involved compartment that develops either during exercise or within several hours following the activity. The pain is not relieved with rest. AECS often occurs in an unconditioned individual who performs a large amount of exercise. Although not typical, a preceding history of symptoms consistent with CECS has been reported.[63]

Physical examination

Pain out of proportion to the perceived severity of injury is noted. The skin may appear shiny and feel warm. Other findings include paresthesias, palpable tension or firmness over the involved compartment, increased pain with passive muscle stretching, and muscle weakness. Pulses are often present. If absent, severe ischemia and/or arterial injury has occurred.[62]

Special Studies

Acute exertional compartment syndrome is largely a clinical diagnosis. Intracompartmental tissue pressure measurements can aid in making the diagnosis; however, false positives and negatives do occur.[62] Pressure measurements ranging from 30 to 45 mmHg have been proposed as indications for fasciotomy. However, because a critical factor in the development of muscle ischemia is the difference between the mean arterial and compartment pressures, intracompartmental tissue pressure measurements should be interpreted in the context of the patient's systemic pressure.[62]

Treatment

Fasciotomy is the definitive treatment for AECS. Immediate referral to an orthopedic surgeon should be made in all cases of suspected AECS. All constrictive materials (clothing, splints, dressings, casts, walking boots) should be removed. Ice should not be used as it may interfere with the microcirculation. The leg should be placed at the level of the heart. Early intervention improves outcomes and reduces complications, including renal failure from myoglobinuria, arrhythmias, amputation, or death. In one report, 68% of those with AECS who had fasciotomy within 12 hours after the onset of symptoms regained extremity function vs. 8% who received fasciotomy beyond 12 hours.[64]

Acute Gastrocnemius Strain ("Tennis Leg")

Anatomy

The gastrocnemius muscle spans two joints. Proximally, the medial and lateral heads insert in the supracondylar region of the femur. Distally, the gastrocnemius combines with the soleus to form the Achilles tendon, which inserts onto the calcaneus.

Etiology

Injury of the medial head occurs at the myotendinous junction when ankle dorsiflexion and knee extension occur simultaneously with muscle contraction. This can occur when leaping or when sprinting forward from a "ready" position. In such cases, the knee is initially in a position of flexion and the ankle is in dorsiflexion. The injury occurs when the athlete then suddenly extends the knee in combination with contraction of the gastrocnemius. The injury has been referred to as "tennis leg" because of the propensity for this injury to occur in this way among middle-aged tennis players. The injury may also occur in runners when the gastrocnemius contracts eccentrically to control the dorsiflexion produced by the ground forces that occur during the contact phase. This is most pronounced when the footstrike occurs on a curb or the edge of step. In either case, a previous strain injury, muscle soreness, tightness, or intermittent sharp pain (a "twinge") may be noted to precede the acute injury.

Symptoms

Sudden onset of posterior leg pain is typically medially located and is often described as a stabbing or tearing sensation, as if being shot or kicked in the leg. Patients are frequently unable to continue the activity and usually have pain with walking after the injury. After the acute pain dissipates, a sensation of a painful knot at the site of injury may be described.

Physical Examination

Tenderness to palpation over the medial head of the gastrocnemius in the region of the distal myotendinous junction is noted. Swelling and ecchymoses may be seen. A palpable defect in the muscle may be present. It may initially be obscured due to swelling. Passive ankle dorsiflexion with the knee extended causes pain. The patient may have difficulty performing a single leg or double leg heel raise. Hopping on the injured leg causes pain in most cases but doing so should be avoided when the injury is obvious.

Special Studies

In most cases, imaging studies are generally not needed to establish the diagnosis. The setting in which the injury occurs and the exam findings usually permit the diagnosis to be made. In the absence of a compelling injury mechanism, it may be difficult to distinguish the injury from a deep venous thrombosis (DVT). A Doppler study may be used to exclude the diagnosis of a DVT. If this is not revealing, venography should be performed. Treating a suspected DVT without confirming the diagnosis can be disastrous, as the use of anticoagulants in a patient with an acute gastrocnemius strain can result in an acute compartment syndrome due to bleeding.[65] Ultrasound or MRI may be used to confirm the diagnosis of an acute gastrocnemius strain if needed; however, in the vast majority of cases, the history and physical exam are sufficient to establish the diagnosis.

Complications

Deep venous thrombosis and acute compartment syndrome are rare but serious complications. Thrombophlebitis and DVT formation can occur when leg swelling impedes venous return. An acute compartment syndrome can develop as a direct complication of the muscle injury.

Treatment

Acute Phase

Frequent ice applications, the use of a compression wrap, elevation, and a heel lift are used to limit swelling and pain. Use of the heel lift should be limited to no more than 2 weeks to avoid loss of flexibility. Crutches (non-weight-bearing) should be used for the first 48 hours. Active range of motion exercises may be performed if pain free. Transition to partial and then full weight bearing occurs over the first week.

Subacute Phase

Gentle range of motion and flexibility exercises can be performed as pain decreases. Add strengthening exercises within the pain-free range of motion. It is important that these exercises do not produce pain. Begin with a bilateral heel raise, on a flat surface. As symptoms and function improve, perform heel unilateral heel raises on a flat surface, then on a step (increasing the range of motion). Weights can then be added for further strengthening. The eccentric phase should be emphasized. Single leg hopping and the use of a recumbent sled can be added to provide controlled plyometric training. Ice should be applied after rehabilitation sessions.

Return to Play

Return to play is attempted when full pain-free range of motion has been restored, strength is within 90% of the contralateral leg, and sport-specific skills are pain free. Recovery may be as brief as 2 weeks for mild strains or 8 weeks or longer for severe injuries. An ongoing program of flexibility, resistance training, and plyometrics is important to prevent reinjury.

POPLITEAL ARTERY ENTRAPMENT SYNDROME

Introduction

Leg pain caused by popliteal artery entrapment syndrome (PAES) is rare. The incidence is not known; 80% of reported cases have been in men, and more than half are under 30 years of age.[66] Although both legs may not be symptomatic, the syndrome is present bilaterally in 33%.[66] Complications consist of occlusive disease and its complications, including limb loss.

Etiology

Popliteal artery entrapment syndrome involves intermittent compression of the popliteal artery. Two types of PAES have been described: anatomic and functional. Anatomic, or classical, PAES occurs as a result of an abnormal anatomic relationship of the popliteal artery and the medial head of the gastrocnemius. The Heidelberg classification has three categories: Type I is an abnormal course of the popliteal artery (it runs medial to the medial head of the gastrocnemius); type II is abnormal muscular insertion that causes compression of a normally situated artery; and type III is a combination of types I and II.[67] Functional PAES has been described by Rignault et al.[68] as claudication symptoms that occur with an anatomically normal popliteal artery and gastrocnemius.

In athletes, this has been thought to be due to muscular hypertrophy that results in lateral deviation of the popliteal artery and nerve with plantarflexion causing compression against the lateral condyle of the femur.[69,69]

History

The patient has a history of intermittent posterior leg pain or cramping with exercise. Paresthesias, coldness, and pallor of the foot may occur. Symptoms may develop with walking but not running. Acute occlusion of the artery can cause sudden symptom onset. The symptoms may be unilateral, even when both legs are affected. The symptoms typically improve with rest.

Physical Examination

Exam may be normal. Be sure to examine both legs even if only one is symptomatic. Obliteration of the dorsalis pedis pulse with resisted plantarflexion or passive dorsiflexion suggests PAES but can be observed in normal individuals. A popliteal aneurysm or bruit may be present.

Diagnostic Tests

Doppler studies are normal unless an occlusion is present. A duplex-scan examination with pulsed-Doppler of the popliteal fossa may reveal an abnormal popliteal artery course or abnormal spacing between the artery and vein due to an interposing structure.[66] Dorsiflexion and/or plantarflexion during the study can demonstrate entrapment. MRI and MR angiography have recently been reported to be useful in the diagnosis of PAES.[69,70] The diagnostic benchmark remains bilateral arteriography. Because compression of an anatomically normal popliteal artery may occur with provocation in asymptomatic individuals, it may be difficult to distinguish cases of functional PAES from a compartment syndrome. In such cases, intracompartmental pressure measurements should be considered.

Treatment

Surgical release is the most common procedure.[66] Thromboendarterectomy and/or grafting procedures are also performed if necessary. Graft occlusion and recurrent ischemia are potential postoperative complications.[67] For functional PAES, release of the soleus muscle from its tibial insertions with resection of its fascial band and the plantaris muscle has been described.[69] Data on long-term outcomes following surgical procedures to treat PAES are limited. Hoelting and colleagues[67] reviewed 19 patients (23 limbs) who had surgery for classic PAES; 16 of the 23 extremities were free of complications an average of 9.5 years post-operative. The outcomes for those surgically treated for functional PAES is somewhat better.[69]

OTHER VASCULAR CAUSES OF LEG PAIN

External Iliac Artery Fibrosis

External iliac artery fibrosis was first described by Wolden et al.[73] in two cyclists. Multiple cases in cyclists have been reported since.[74] Other cases, including dissection and occlusion, have been reported in runners and ultra-endurance athletes.[75–77] The condition is thought to develop from chronic stress of the arterial wall producing intimal hyperplasia and stenosis.[71] A recent histopathologic study found medial and advential thickening in addition to intimal fibrosis.[72] The most typical symptom is exercise-related thigh pain, although leg pain can occur.[70] The physical examination is usually normal. In some cases, a bruit may be auscultated over the femoral artery when the hip is flexed.[74] The ankle–arm index can be used as a screening test comparing pre- and post-exercise ratios.[78] Color Doppler may demonstrate kinks and intravascular lesions.[79] The diagnostic study of choice is arteriography.[70] Treatment has been described using angioplasty techniques, surgical bypass, and open endarterectomy.[70] Return to competitive sports after interventional procedures has been reported, but no long-term follow-up data are available.[74]

Lesions Affecting the Distal Segment of the Superficial Femoral Artery

Adductor canal syndrome is caused by external compression of the superficial femoral artery (SFA) by an abnormal musculotendinous band.[80] Intimal hyperplasia of the SFA has also been reported.[81] Both can affect the distal portion of the SFA and produce exercise-associated leg pain. MR angiography can be diagnostic. Surgical release is performed for treatment of adductor canal syndrome.[80] An autologous saphenous vein patch has been described as treatment for intimal hyperplasia of the SFA.[81]

SUPERFICIAL PERONEAL NERVE ENTRAPMENT

Introduction

This syndrome is an uncommon cause of anterolateral leg pain. Among 480 patients studied with chronic leg pain, only 3.5% were found to have it.[82]

Etiology

Entrapment of the superficial peroneal nerve usually occurs as the nerve emerges from the deep fascia in the lower one third of the leg. It then divides into two subcutaneous sensory branches that supply the dorsum of the foot. The syndrome may develop as a result of:[82]

- Previous trauma
- Inversion ankle injuries
- Muscle herniation through a fascial defect
- An abnormal course of the nerve
- A post-operative complication of surgery for chronic anterior compartment syndrome[82]

History

Patient reports a history of anterolateral leg pain. Pain symptoms may include the dorsum of the foot. Loss of sensation of the dorsum of the foot may also occur. Symptoms are typically chronic as a delay in diagnosis of months to years is common.[82]

Physical Examination

Styf and Morberg[82] have described three provocation tests:

- Pressure applied 8 to 10 cm above the lateral malleolus, over the anterior intermuscular septum, during active ankle dorsiflexion
- Passive plantarflexion and inversion of the ankle
- Pressure over the course of the nerve as the ankle is plantarflexed and inverted

A clinical diagnosis of superficial peroneal nerve entrapment is then made if pain and loss of sensation over the dorsum of the foot are experienced at rest or during an exercise test *and* the first two of the provocation tests are positive.

Diagnostic Studies

Radiographs of the leg are normal. Nerve conduction studies are also usually normal. Intracompartmental pressure measurements may be helpful in ruling out a chronic lateral compartment syndrome. A diagnostic injection of lidocaine can be performed at the painful site above the lateral malleolus.[83] MRI may confirm the entrapment.[83]

Treatment

Surgical decompression is the recommended treatment. A complete opening of the superficial peroneal tunnel is usually performed. If the peroneal tunnel is absent, fasciectomy is performed. A complete fasciotomy is needed if an associated chronic lateral compartment syndrome is present. The benefits of surgery appear somewhat limited. Styf and Morberg[82] reported that 82% were satisfied with the outcome of the procedure 26 months post-operatively; however, 72% reported residual symptoms, with nearly half reporting pain with walking. Four of 17 were reported as having returned to an unlimited level of activity.[82]

COMMON PERONEAL NERVE ENTRAPMENT

The common peroneal nerve branches from the sciatic nerve above the popliteal fossa. It runs laterally between the two heads of the peroneous longus, entering the peroneal or fibular tunnel where it divides into the deep, superficial, and recurrent peroneal nerves. The incidence of common peroneal nerve entrapment is not known. Cases have been reported in runners and in a soccer player;[84,.85] Stack and colleagues[86] reported on nine cases caused by compression of the nerve by ganglion cysts. Symptoms include lateral leg pain and paresthesias of the leg and dorsum of the foot. Symptoms are provoked with exercise and typically resolve with rest.[85] The exam can be normal. A positive Tinel's at the fibular neck can be elicited in some cases. Post-exercise examination may reveal muscle weakness. The diagnosis can be confirmed with nerve conduction studies. Surgical treatment appears to be effective in the limited cases described.[84–86]

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53 THE ANKLE

Richard B. Birrer

INTRODUCTION

The ankle is a key focal point in the transmission of body weight, capable of the adjustments necessary for fine balance on a wide variety of terrains. The ankle is often involved in static and dynamic deformities that ordinarily do not affect other parts of the body because the joint is subject to the concentrated stresses of standing and movement. Ankle injuries are not always minor and may be associated with prolonged disability and recurrent instability in 25 to 40% of patients for several months to several years; therefore, a casual approach (e.g., "It is only a sprain") to the diagnosis and management of these injuries is inappropriate. The ubiquitous and unpredictable nature of ankle injuries mandates a precise understanding of the mechanism of injury, a thorough knowledge of the anatomy of the ankle joint, a clear ability to assess the degree of damage, and a solid understanding of appropriate treatment modalities in the acute and rehabilitative phases.

GENERAL EPIDEMIOLOGY

The majority of sports injuries (55 to 90%) involve the lower extremity, with the knee, ankle, and foot being the most common sites.[1] Ankle sprains are probably the most common (15%) generic sports injury, with over 2 million annually and an

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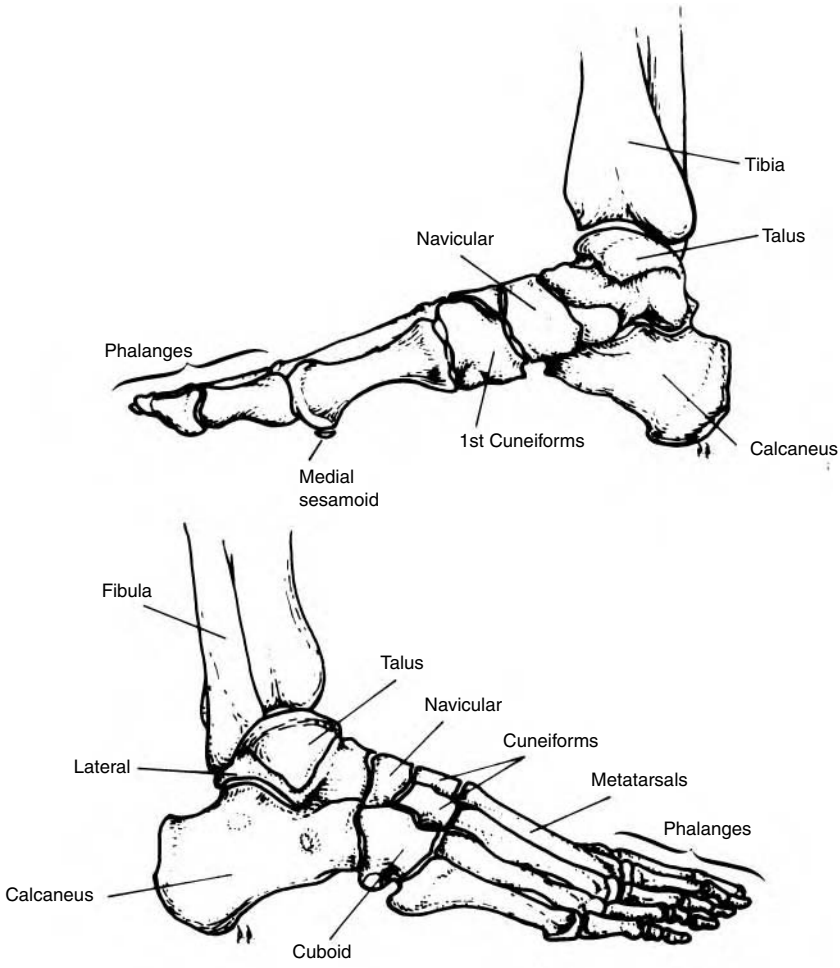

Figure 53.1 The bony structure of the ankle.

incidence rate of 1/10,000 person-days. While most ankle injuries follow acute trauma, 25 to 30% of ankle injuries are of the overuse category. Low-velocity athletics (<20 mph) such as volleyball, baseball, basketball, football, tennis, racquetball and others such as track and field events, swimming, and golf produce strains, sprains, and occasionally simple fractures such as nondisplaced fibular fractures of the ankle. These simple injuries are usually best dealt with nonoperatively (RICE: rest, ice, compression, elevation). Simple bony injuries generally require 4 to 6 weeks of casting or bracing with weight-bearing during the last 2 weeks. Complex and severe fracture/dislocation problems are rare. Ligamentous injuries are rare in children and adolescents; trauma usually involves the growth plate in the form of fracture.

High-velocity sports such as skydiving, skateboarding, ice hockey, in-line skating, and downhill skiing or sports that use machinery or animals (e.g., snowmobiling, horse racing, motor racing) produce accelerations exceeding human capabilities. The resulting ankle injuries are typically open and associated with other injuries. Such fractures may be quite comminuted and widely displaced and may disrupt major portions of the tibial articular surface. Their management is complex and usually is handled through the consultative process. An intensive rehabilitation program must follow all ankle trauma.

ANATOMY

The mortise or hinge joint of the ankle (Figure 53.1) is formed by the distal articulation of the tibia, the fibula, and the dome of the talus. Range of motion occurs in one plane: plantar flexion and dorsiflexion. Dorsiflexion is somewhat restricted due to the anterior widening of the talar

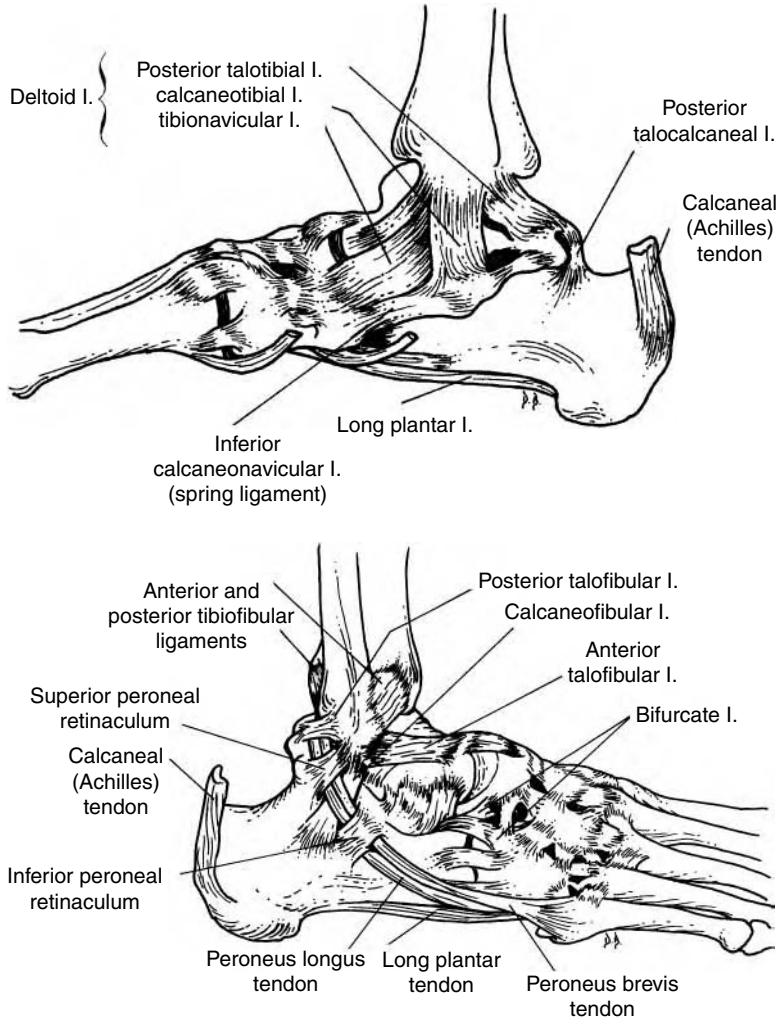

Figure 53.2 (Top) The medial ligamentous complex of the ankle; (bottom) the lateral ligamentous complex of the ankle.

dome. The subtalar joint of the foot allows for the full range of inversion, eversion, supination, and pronation. The two joints often work together as a universal-type joint, with modification in one affecting the biomechanics and normal activity of the other. The medial malleolus (distal tibia) and the longer lateral malleolus (distal fibula) provide a significant amount of bony stability to the ankle joint through their downward extension along the talar dome. Functional integrity of the hinge joint is thus sacrificed to structural stability.

Ligaments are the second important element in ankle stability. The larger and stronger deltoid ligament is fan shaped, arises from the medial malleolus, and inserts on the navicular, calcaneal, and talar bones. The medial ligament has both superficial (anterior talotibial, calcaneotibial, and tibionavicular) and deep (posterior talotibial) components (Figure 53.2A). The ligament, particularly the latter component, stabilizes the joint during eversion and prevents subluxation. Three distinct lateral collateral ligaments can be identified (Figure 53.2B): The *anterior talofibular ligament* arises from the anterior tip of the fibula and attaches to the lateral neck of the talus; its function is to prevent anterior and lateral subluxation of the talus during plantar flexion. Running from the fibular tip to the lateral aspect of the calcaneus in a posteroinferior direction is the *calcaneal fibular ligament*; this ligament functions to prevent lateral subluxation of the talus during strong adduction of the calcaneus. The *posterior talofibular ligament*

arises from the posterior aspect of the lateral malleolus and inserts on the posterolateral margin of the talus as the strongest of the three lateral collateral ligaments. It is responsible for preventing posterior subluxation of the talus during forced dorsiflexion.

The tibia and fibula are joined proximally by the interosseus membrane, whereas distally the two bones are held together by a strong syndesmosis consisting of the anterior and posterior inferior tibiofibular ligaments, the inferior transverse ligament, and the interosseus ligaments. The syndesmosis functions to maintain mortise stability, especially during dorsiflexion associated with weight bearing or the exertion of upward or outward pressure.

The synovial joint capsule also provides additional support, as it encapsulates the entire joint. It is recessed and lax in its anterior and posterior aspects to permit joint motion, although in some individuals it may be thickened anteriorly. The capsule is taut in the medial and lateral aspects, thus contributing to associated ligamentous support.

Finally, a variety of tendons support the ankle, but these are secondary stabilizers because they transit the joint and have no firm attachments. Their superficial nature also makes them vulnerable to injury. The extensors are located anteriorly, whereas the flexors are situated posteriorly. The inverters are located medially and the everters laterally (see Figure 53.2). The Achilles tendon is the largest in the body. It connects the triceps surae (medial and lateral gastrocnemius plus soleus muscles) to the os calcis and transmits the forces necessary to propel the body in walking, jumping, and running. These forces range from two to three times body weight in walking to four to six times body weight in running and jumping.

Additionally, a number of important soft-tissue structures are important for the functional integrity of the joint and the foot. The dorsal pedal artery lies on the anterior portion of the ankle and the foot between the extensor hallucis longus and the extensor digitorum longus tendons. In 10 to 15% of cases, it is congenitally absent. It is a secondary source of blood supply to the foot. The long saphenous vein can be palpated medially and just anteriorly to the medial malleolus. The posterior tibial artery, the main blood supply to the foot and anatomical constant, is located between the tendons of the flexor digitorum longus and the flexor hallucis longus muscles. It passes posterior and inferior to the medial malleolus and is not easily palpated unless the foot is non-weight-bearing and slightly plantarflexed. Immediately posterior and lateral to the posterior tibial artery lies the tibial nerve. Although difficult to palpate as an isolated structure, the neurovascular bundle is joined to the tibia by a ligament that creates the tarsal tunnel. The tibial nerve is the main nerve to the sole of the foot. Posteriorly, the calcaneal bursa lies between the Achilles tendon and the overlying skin. The retrocalcaneal bursa is located between the posterosuperior angle of the calcaneus and the anterior surface of the Achilles tendon. The deep peroneal nerve cannot be palpated. Sensation to the ankle is provided by L4 medially, L5 anteriorly, and the S1 dermatome laterally. The following peripheral nerves cover approximately the same area: medially, the long saphenous; anteriorly, the peroneals (deep supplies the first dorsal web space); and laterally, the sural nerve. Finally, lymphatic channels accompany the corresponding vascular bundles. A minimum amount of adipose tissue is found in and about the ankle, and a thin amount of elastic skin overlies all of these structures.

GENERAL MECHANISMS OF INJURY

Ankle mortise asymmetry creates inherent instability during inversion. The longer lateral malleolus provides a mechanical barrier to eversion ligamentous injury due to its greater surface contact with the talus. In addition, the dome of the talus is appreciably wider anteriorly than posteriorly. During inversion and plantar flexion, the narrow posterior aspect of the talus occupies proportionately less space within the mortise. As a result, ankle joint play is increased, which, together with the inherent block to eversion, results in predominantly lateral stress forces. Finally, the lateral ligaments of the ankle are smaller and weaker (20 to 50%) than the medial deltoid ligament. Additional complicating factors include tight heel cords and deficient proprioception. Many athletes, particularly females, have tight heel cords that force heel inversion. Regular walking on smooth, flat surfaces leads to a proprioceptive deficiency, which is aggravated on irregular, rough playing surfaces. Irregular surfaces, particularly those with holes, can produce obvious damage to the ankle. Less obvious circumstances, such as banked tracks, can lead to repetitive ankle trauma and long-term ankle disability. Defective, old, or inappropriately fitted footwear can result in inversion or eversion stress. All of these factors, when combined with a three- to fivefold impact force placed on the joint during heel strike, put the ankle at risk for injury.

CLINICAL EVALUATION

History

The following items should be carefully investigated in all ankle injuries:

1. What was the position of the foot and direction of stress when the injury occurred (e.g., eversion, inversion, flexion, extension, or a combination)?
2. Was disability immediate or did symptoms occur at a later time? Was play continued?
3. At the time of injury, were any snaps, pops, or crunches noted?
4. What were the surface conditions at the time of injury?
5. When and to what degree were pain, swelling, and discoloration noted?
6. Were any preexisting problems associated with the joint (i.e., previous injury or systemic disease)?
7. Was medical care sought out? What did the evaluation show? Was treatment initiated, and if so, what were the results?
8. Did the injury occur acutely or from overuse?
9. What is the functional capacity of the joint at present?

Examination

A careful exam, particularly during the "golden period," will usually identify the site of pathology and severity of tissue trauma. In some situations, however, a misdiagnosis can result. The clinical examination begins with an inspection of the contour and alignment of the joint, particularly noting any swelling, abrasions, lacerations, or discoloration. In general, swelling is an unreliable sign as edema is mostly a function of how much time has gone by after the trauma and what has occurred in the interim. The patient should be asked to demonstrate the overall functional capacity, strength, range of motion (20° dorsiflexion, 30° plantarflexion, 10° abduction/adduction, 17° internal/external rotation) and agility of the joint (i.e., while sitting, standing, walking, running, and at rest). The ankle should be carefully examined for painful trigger points, crepitus, temperature, passive range of motion, and neurovascular status (e.g., sensation, strength, reflexes, and pulses). Begin palpation farthest from the suspected site of injury, as 30 to 45% of patients will complain of tenderness in uninjured adjoining ligaments. The abnormal ankle should be compared with the normal ankle throughout the examination and should include the foot and footwear, leg, knee, hip, and lower back.

Once the patient's confidence has been gained, the joint should be stressed.[2] The anterior drawer test is performed with the patient lying on the examination table with the ankle at 90° and consists of drawing the calcaneus and talus anteriorly while stabilizing the tibia (Figure 53.3). The Gungor test is useful if the patient cannot relax due to pain. While the patient is lying prone, the heel of the extended ankle is pressed steadily downward.[3] Capsule rupture leads to forward talar displacement and skin retraction on both sides of the Achilles tendon when compared to the normal side. Sliding of the talus anteriorly by more than 4 mm between ankles during either test is abnormal. A soft endpoint or the perception of a "clunk" may be appreciated and is considered a positive anterior draw sign.

The talar tilt test is performed by stressing the ankle laterally (inversion) and medially (eversion) while stabilizing the patient's leg (Figure 53.4). A tear of the deep deltoid ligament will produce a palpable gap on the medial aspect of the ankle mortise. Gapping or rocking of the ankle mortise on the lateral side during lateral inversion stress indicates tears of both the anterior talofibular and the calcaneofibular ligaments. The normal angle of inversion talar tilt is up to 15°, although some individuals with ligamentous laxity may tilt up to 20° without a tear. A difference of 10° between ankles is highly suggestive of a tear, and 15° is diagnostic. The fibular compression or squeeze test assesses the integrity of the syndesmotic ligaments. Distal ankle pain when the proximal tibia and fibula are squeezed suggests a syndesmotic sprain. Rotation of the foot while the tibia is stabilized produces similar pain and a sensation of talar motion if a diastasis is present. Stress tests can be facilitated by a joint block. If necessary, 5 to 10 mL of 1% lidocaine are infiltrated into the joint opposite the side of injury and around the injured ligaments.

Routine x-rays include anterior, lateral, mortise (an anteroposterior view with 10 to 15° internal/external rotation). Oblique (45° of internal/external rotation) films are excellent for visualizing chip fractures and articular surfaces. It is occasionally advisable for the purpose of diagnostic accuracy, particularly in children, that radiographs be taken of the normal side for comparison. While 15% of ankle sprain adults have fractures, high yield criteria have been identified

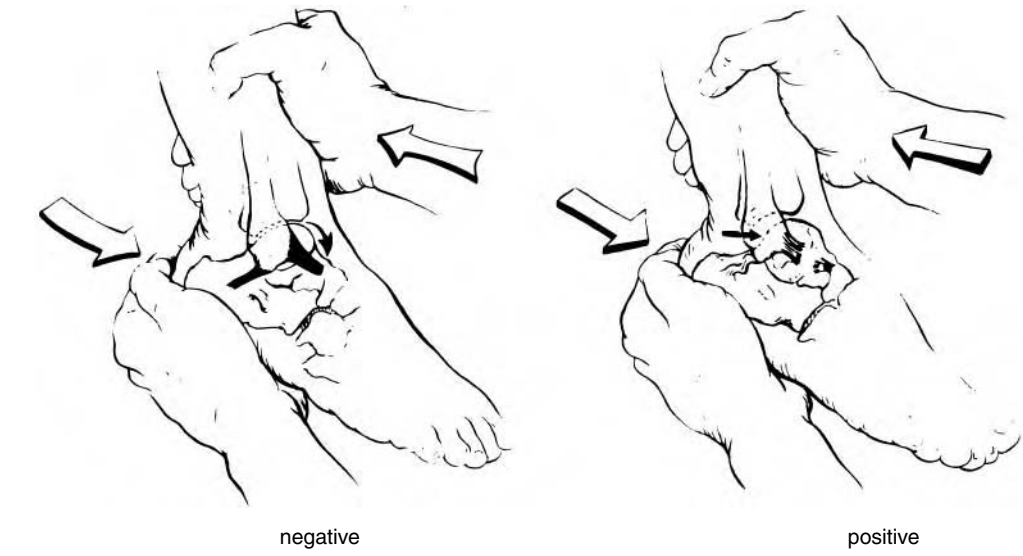

Figure 53.3 The anterior drawer test.

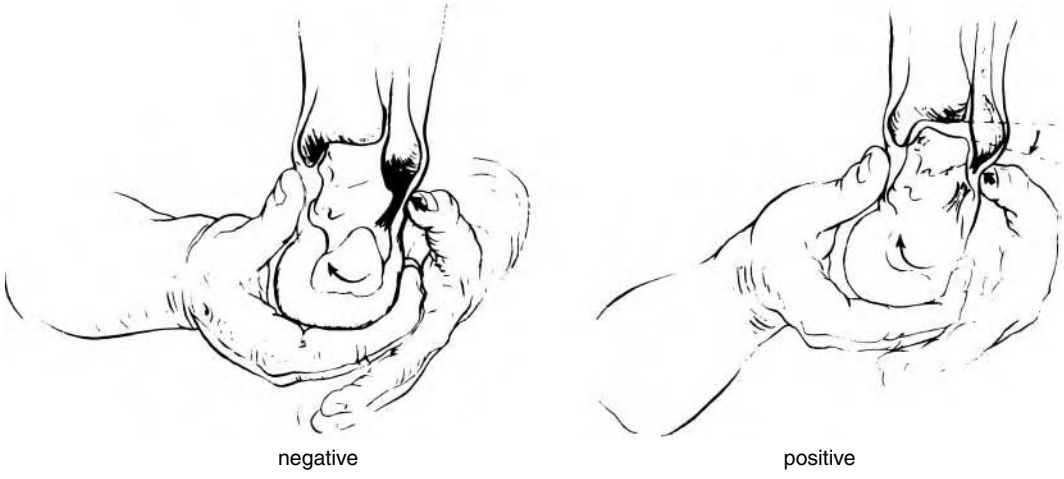

Figure 53.4 The talar tilt test.

(Table 53.1) (Level of Evidence C, consensus/expert opinion).[4]

Soft tissue should be examined for swelling and foreign bodies, joint spaces for widening or narrowing, and bones for dislocation or fractures (avulsion or osteochondral). Syndesmotic injury is suggested by widening of the mortise (>4 mm) and the distal tibia–fibula clear space (>6 mm) and reduced overlap (<1 mm) of the tibia and fibula. Passive stress-test radiographs performed after the acute period are indicated when clinical findings are inconclusive or the lesion is chronic

TABLE 53.1
High Yield Criteria for Ankle Radiographs (Ottawa Ankle Rules)[4]

Pain in malleolar zone (distal 6 cm of tibia and fibula and talus) and any of the following:
Bone tenderness at the posterior edge or tip of lateral malleolus
Bone tenderness at the posterior edge or tip of medial malleolus
Inability to bear weight immediately and in the emergency department for 4 steps

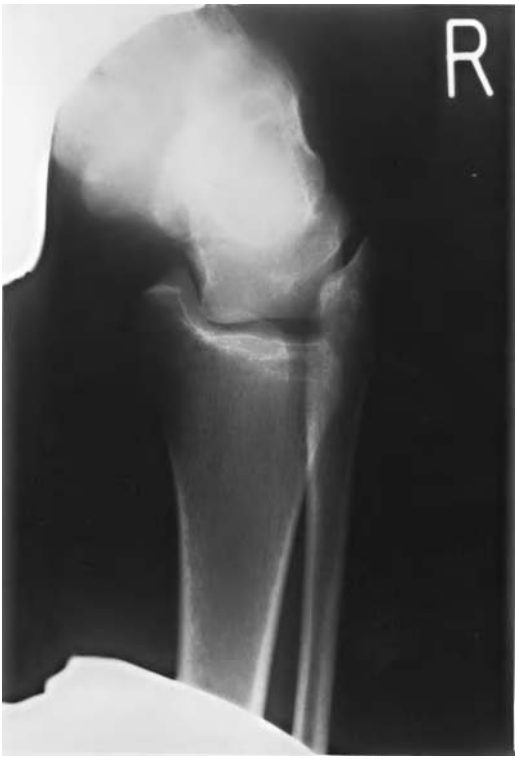

Figure 53.5 Normal stress film of the lateral right ankle.

(Figure 53.5). Stress films are easy to perform and may be difficult to interpret unless a standardized stress device is used. They correlate well with both arthrographic and operative findings.[5] If the stress films are positive, further evaluation is unnecessary. If stress films are inconclusive and there is a strong clinical suspicion of a complete tear, an arthrogram may be ordered. Dye appearing outside the joint capsule, particularly the anterolateral malleolus, or the peroneal tendon sheath is abnormal. False positive results occur in 10 to 25% of cases. The incidence of false negative examinations rises significantly due to post-injury healing. Also, the test is expensive, requires scheduling, and is associated with allergic reactions. Arthroscopy can also detect tears, defects, and debris. Isotopic bone scans are reserved for potential stress fractures.

Real-time ultrasound can be beneficial and relatively inexpensive in cases of tenosynovitis/tendinosis, and partial and complete tears. Computed tomography (CT) and magnetic resonance imaging (MRI) have been employed in diagnostic workups due to their high-resolution, multiplanar, thin-slice capabilities.[4] CT is excellent for tenosynovitis, complete and some partial ruptures, and associated fractures, particularly osteochondral lesions (OCLs), whereas MRI is useful in identifying soft-tissue, intraosseous, and neurovascular anatomy, particularly tenosynovitis, tendinosis, partial and complete tendon rupture, bone contusions, and large associated fractures. CT does not pick up bone contusions and the changes of tendinosis and other intratendinous abnormalities. Plain tomography is an inexpensive alternative to CT, although it is less sensitive. It is useful with the following criteria: ecchymoses or edema that covers two or more major stabilizing ligaments along with radiographic evidence of an effusion that distends the capsule by more than 5 mm; 35% of such patients have occult fractures.

While most ankle injuries are uncomplicated and can be treated in the office setting the diligent sports physician should know when to refer an injury to an orthopedic surgeon. Depending on the patient's progress and the physician's experience and clinical judgment, only difficult fractures (bi- and trimalleolar fractures), rare tendon injuries (peroneal or posterior tibialis), continuing pain 5 to 7 days after injury, failure to recover as expected, or general discomfort treating an injury should prompt a referral.

LATERAL COMPARTMENT LIGAMENT SPRAINS

I. Epidemiology[1,6]

This ligament is the most frequently injured structure in sports (25% of all sports injuries) where running and jumping are important, particularly basketball (70 to 80% of players have had one sprain, 60 to 80% have had multiple sprains, 50% have had residual symptoms, 15% have compromised performance),[7] volleyball, football, soccer, field hockey, and racquetball. The incidence rate is 1.6 (male) and 2.2 (female) significant sprains per 1000 person-days of sports; 85% of ankle injuries are sprains; 85% involve the lateral compartment; 85% involve the anterior talofibular ligament; 25% involve the anterior talofibular and calcaneofibular ligaments; 1% involve the posterior talofibular ligament; 30 to 40% have a history of previous injury; and 25% result in recurrent instability.[8,9] An estimated 50 to 60% do not seek professional treatment. Risk factors include tibial varum and calcaneal eversion in females and increased talar tilt in males.[8] For both genders, a history of ankle injury increases the risk of repeat trauma 5-fold; shoes with air cells in the heel, 4.3-fold; and lack of stretching, 2.2-fold.[9]

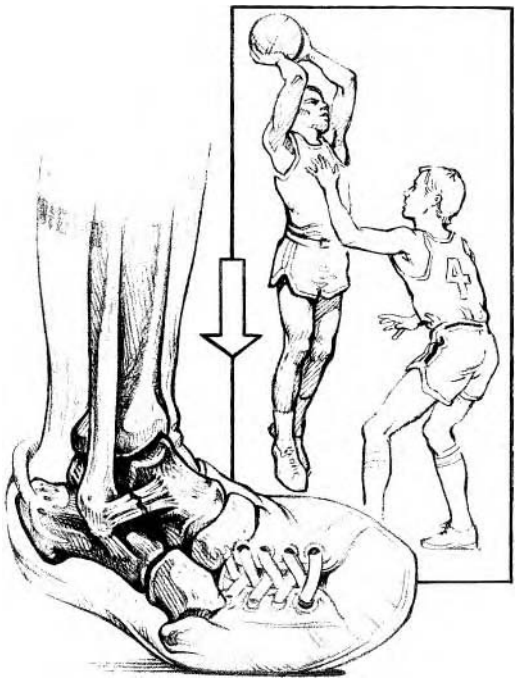

Figure 53.6 Spraining of the anterior talofibular ligament following forced plantar flexion and inversion.

II. Mechanism of Injury

Inversion, plantar flexion, and adduction (supination) especially during landing (Figure 53.6).

III. Anatomy

Sequential tearing of anterior talofibular (ATF), calcaneofibular (CF), and posterior talofibular (PTF) ligaments (see Figure 53.2B); grade 1 (<30% of fibers torn), 2 (30–70% of fibers torn), or 3 (>70% of fibers torn).

IV. Symptoms

- Grade 1 — minimal pain and disability; weight bearing not impaired
- Grade 2 — moderate pain and disability; weight bearing difficult
- Grade 3 — severe swelling, pain, discoloration; no weight bearing possible; significant functional loss

V. Signs[6]

- Grade 1 — slight tenderness and swelling over ligament with no laxity; anterior drawer test and talar tilt negative (see Figures 53.3 and 53.4).
- Grade 2 — moderate tenderness and edema; hemarthrosis (40 to 50%), ecchymosis (30 to 40%), positive talar tilt (5 to 10° difference), positive anterior drawer (4 to 14 mm).
- Grade 3 — pronounced edema and loss of function, typically with severe pain but may be painless; hemarthrosis (80 to 90%), ecchymosis (60 to 70%), positive talar tilt (>10° difference), anterior drawer (>15 mm).

VI. X-Rays

(See Table 53.1.) Anteroposterior, lateral, and mortise views may show localized tissue swelling, lateral clear space >2 mm.

VII. Special Studies

Interpretation of stress films (see Figures 53.7 and 53.8) may be difficult, and a mechanical stress device is recommended.

- Grade 1 — negative
- Grade 2 — 5 to 10° tilt difference; 3- to 14-mm anterior displacement of talus.
- Grade 3 — >20° tilt difference; >15-mm anterior displacement of talus.

An arthrogram has the advantages of being able to identify calcaneofibular tears and extravasation of dye with calcaneofibular tears. MRI provides direct visualization of the ATF and CF ligaments in acute and chronic injuries; 27% show bone contusions.[10]

VIII. Diagnosis

Lateral compartment sprain grade 1, 2, or 3.

IX. Differential Diagnosis

Posterior compartment osteochondral fracture, peroneal tendon subluxation/dislocation, peroneal quartus strain, physeal injury of the distal fibula in skeletally immature patients, interosseus membrane tear, tibiofibular ligament sprain, Achilles tendon rupture, subtalar and Lisfranc joint sprain, avulsion fracture of fibula or break of os calcis.

X. Treatment[11–15]

A. Initially: Treat with RICE, posterior plaster or air splint, no weight bearing; NSAIDs and analgesics as needed. Homeopathic ointment for 10 days has been found to significantly improve pain control vs. placebo.

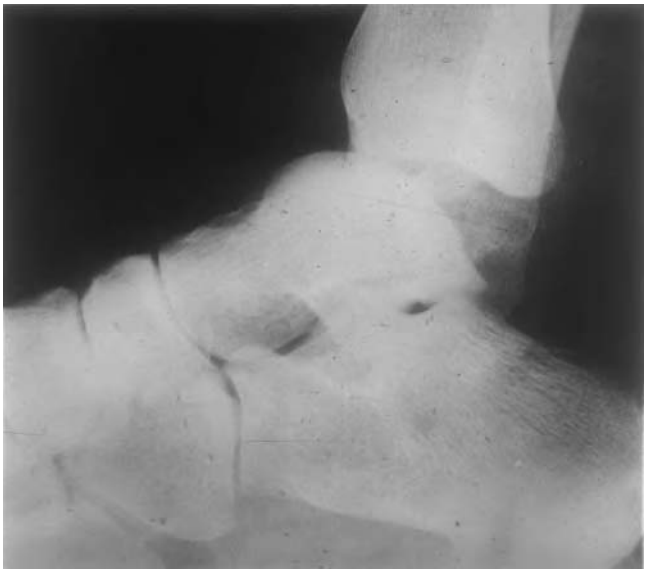

Figure 53.7 Stress film of the ankle.

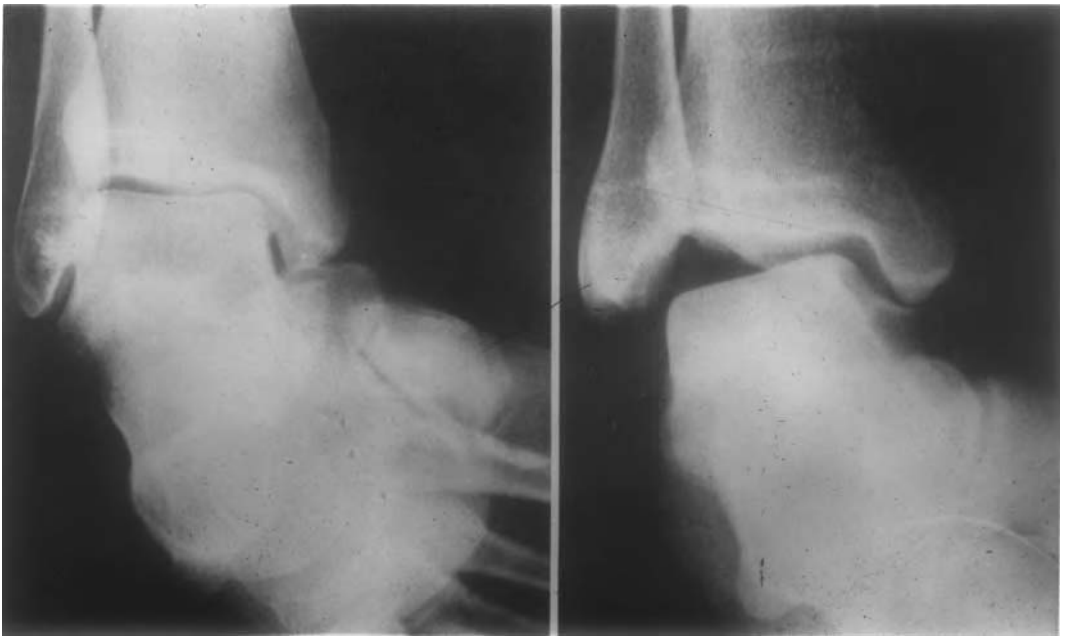

Figure 53.8 Stress film of the ankle.

B. Long-term: Functional treatment appears to be the favorable strategy when compared with immobilization. Limited evidence suggests improved outcome with functional treatment compared to minimal treatment (Level of Evidence A, randomized controlled trial; Level of Evidence B, other evidence).[13] No good evidence has been found to suggest that surgery is superior to immobilization, and the evidence comparing surgical vs. functional treatment is conflicting (Level of Evidence A, randomized controlled trial; Level of

Evidence B, other evidence).[14] Associated bone contusions have little clinical significance.[10]

- Grade 1 — Weight-bearing brace or strapping for 2 to 3 weeks.
- Grade 2 — Walking, well-padded dorsiflexion cast, Unna® boot weight-bearing brace or air-stirrup for 2 to 4 weeks, followed by strapping at 90° for 2 to 4 weeks. Avoid casting severely swollen limbs.
- Grade 3 — Dorsiflexion cast or weight-bearing brace for 3 to 6 weeks, followed by orthotic or strapping for 3 to 6 weeks; surgical repair. Best approach is unresolved, although age <40 and athletic competition favor surgical repair.

Rehabilitation should include isometrics while the cast is on, then range of motion (ROM), progressive resistance exercise (PRE), and proprioceptive exercises (balance/wobble board), plus functional activity:

- Grade 1 — 2 weeks
- Grade 2 — 4 weeks
- Grade 3 — 6 weeks

Injection therapy can be used in a stable ankle that is chronically inflamed, preventing progression in a rehabilitative program. Ultrasound and cold packs have been found to offer no significant benefit over placebo, and the benefit for diathermy over sham therapy is conflicting.

Consultation: grade 3 and recurrent injuries

XI. Complications
Recurrent sprains (10 to 30%), osteochondral fracture (6–7%) instability (10%), peroneal and/or tibial nerve injury, peroneal tendon subluxation/dislocation.

XII. Prevention[16,17]
Peroneal muscle conditioning program; teaching players to land with a relatively wide-based stance; support with tape or an orthosis (semirigid or air-cast) for previously injured ankles, as well as uninjured ones, particularly in high-risk sporting activities such as basketball or soccer (Level of Evidence A, randomized controlled trial; Level of Evidence B, other evidence),[16] with an ankle brace/stabilizer perhaps being superior to taping; use of an outer heel wedge; coordination training on balance board; wearing high-top, flexible shoes with cushioned midsoles and adequate toe-box.

It is best to err on the conservative side for diagnosis and on the liberal side for treatment and rehabilitation of a sprain.

MEDIAL COMPARTMENT LIGAMENT SPRAINS

I. Epidemiology
Of all ankle sprains, 5 to 10% involve the medial compartment; usually this is a more serious injury than a lateral sprain.

II. Mechanism of Injury
Eversion, dorsiflexion, and abduction (pronation).

III. Anatomy
Sequential tearing of the superficial deltoid ligament (tibionavicular), anteromedial capsule, anterior deep deltoid component, anterior tibiofibular ligaments, interosseus membrane, and remaining superficial and deep components (see Figure 53.2A); grades 1, 2, and 3.

IV. Symptoms
See Lateral Compartment Ligament Sprains section, above.

V. Signs[6]
See Lateral Compartment Ligament Sprains section, above.

VI. X-Rays
See Lateral Compartment Ligament Sprains section, above.

VII. Special Studies
Stress films show increased talar tilt on eversion stress; arthrography shows extravasation of dye; MRI is positive.

VIII. Diagnosis
Medial compartment sprain grade 1, 2, or 3.

IX. Differential Diagnosis
Avulsion fracture of medial malleolus (15%), sprain of anterior tibiofibular ligament and interosseous membranes, osteochondral fracture, strain of the tibialis posterior, Tillaux fracture.

X. Treatment[6,11–15]

A. Initially: RICE, posterior splint, no weight bearing, NSAIDs, analgesics as needed.
B. Long-term: Grade 1 — weight-bearing cast for 2 to 3 weeks, followed by brace/strapping; grades 2 and 3 — weight-bearing cast for 5 to 6 weeks, followed by brace/strapping. Operative repair, as necessary. For rehabilitation, see discussion in Lateral Compartment Ligament Sprains section, above.

Consultation: see lateral ankle sprains

XI. Complications

Chronic ankle instability; recurrent sprains.

XII. Prevention[16,17]

Strengthening of posteromedial muscles; taping/strapping/bracing (an ankle brace/stabilizer may be superior to taping); inner heel wedge; balance board training.

SYNDESMOTIC INJURY

Syndesmotic sprains are not rare (10% of sprains), follow forced external rotation or hyperdorsiflexion and eversion of the joint with internal tibial rotation (collision sports), and are frequently incomplete.[15,18] The athlete may note difficulty running uphill, with an inability to push off, especially to the opposite side, and a preference to walk on the toes. The complaints are usually greater than the findings (e.g., less swelling compared to medial or lateral ankle injuries). Tenderness is located over the syndesmosis and anterior and posterior tibiofibiluar ligaments, especially with external foot rotation and dorsiflexion (Kleiger test). The squeeze test is positive. Standard films during the acute period show a >6-mm tibiofibular clear space and widening of the mortise and demonstrate avulsion fractures of the anterior or posterior tibial tubercle in 20 to 50% of cases. A fracture of the anterolateral distal tibial epiphysis may be noted in the adolescent population. Films taken 3 to 5 weeks after the injury may demonstrate calcification of the syndesmosis or adjacent deltoid ligament. Grade 1 injuries are managed with an aggressive functional rehabilitation program. Cast immobilization with progressive weight bearing over 3 to 6 weeks followed by a vigorous rehabilitation program is appropriate for grade 2 injuries. The prognosis for these injuries is usually excellent with recurrence and residual instability uncommon. Grade 3 injuries are managed with open reduction and internal fixation with a syndesmotic screw. Recovery is prolonged.

PERONEAL TENDON DISLOCATION/SUBLUXATION

I. Epidemiology[19]

This syndrome is common in sports with significant dynamic forces applied to the foot, as in cutting, turning, or falling forward (e.g., wrestling, football, skiing, ice skating, basketball, soccer).

II. Mechanism of Injury[19]

Sudden forceful plantarflexion of everted foot or inversion stress with violent reflex contraction of peroneals and plantar flexors; direct blow to posterior lateral malleolus.

III. Anatomy

Disruption of the peroneal retinaculum (see Figure 53.2B) from its periosteal attachment at the lateral malleolus; absent or flat peroneal groove; flat or convex distal fibula.

- Grade 1 — Retinaculum with periosteum is stripped off the lateral malleolus.
- Grade 2 — Distal 1 to 2 cm of dense fibrous lip is elevated along with retiunaculum.
- Grade 3 — Retinaculum avulses a thin fragment of bone along with the fibrous lip.

IV. Symptoms

Acutely: painful snap, swelling, discoloration posterior to lateral malleolus; *chronically:* unpleasant snapping or clicking sensation and instability on dorsiflexion.

V. Signs[6]

Acute: ecchymosis, edema, tenderness with crepitus over peroneal tendons and retromalleolar region (posterior fibular area), palpation of highly mobile irregular bone fragment; *dislocation:* relocation click and crepitus chronically by ankle rotation, particularly during resisted active dorsiflexion, pronation, and eversion with the patient prone and the knee flexed 90° or by anterior/posterior-directed motion.

VI. X-Rays

X-rays are normally negative but may reveal associated distal avulsion fracture of fibula (15 to 50% of cases).

VII. Special Studies
Magnetic resonance imaging/CT scans specific to peroneals are positive but rarely required and should be used for confirmation or doubtful cases. Ultrasound is positive but rarely necessary.

VIII. Diagnosis
Peroneal tendon dislocation/subluxation.

IX. Differential Diagnosis
Sprain of lateral compartment ligaments; peroneal tenosynovitis, which occurs as it passes around the cuboid (e.g., in modern dancers) or retrofibular sulcus; rupture of peroneal tendon (rare), which is associated with compartment syndrome; contusion.

X. Treatment[19]
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; posterior splint; no weight bearing.
B. Long-term: Corticosteroid injection is not recommended; a well-molded, weight-bearing cast in slight plantar flexion and pronation should be worn for 4 to 8 weeks; a dynamic ankle brace or strapping plus horseshoe- or J-shaped pad can be used. Referral is recommended for possible surgical repair of retinaculum in acute unstable or chronic injuries (Jones or Duvries methods). Rehabilitation should include ROM, progressive resistance, and proprioceptive exercises (2 months).

XI. Complications
Recurrent dislocation.

XII. Prevention
Modification of cutting and turning techniques.

ACHILLES TENDINITIS
I. Epidemiology[20,21]
Achilles tendinitis constitutes 9 to 18% of running injuries (from hills, uneven surfaces, increased mileage or intensity of training, or recommencement of training after prolonged inactivity); also seen in jumping, skiing, basketball, gymnastics, dancing, cycling. Patient has a background of tight heel cord, congenitally small or thin tendon, tibia vara, or pronated or cavus foot or a history of injected or oral steroids.

II. Mechanism of Injury[21]
Repetitive overloads, particularly with faulty technique; use of rigid-soled shoes; landing hard upon the heels; or hyperpronation in association with weakness and inflexibility of the gastrocsoleus complex.

III. Anatomy
Microscopic tears of collagen fibers on the surface or in the substance of the tendon; pseudosheath thickening at its relatively avascular isthmus (3 to 4 cm above insertion into os calcis), and, chronically, nodule and adhesion formation (see Figure 53.2).

IV. Symptoms
Pain on walking that gradually worsens with the degree of injury.

V. Signs[6]
Tenderness; thickening, erythema, crepitus, and edema of the tendon; dorsiflexion weakness secondary to pain; nodules.

VI. X-Rays
None indicated.

VII. Special Studies
Not indicated, but MRI shows edema.

VIII. Diagnosis
Achilles tendinitis/tendinopathy.

IX. Differential Diagnosis
Calcaneal/retrocalcaneal bursitis, tendon rupture, calcaneal contusion or stress fracture, gastrocnemius strain, arthropathies.

X. Treatment[20]
A. Initially: Treat with RICE, NSAIDs, and analgesics as tolerated; steroid injection is contraindicated.
B. Long-term: Recommend ROM and PREs before and after activities (1 to 3 weeks) with full eccentric strengthening of the gastroc–soleus complex; heel lifts and cross-training should be prescribed but no evidence supports any one intervention (Level of Evidence A, randomized controlled trial).[22] Surgical tenolysis and debridement can be performed at 3 to 6 weeks; short leg cast.

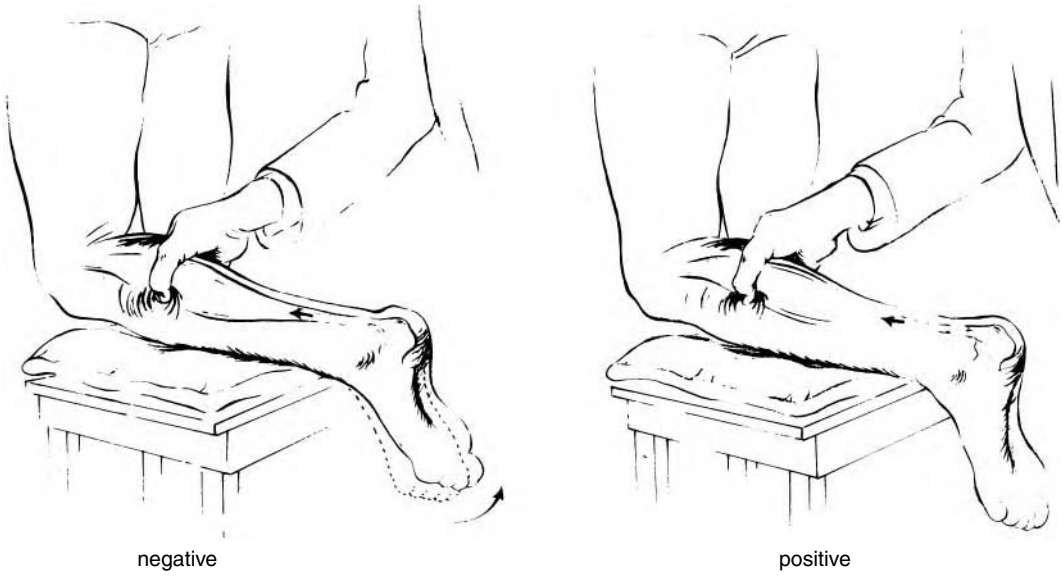

Figure 53.9 Thompson–Doherty squeeze test.

XI. Complications
Chronic tendinopathy, weakness, rupture.

XII. Prevention
Wear shoes with a soft-flared heel counter, adequate heel wedge (10 to 15 mm), and molded Achilles pad; do heel cord stretching exercises such as wall push-ups; avoid hill running, sudden increases in training intensity, or running on uneven/hard surfaces.

ACHILLES TENDON RUPTURE

I. Epidemiology[20,21]
Achilles tendon rupture occurs in poorly conditioned 30- to 45-year-old male patients, particularly weekend athletes. Incidence is 1 per 1000 athlete-years; background of tendinitis/degenerative changes; history of oral/injected steroids.

II. Mechanism of Injury[20]
Sudden dorsiflexion of a plantarflexed foot; sudden unexpected dorsiflexion of the ankle; pushing off the weight-bearing foot with the knee locked and extended; direct blow.

III. Anatomy
Rupture of the tendon 2 to 6 cm proximal to insertion, secondary to poor vascular supply; rarely, avulsion fracture of the calcaneus (see Figure 53.2).

IV. Symptoms
Sudden calf pain associated with audible snap, followed by antalgia and difficulty tiptoeing and climbing; pain may be inconsistent.

V. Signs[6]
Positive Thompson–Doherty squeeze test — with the patient prone or kneeling, a loss of plantar flexion is noted when mid-third of calf is squeezed (may be falsely negative due to sympathetic activity of peroneal and posterior tibial tendons in combination with lateral collateral ligaments; see Figure 53.9). Positive heel resistance test — easy dorsiflexion of the heel and foot against plantarflexion. Positive gap sign — palpable gap in tendon (may be absent if edema is significant).

VI. X-Rays
A loss of radiolucency or distortion of Kager's triangle may occur on lateral view; usually negative.

VII. Special Studies
Ultrasound, CT, and MRI are positive.

VIII. Diagnosis
Achilles tendon rupture.

IX. Differential Diagnosis
Acute strain of medial gastrocnemius, plantar's rupture, lateral collateral sprain, bursitis.

X. Treatment[20]
Varies with severity of injury, length of time between diagnosis and treatment, and age and caliber of athlete.

A. Nonoperative: Treat with RICE, NSAIDs, and analgesics; non-weight-bearing, long leg, gravity equinus cast for 6 weeks, followed by short leg equinus cast for 2 weeks, and then a short leg walker for 2 weeks. This regimen results in 75 to 85% normal function and is good for recreational athletes.
B. Operative: Surgery offers improved strength and endurance and decreased risk of rerupture (<5%) but significant complications (15 to 25%); it is the method of choice for serious professional athletes (75 to 90% function restored).
C. Rehabilitation: ROM plus PREs for 6 months); 2-cm heel orthotic for 3 months.

XI. Complications
Recurrent rupture (approximately 10%).

XII. Prevention
See Achilles Tendinitis section, above.

IMPINGEMENT SYNDROMES (ANTERIOR AND POSTERIOR)

I. Epidemiology
Impingement syndromes are a rare condition affecting high-arched athletes and involving loose lateral ligaments secondary to prior ankle sprain; caused by jumping (jumper's ankle), football, running, dancing, gymnastics, rugby (footballer's or English ankle).

II. Mechanism of Injury[21,23]
Anterior: hitting bottom in the plié (deep knee bend, with the "bravura" technique); explosive "drive off" from the planted foot. *Posterior:* full weight bearing in maximum plantarflexion (demi-pointe or full pointe).

III. Anatomy
Anterior: proliferative bony spur formation on the anterior marginal lip of the tibia and sulcus of the talus (talotibial exostoses). *Posterior:* os trigonum irritation (ununited lateral tubercle on posterior talus) present in 15% of population.

IV. Symptoms
Anterior: vague anterolateral pain with extreme dorsiflexion during running, cutting, or pushing off at full speed; weakness/loss of "drive." *Posterior:* posterolateral ankle pain on leaving the ground in a jump or during such dance maneuvers as the tendu, frappé, relevé.

V. Signs[6]
Anterior: point tenderness and edema over anterior ankle, decreased dorsiflexion that produces pain. *Posterior:* posterolateral tenderness and tenderness with forced passive plantarflexion; relief of pain following 1-cc 1% lidocaine injection into posterior capsule of ankle behind peroneal tendons.

VI. X-Rays
Anterior: spur in talar sulcus, normal joint line, and positive divot sign (localized wedge in the talar neck accepting the tibial osteophyte during dorsiflexion). *Posterior:* presence of an os trigonum on lateral view in full plantarflexion.

VII. Special Studies
Computed tomography/MRI are useful if diagnosis of posterior impingement is unclear; MRI shows bone contusions of the lateral talar tubercle and os trigonum.

VIII. Diagnosis
Impingement syndrome (anterior or posterior).

IX. Differential Diagnosis
Anterior: tibiotalar or talonavicular degenerative disease, osteoid osteoma of tarsal navicular. *Posterior:* marsupial meniscus, peroneal tendinitis, FHL /Achilles tendinitis, calcaneal apophysitis, retrocalcaneal bursitis.

X. Treatment[21,23]
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Modification of technique/activities, use of appropriate stretching, injection of a long- and short-acting corticosteroid after confirmation

with lidocaine. Surgical excision is recommended if conservative therapy fails.

XI. Complications
Recurrence.

XII. Prevention
Modification of dance and "drive-off" techniques.

POSTERIOR TIBIAL (PT) TENDINITIS

I. Epidemiology[20,21]
Posterior tibial tendinitis occurs in sports requiring a quick change of direction (tennis, dancing, soccer, basketball, ice hockey, football); it is more common with hypermobile flat feet and in individuals with navicular accessory.

II. Mechanism of Injury[20,21]
Repetitive microtrauma during the pronation phase of running/cutting/jumping, etc.

III. Anatomy
Inflammation and degeneration of the tendon/sheath posterior and inferior to the medial malleolus or adjacent to its partial insertion on the navicular tubercle.

IV. Symptoms
Diffuse swelling and pain at medial malleolus.

V. Signs[6]
Edema and tenderness behind medial malleolus or proximal to its insertion on navicular tubercle; pain worsened by inversion and plantar flexion against resistance.

VI. X-Rays
Not indicated.

VII. Special Studies
Not indicated, but ultrasound (less commonly) and MRI are positive.

VIII. Diagnosis
Posterior tibial tendinitis.

IX. Differential Diagnosis
Tendon strain; sprain of medial collateral; arthropathies, medial plantar neuropraxia; fracture of the sustentaculum tali; medial tibial stress syndrome (chronic shin splints).

X. Treatment[20,21]
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Non-weight-bearing short leg cast with foot in inversion is worn for 2 to 4 weeks; medial posted orthosis; surgical tenolysis.

XI. Complications
Recurrence, tendon rupture (very rare).

XII. Prevention
Regular use of orthotics, reduction of training program (e.g., distance, intensity).

POSTERIOR TIBIAL (PT) DISLOCATION

I. Epidemiology
Isolated dislocation is rare; common with severe ankle fractures.

II. Mechanism of Injury[20,21]
Inversion with dorsal flexion; may follow minor ankle trauma (e.g., stumbling over a fixed object, running on an uneven surface).

III. Anatomy
Tear of the flexor retinaculum with dislocation of the PT tendon from its groove to above the medial malleolus.

IV. Symptoms
Pain on the inside of the ankle that may be associated with a locking sensation.

V. Signs[6]
Increased pain with inversion; FROM ankle and foot; crepitus over groove to tibialis posterior tendon.

VI. X-Rays
Negative unless associated with medial fracture.

VII. Special Studies
Magnetic resonance imaging shows displacement of tendon; edema (superior to CT).

VIII. Diagnosis
Tibialis posterior tendon dislocation.

IX. Differential Diagnosis
Medial compartment sprain/fracture, tendon rupture or strain, tendinitis, arthropathies.

X. Treatment
Surgical repair of the flexor retinaculum followed by 5 to 6 weeks of casting, then rehabilitation.

XI. Complications
Recurrent dislocation (rare), rupture (rare).

XII. Prevention
None.

POSTERIOR TIBIAL (PT) RUPTURE

I. Epidemiology[21]
This type of rupture typically involves 40- to 60-year-old females, as well as marathon athletes who have had cortisone injections for tendinitis.

II. Mechanism of Injury[21]
Repetitive microtrauma in a setting of degenerative chronic tendinitis; traumatic blow (rare).

III. Anatomy
Transverse rupture of the PT tendon in its avascular zone just posterior to the medial malleolus or rupture at its insertion into the navicular and first cuneiform.

IV. Symptoms
Chronic pain and swelling can occur from the midposterior medial shaft of the tibia to the insertion of the tendon at the navicular; symptoms are worse in the pronated foot, with instability on ambulation.

V. Signs[6]
Erythematous swelling occurs slightly over or inferior to the medial malleolus; tenderness occurs 2 to 3 inches above the medial malleolus to the navicular and, in some cases, loss of foot supination to the inferior aspect of the first and second cuneiforms occurs; loss in height of medial longitudinal arch is noted; a palpable gap is found in the tendon with weakness during resisted inversion in complete tears; in complete tears, patient is unable to raise the heel more than 1 to 2 inches while standing on one foot; valgus position with compensatory adduction of the forefoot; more toes sign — abduction of the forepart of the foot at Chopart's joint.

VI. X-Rays
Comparison views show increase in the talocalcaneal angle (axial view), a talonavicular break (i.e., decreased longitudinal arch) sagittally (lateral view), and a decrease in the talonavicular articulation transversely in the injured foot (AP view).

VII. Special Studies
Magnetic resonance imaging is positive (superior to CT).

VIII. Diagnosis
Tibialis posterior tendon rupture (partial vs. complete).

IX. Differential Diagnosis
Tendinitis/strain of the tendon, medial compartment sprain.

X. Treatment[20]
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Nonoperative (incomplete lesions): Non-weight-bearing cast in slight inversion is worn for 6 to 8 weeks and a walking cast for 4 to 6 weeks, followed by shoe modification (medial sole and heel wedge and longitudinal arch support).
C. Operative: Surgical repair is reserved for complete tears and is followed by 6 to 8 weeks of wearing a non-weight-bearing cast in inversion and plantarflexion; a walking cast is worn for 4 to 6 weeks.
D. Rehabilitation: ROM and PREs; orthotic and strapping are utilized to decrease unwanted pronation during running, jumping, skiing.

XI. Complications
Recurrence.

XII. Prevention
None.

FLEXOR HALLUCIS LONGUS (FHL) TENDINITIS

I. Epidemiology[23]
This type of tendinitis is common in dancers (dancer's tendinitis).

II. Mechanism of Injury[23]
Overuse from repetitive push-offs, use of en pointe or demipointe positions.

III. Anatomy
Irritation and edema of the FHL tendon in the fibro-osseous tunnel behind the medial malleolus or between the sesamoids at the base of the first metatarsal (Henry's knot).

IV. Symptoms
Pain with toe flexion.

V. Signs[6]
Tenderness and swelling over the tendon behind the medial malleolus; crepitus, especially with active and passive motion of the hallux; decreased ROM, triggering, and clawing of the big toe (hallux saltans); decreased first toe flexion at interphalangeal joint (IPJ).

VI. X-Rays
Negative.

VII. Special Studies
Not indicated, but MRI may be positive for edema.

VIII. Diagnosis
Flexor hallucis longus tendinitis.

IX. Differential Diagnosis
Achilles or PT tendinitis, fibrous subtalar coalition, FHL rupture, arthropathies.

X. Treatment[21,23]
A. Initially: Treat with RICE, NSAIDs, and analgesics as needed.
B. Long-term: Surgical tenolysis is performed for severe cases of chronic recurrent tenosynovitis; steroid injections are not recommended.

XI. Complications
Recurrence.

XII. Prevention
Stretching, inflexible shoe.

MISCELLANEOUS TENDON INJURIES[20,21]
Factors that contribute to tendon injury include poor sports technique, increased patient's age, improper conditioning, malalignment, and excessive intensity or duration of activity. MRI, CT, and ultrasound are reliable diagnostic modalities. Trauma, training on hills, downhill hiking, direct shoe wear compression (ski boots), overuse, and rheumatologic disorders lead to tibialis anterior tendinitis. Its relatively straight course under the retinaculum causes minimal mechanical irritation. Modifications of training gear and program in conjunction with RICE, anti-inflammatory agents, and stretching are remedial for overuse injuries.

Ruptures of the anterior tibialis, long flexors, and peroneal and extensor tendons have been reported but are very rare.[21–24] Such injuries have been associated with multiple intratendinal steroid injections and age over 50 years. Rupture of the tibialis anterior causes a drop-foot gait and difficulty in walking on the heels. Treatment options include surgical repair (younger or competitive patients) or casting and orthotic devices with a 90% dorsiflexion brace for 6 to 8 weeks. A posterior leaf spring brace may aid ambulation, particularly in the older patient. Peroneal brevis tears have occurred without prior subluxation/dislocation. Rigid extension or flexion of the toes (e.g., running, skiing, jumping) with a forceful isometric contraction may cause avulsion or rupture of the long extensor and flexor tendons. Treatment is surgical repair.

FRACTURES
I. Epidemiology[18,25]
Fractures are seen most commonly in high-velocity, high-impact sports (e.g., football, soccer, skiing, hockey, automobile racing); stress fractures occur in running, gymnastics, and dancing; Salter type I and II fractures of the fibula are the most common ankle injuries in children.

II. Mechanism of Injury

- Type A — lateral displacement of the talus:
  Eversion force + supinated ankle (50% of all fractures)
  Eversion force + pronated ankle
  Abduction force + pronated ankle
- Type B — medial displacement of the talus:
  Adduction force + supinated ankle
  Adduction force + dorsiflexed/plantarflexed ankle
- Type C — axial compression of the talus
- Type D — repetitive microtrauma

III. Anatomy
Sequential
See Figures 53.10 and 53.11.

- A1 — Rupture of anterior inferior tibiofibular ligament → spiral oblique fracture of

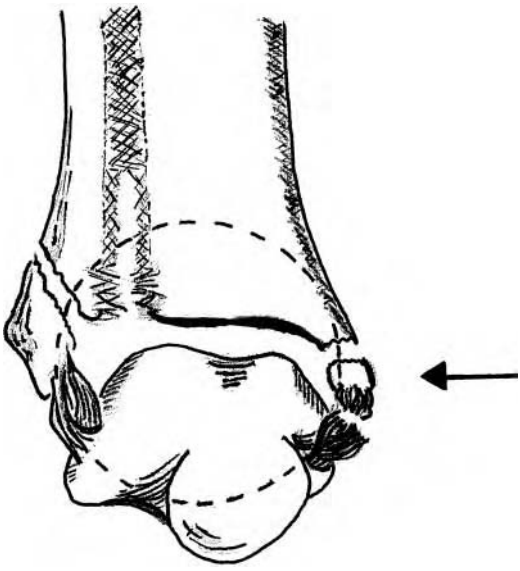

Figure 53.10 Type A fracture. Lateral displacement force (eversion) with rupture of the mortise ring: spiral/vertical fracture of fibula, rupture of the anterior inferior tibiofibular ligament, transverse fracture of medial malleolus, deltoid ligament rupture, and interosseus membrane tear.

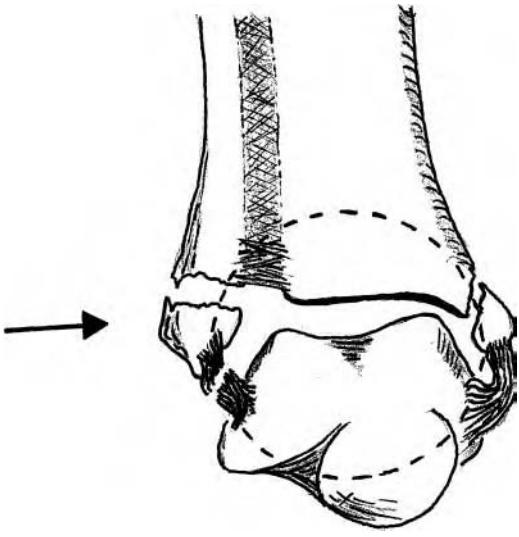

Figure 53.11 Type B fracture. Medial displacement force (inversion) with rupture of the mortise ring; vertical fracture of medial malleolus, transverse fracture of fibula, and rupture of the talofibular ligament.

fibula → posterior malleolar fracture → medial malleolar fracture or deltoid ligament rupture.

- A2 — Rupture of deltoid ligament or fracture of medial malleolus → rupture of anterior inferior tibiofibular ligament → tear of interosseus membrane → spiral fracture of fibula 2 to 3 inches above its top → rupture of posterior inferior tibiofibular ligament or avulsion fracture of posterior malleolus (in children, type II or VI).

Salter Fracture of Tibia

- A3 — Fracture of medial malleolus or rupture of deltoid ligament → rupture of anterior inferior ligament → oblique fracture of distal fibula → posterior malleolar fracture.
- B1 — Avulsion of the lateral malleolus or rupture of the talofibular ligament → vertical fracture of the medial malleolus; in children, type I or II Salter fracture of fibula → type III (Tillaux) or IV fracture of medial malleolus.
- B2 — Medial malleolar fracture or tibiofibular diastases; medial or lateral osteochondral fractures.
- C — Impaction fracture → marginal fractures → tibiofibular diastases.
- D — Medial osteochondral fractures or distal fibular stress fractures.

IV. Symptoms

Acute: Audible pop or crack, abrupt moderate to severe pain and swelling, immediate disability, discoloration. *Chronic:* History of "sprained" ankle resistant to all treatment, dull ache with slight swelling after excessive walking, increased pain with activity and complete relief with rest.

V. Signs[6]

Acute: Localized/point tenderness, edema, possible crepitus, ecchymosis; positive osteophony with certain fractures. *Chronic:* Usually negative except point tenderness on talar dome with ankle plantarflexed (osteochondral fracture) or on distal fibula (stress fracture).

VI. X-Rays

Comparison AP, lateral, oblique, and mortise views. Mortise and oblique views good for osteochondral fragments (OCL); routine films are negative for stress fractures for first 2 to 3 weeks following injury.

VII. Special Studies

Stress films may suggest associated ligamentous injury and joint instability. Arthrography shows leakage of dye. CT arthrography is useful in the evaluation of articular cartilage integrity (osteochondritis dissecans and osteochondral fracture) and in the diagnosis of intraarticular loose bodies. MRI is good for defining location of larger fragments and percentage of involved joint surface. Bone scan is good for stress fractures.

VIII. Diagnosis

Fracture:

1. A fibular spiral fracture 2 to 3 inches proximal to the mortise is associated with a rupture of the deltoid ligament and/or a medial malleolar fracture and a rupture of the anterior inferior tibiofibular ligament.
2. A distal fibular fracture at the joint line should suggest a deltoid ligament injury. Displacement of the lateral malleolus is often accompanied by a medial malleolar fracture and/or deltoid ligament rupture.
3. A vertical medial malleolar fracture is associated with either a rupture of the lateral ligament or a lateral malleolar fracture.
4. Transverse malleolar fractures are ligamentous avulsion injuries.
5. Vertical, comminuted, spiral malleolar fractures follow talar compression (check spine and calcaneus).
6. Displaced fractures are usually associated with ligament injuries.
7. Potentially serious ligamentous injury may be present despite negative x-ray findings.
8. Stable injuries consist of one break in the mortise ring; unstable injuries consist of two or more breaks. The breaks may be any combination of fractures and ligament ruptures (see Figure 53.11).
9. A nondisplaced physeal injury (Salter–Harris I or II) must be considered in any child with normal radiography, well-documented trauma, and ankle pain.
10. Chronic or subacute pain and ankle effusion in association with running activities suggest stress fracture of the malleoli.

IX. Differential Diagnosis

Sprains of either compartment, tenosynovitis, contusion.

X. Treatment[18,25]

A. Initially: Treat with RICE, NSAIDs, and analgesics as needed; posterior splint; no weight bearing.
B. Long-term:
   1. Stable fractures — Neutral-positioned walking cast for 4 to 6 weeks.
   2. Unstable fractures — Reduction (closed/open) followed by immobilization for 4 to 8 weeks. Hinge cast provides early ROM and minimizes harmful effects of immobilization. Outcome is directly related to the degree of anatomical reduction.
   3. Epiphyseal injuries — For type I and II fibular fractures, precise anatomic reduction by closed manipulation and 2 to 4 weeks in a short leg weight-bearing cast; additional 2 to 3 weeks if region still tender. Open reduction is rarely necessary. For type II tibial fracture, long leg cast for 6 weeks with precise anatomic reduction by closed manipulation. Open reduction is rarely necessary. For types III and IV tibial fracture, open reduction and internal fixation for displacements >2 mm. For type IV medial malleolar fracture, 3 to 4 weeks of mid-adduction/inversion casting. For a Tillaux fracture, 3 weeks in a long leg nonweight-bearing cast plus 3 weeks in a short leg walking cast following closed reduction for undisplaced fragments or open reduction plus internal fixation for displaced fractures. All growth-plate injuries should be followed up regularly for at least 2 years or to skeletal maturity.
   4. Osteochondral fragments — Medial, 12 to 24 weeks in nonweight-bearing cast; lateral, arthroscopic repair, pinning.
   5. Rehabilitation — ROM, progressive resistance, proprioceptive, and functional exercises.

Consultation: advised for unstable, epiphyseal, and osteochondral fractures; continuing pain 5 to 7 days after injury.

XI. Complications

Complications are seen mostly after unstable fractures and include traumatic arthritis (20 to 40%), particularly with lateral osteochondral fracture; persistent talar instability; Sudeck's atrophy; interosseous membrane ossification; epiphyseal injuries such as chronic epiphysiolysis, premature closure, deformity, and early arthritis. Prognosis for growth-arrest-induced deformities is worse for children under 10 years.

XII. Prevention

Regular use of properly fitted protective equipment, application of approved and supervised techniques, avoidance of overtraining.

SUMMARY

Sacrificing structure for function, the dynamic ankle is the most frequently injured area of the body in sports and recreational activities. The generic sprain is probably the most frequent sports injury and can be associated with a significant amount of morbidity. A solid understanding of pathomechanics and good clinical acumen are essential in distinguishing bony, ligamentous, and muscle–tendon pathology. The majority of low-velocity ankle injuries, including stable fractures, can be quickly identified and successfully managed by the knowledgeable primary care physician. High-velocity injuries invariably require surgical consultation due to their complexity and potential poor prognosis.

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54 FOOT INJURIES

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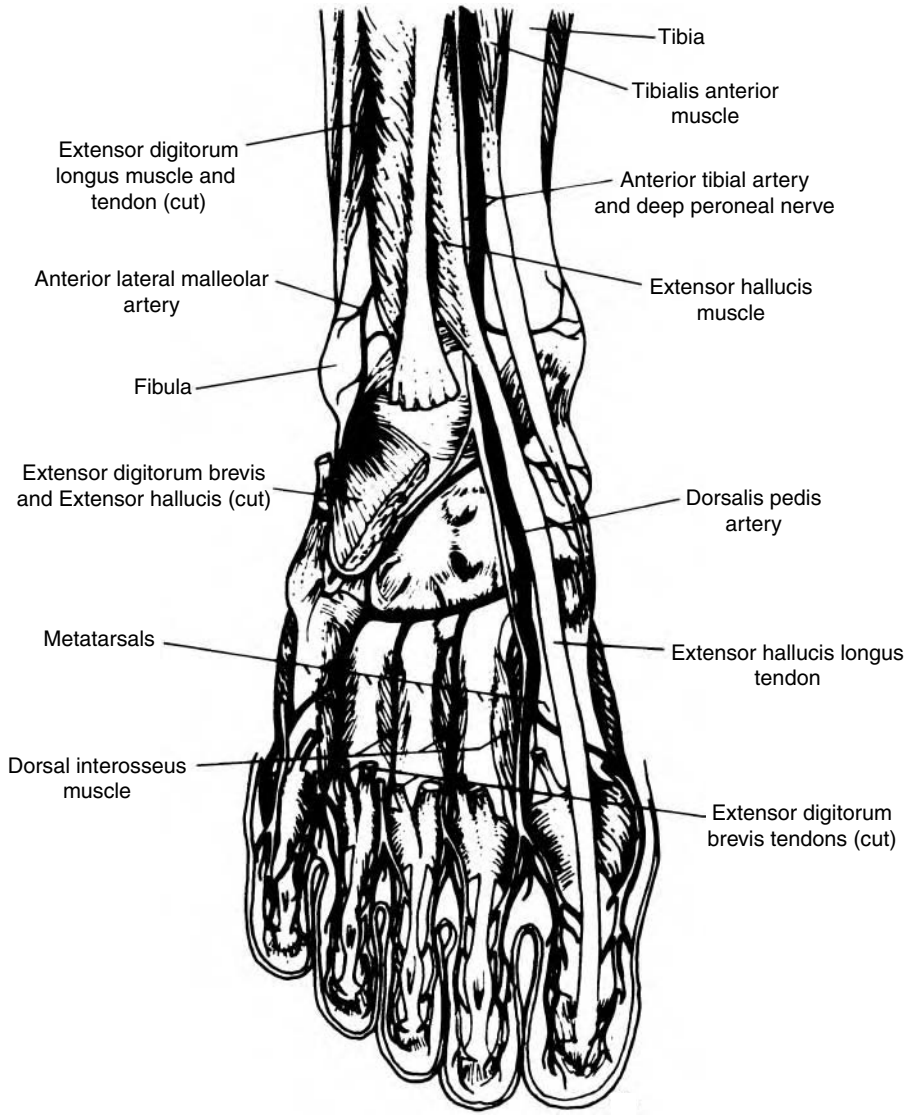

Figure 54.1A The anatomy of the instep. (Facing page) Important plantar anatomy.

INTRODUCTION

The foot is an amazing combination of bones — tarsals (7), metatarsals (15), phalanges (14), and sesamoids (2); ligaments; and muscles and their tendons (Figures 54.1A,B). It must be strong enough to support our body weight and propel us forward, yet flexible enough to absorb the impact of running or jumping. While performing these varied functions, the foot can easily be traumatized or subjected to overuse. An injury to the foot may be disabling; however, even when it is not, it predisposes other links in the ambulatory chain to significant injury.

The two main normal motions of the foot are pronation and supination. When pronating, the foot uses the longitudinal arch, which is supported by the plantar fascia, to help absorb contact forces. When supinating, the foot becomes more rigid, helping it to act as a stable lever to push off. Many foot problems are associated with excessive degrees of these two normal functions. Injuries can result from either abnormal stresses on a normal structure, such as increased running mileage, or normal stress placed onto an abnormal structure, such as a high arch.[1]

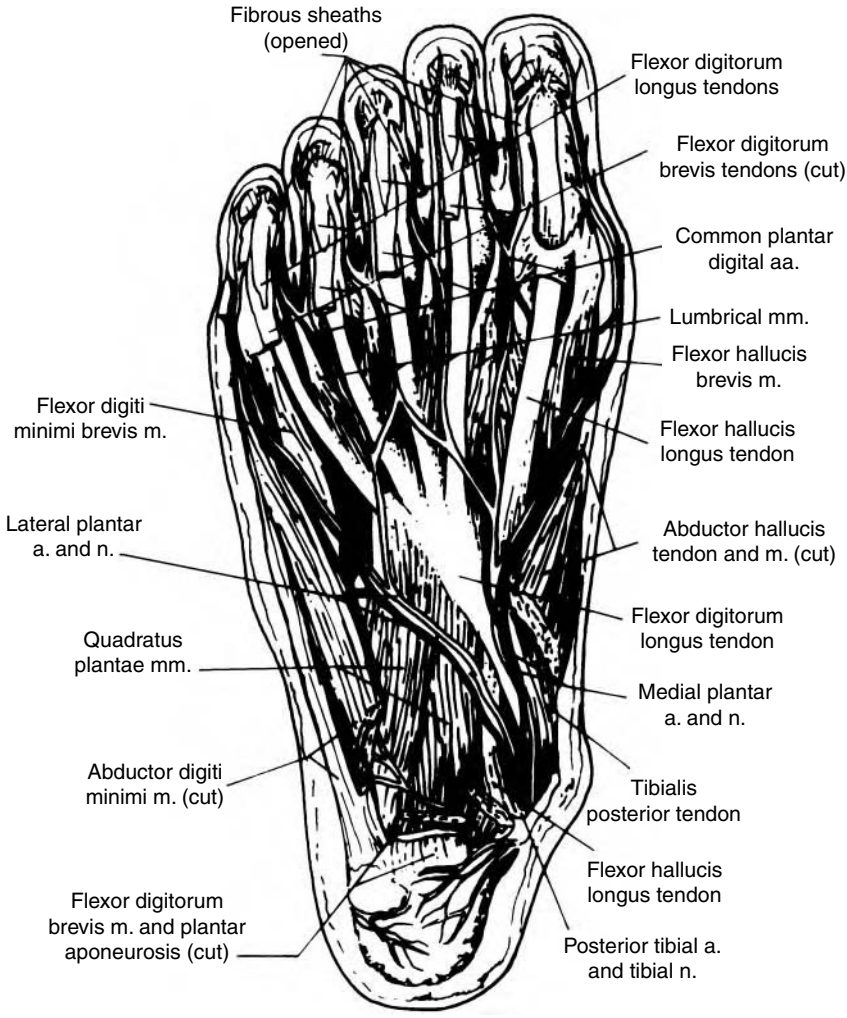

FIGURE 54.1B

Using gait analysis, the interplay between these motions becomes clear. When preparing for heel strike, the foot begins to supinate because the muscles that are responsible for ensuring that the foot descends in a slow, controlled fashion also invert the foot, causing the heel to be supinated. Thus, the bones of the foot are tightly bound together during heel strike. Moving into pronation (or rolling in), the bones of the foot become looser, allowing the arches to absorb some of the shock of contact which can be five times an individual's body weight while running. Next, at toe-off, the foot moves back into supination creating a rigid lever for propulsion.

APPROACH TO THE PATIENT

History

Important questions include inquiries about previous injury; whether the athlete has started playing a new sport, purchased new shoes, or suddenly increased mileage or rate of training; or whether relatively no change has occurred, thus implying constant overuse of the same portion of the foot over the course of many weeks, months, or years.

Examination

Look at the foot both statically and dynamically while comparing the involved with the uninvolved side. Have the athlete bring in his or her

shoes to observe for any abnormal wear patterns, again comparing both shoes. All shoes have some wear on the lateral portion of the heel, as this is the location of heel strike in most individuals. Callus formation at the bottom of the foot indicates stress patterns to which that the foot is subjected. It is often helpful to observe the patient's foot while standing, particularly from behind, to determine the height of the longitudinal arch, angulation of the Achilles tendon, and degree of pronation of the foot at rest. Observe the individual walking, jogging, and cutting. Skillful palpation, beginning in non-traumatized areas, should assess trigger points, range of motion, foot flexibility, and neurovascular status. Results should be compared with the normal foot. When indicated, X-rays are often invaluable but may not initially show any findings in the case of a stress fracture. Additionally, the practitioner must order the appropriate x-ray for the injury (e.g., weight bearing, on toes, navicular). A bone scan may be indicated (see Figure 54.4C). The use of hematologic analysis (e.g., complete blood count, erythrocyte sedimentation rate) is often not necessary in the acute injury setting.

Treatment

General guidelines include the RICE (rest, ice, compression, elevation) + progressive resistance exercise (PRE) regimen (see Chapter 27). The use of orthotics or better constructed athletic footwear to resist abnormal motion is a critical part of the treatment and prevention of injuries; however, the high-arched foot may require a more cushioned design as opposed to motion control. Sometimes, the use of taping, strapping, or casting may be necessary. Surgery should always be a last resort.

CONTUSION: STONE BRUISE

I. Epidemiology

All running sports, all ages, both sexes.

II. Mechanism of Injury

Contact with ground or stone without adequate padding.

III. Anatomy

Any weight-bearing bone in foot; most commonly calcaneus and first metatarsal head.

IV. Symptoms

Pain over involved area; patient usually can remember offending action.

V. Signs

Occasional warmth, redness over tender area.

VI. Radiology

X-rays are negative.

VII. Special Studies

Bone scans are negative.

VIII. Diagnosis

Stone bruise.

IX. Differential Diagnosis

Stress fracture.

X. Treatment/Rehabilitation

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Use orthotics such as felt donut; return to play is immediate after adequate padding is provided.

Consultation: not indicated

XI. Complications

None.

XII. Prevention

Shock-absorbent footwear, careful observation of terrain.

BURSITIS: RETROACHILLES AND RETROCALCANEAL BURSITIS

I. Epidemiology

Post-pubescent athletes (both sexes); common in running, basketball, and tennis.

II. Mechanism of Injury

Repetitive microtrauma from shoe.

III. Anatomy

Bursa between Achilles tendon and overlying skin (retroachilles); bursa between Achilles tendon and calcaneus (retrocalcaneal).

IV. Symptoms

Pain behind heel.

V. Signs

Must palpate for tenderness posterior to Achilles tendon (retroachilles), yet patient should not have bony tenderness or Achilles tendon tenderness;

may also have swelling. Retrocalcaneal bursitis has pain anterior to Achilles tendon.

VI. Radiology
Not indicated.

VII. Special Studies
None.

VIII. Diagnoses
Retroachilles bursitis, retrocalcaneal bursitis.

IX. Differential Diagnoses
Achilles tendinitis, calcaneal stress fracture.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, nonsteroidal anti-inflammatory drugs (NSAIDs), and analgesics, as needed.
B. Long-term: Use padded heel counter, injection therapy. Return to play is allowed after pain subsides.

Consultation: not indicated

XI. Complications
Chronic bursitis.

XII. Prevention
Adequate, padded heel counter.

STRAINS: PLANTAR FASCIITIS (HEEL SPUR SYNDROME)

I. Epidemiology
Running, aerobics (high-impact); found in both sexes but not as common in adolescents.[1]

II. Mechanism of Injury[2]
Excessive torsion and hyperpronation; poor shock dissipation with cavus foot.

III. Anatomy
Microtears (straining) and inflammation of the plantar fascia (broad, connective tissue that extends from bottom of calcaneus to the toes).

IV. Symptoms
Insidious onset of pain at the base and medial aspect of the heel is often disabling; often worse right after first waking up in the morning or after sitting. Pain may decrease after warm-up period.

V. Signs
Tenderness at plantar fascia insertion and medial aspect of the calcaneus; pain increases with passive dorsiflexion of great toe.

VI. Radiology
X-rays reveal heel spur (Figure 54.2) in 50% of individuals with calcaneodynia but still is not pathognomonic.

VII. Special Studies
Bone scan is negative unless periostitis or stress fracture is present.

VIII. Diagnosis
Plantar fasciitis (heel spur syndrome).

IX. Differential Diagnoses
Calcaneal stress fracture, nerve entrapment (depends on location of pain).

X. Treatment/Rehabilitation[2]
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Correct tendency to hyperpronate with soft orthotic (1/2-inch felt or closed foam rubber) or footwear designed to create a varus heel tilt. A central beveled cutout (3 to 5 cm) should be made in the tender area. Tension night splints can be used (mixed evidence).[2–4] With Cavus foot, provide more shock absorption and recommend adequate stretching. Appropriate shoes should be worn at all times and a heel cup used. Use low dye strapping for support. Injection therapy can be directed to trigger points. The rolling pin exercise can be used to increase fascia flexibility. Return to play is allowed when pain is gone.

Consultation: intractable pain, acute rupture of the fascia

XI. Complications
Recurrence, fascia rupture.

XII. Prevention[5]
Adequate stretching and strengthening exercises (e.g., intrinsic muscles and tibialis posterior muscle); wearing appropriate shoes at all times (e.g., adequate cushion, heel counter, and arch support).

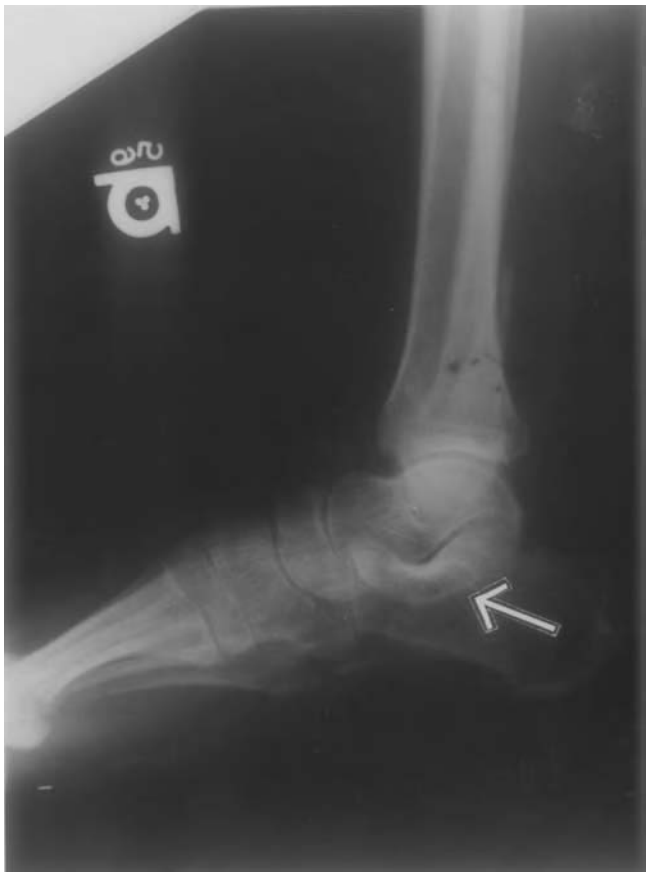

Figure 54.2 Heel spur (arrow); an incidental fibular fracture is also visible.

FOOT SPRAIN

I. Epidemiology
Running and aerobics (high-impact); found among post-pubescent athletes of both sexes.

II. Mechanism of Injury
Sudden forceful movement in excess of normal function; repetitive microtrauma based on abnormal structure (pes planus, or flat foot) or function (hyperpronation).

III. Anatomy
Tearing of the ligamentous attachments or other connective tissue support.

IV. Symptoms
Acute: pain, swelling over involved area; *chronic:* dull, achy sensation, often in forefoot.

V. Signs
Tenderness to palpation (anteromedial aspect suggests spring ligament), inability to walk on toes.

VI. Radiology
X-rays are negative unless an associated avulsion fracture is present.

VII. Special Studies
Bone scan is negative.

VIII. Diagnosis
Foot sprain, acute or chronic (overuse).

IX. Differential Diagnoses
Metatarsal stress fractures, cuboid dislocation, metatarsalgia.

X. Treatment/Rehabilitation[6]

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Use orthotics for tendency to hyperpronate, low dye strapping, physical therapy. Return to play is allowed when patient is pain free.

Consultation: not indicated

XI. Complications
Avulsion fracture.

XII. Prevention
Correct or alter both static and dynamic forces.

TENDINITIS

PERONEAL TENDINITIS

I. Epidemiology
Basketball, football; adolescent and older; both sexes.

II. Mechanism of Injury
Repetitive microtrauma.

III. Anatomy
Inflammation of the peroneal tendons as they pass posterior and inferior to the lateral malleolus.

IV. Symptoms
Pain and weakness, particularly of active eversion of foot.

V. Signs
Tenderness along course of tendon; possible warmth and crepitus.

VI. Radiology
Not indicated.

VII. Special Studies
None indicated.

VIII. Diagnosis
Peroneal tendinitis.

IX. Differential Diagnoses
Lateral ligament sprain, tendon dislocation/subluxation.

X. Treatment/Rehabilitation

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Use ROM, physical therapy, injection therapy. Return to play is allowed when patient is pain free.

Consultation: not indicated

XI. Complications
Rupture (rare).

XII. Prevention
Adequate ankle support through taping, strapping, or padding; strengthening of ligaments and muscles of the lateral ankle compartment.

TIBIALIS POSTERIOR TENDINITIS

See Chapter 53.

EXTENSOR COMMUNIS TENDINITIS

I. Epidemiology
Running, aerobics, skiing, swimming (e.g., dolphin, whip kick); found in post-pubescent athletes of both sexes.

II. Mechanism of Injury
Chronic irritation from footwear or technique.

III. Anatomy
Inflammation of tendons on the dorsum of the foot.

IV. Symptoms
Pain on top of foot; worse when attempting to dorsiflex toes.

V. Signs
Tenderness to palpation along dorsum of foot; warmth and crepitus may be found.

VI. Radiology
Not indicated.

VII. Special Studies
None indicated.

VIII. Diagnosis
Extensor communis tendinitis.

IX. Differential Diagnoses
Metatarsalgia or metatarsal stress fracture.

X. Treatment/Rehabilitation

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Padding of athletic shoe. Steroid injection is contraindicated. Return to play is allowed when patient is pain free.

Consultation: not indicated

XI. Complications

Chronic tendinitis.

XII. Prevention

Proper fitting footwear/padding where appropriate; stretching.

FLEXOR HALLUCIS LONGUS TENDINITIS

See Chapter 53.

ACHILLES TENDINITIS

See Chapter 53.

SUBLUXATION/DISLOCATION

PERONEAL CUBOID SYNDROME

I. Epidemiology[5–7]

Dancing, basketball, football, running (e.g., uneven terrain or early excessive training); found in post-pubescent athletes of both sexes. This syndrome accounts for 4% of all athletic foot injuries; 80% of patients have pronated feet.

II. Mechanism of Injury

Forceful contraction of the muscle, particularly during hyperpronation, brings cuboid plantarward and medially, causing "locking."

III. Anatomy

The peroneus longus muscle passes underneath the cuboid bone in the peroneal groove. During pronation, its greater mechanical advantage at its insertion to the first metatarsal and cuneiform joint allows cuboid subluxation.

IV. Symptoms

Pain with walking along lateral foot or over the fourth and/or fifth metatarsal on the dorsal aspect of the cuboid on the calcaneocuboid joint; foot cannot move through its range of motion.

V. Signs

Tenderness to palpation over cuboid bone, but pain may be felt along the course of the peroneus longus muscle (i.e., peroneal groove); foot hyperpronation.

VI. Radiology

Not helpful.

VII. Special Studies

None indicated.

VIII. Diagnosis

Peroneal cuboid syndrome (locked cuboid, calcaneal cuboid fault syndrome, lateral plantar neuritis).

IX. Differential Diagnoses

Occult fracture of cuboid or metatarsal base, calcaneonavicular coalition; peroneal tendinitis.

X. Treatment/Rehabilitation[6]

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Closed reduction — with athlete prone, by quick downward thrust apply direct pressure with the thumbs (one over the other) to the plantar aspect of the cuboid so that it moves dorsolaterally with respect to the calcaneus. The knee should be flexed adequately to avoid stretching of the superficial peroneal nerve. Pain relief should be immediate, although several sequential manipulations may be required for chronic injuries. A neutral orthotic, cuboid pad, or low dye strapping with medial countertraction should follow. Return to play is allowed when patient is pain free.

Consultation: Recurrence

XI. Complications

Arthritis and aseptic necrosis if not reduced properly.

XII. Prevention

Low dye strapping with medial countertraction, orthotic (neutral), graduated training on even soft surfaces.

DISLOCATED TOE

I. Epidemiology
Karate, wrestling, gymnastics; found in all ages of both sexes.

II. Mechanism of Injury
Direct trauma to the toe (e.g., karate kick hitting olecranon process, uneven contact with the ground).

III. Anatomy
Dislocation of phalanx at the interphalangeal joint.

IV. Symptoms
Pain and disfigurement.

V. Signs
Ecchymosis, decreased ROM, deformity.

VI. Radiology
Indicated before and after reduction; x-ray may reveal associated avulsion fracture.

VII. Special Studies
None.

VIII. Diagnosis:
Dislocation; location: toes.

IX. Differential Diagnoses
Fracture.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Closed reduction, (distraction following traction in the direction opposite to the dislocating force); buddy taping to adjacent toe. Return to play is allowed after ROM and strength are normal.

Consultation: rotational deformities, fractures of the proximal phalanx

XI. Complications
Contusion, fracture, hematoma, tendon injury, post-traumatic arthritis.

XII. Prevention
Protective gear, proper technique.

FRACTURE

FIFTH METATARSAL FRACTURE: AVULSION VS. JONES FRACTURE (TYPES I–III)

I. Epidemiology[7]
Post-pubescent athletes of both sexes; *avulsion fracture:* basketball, football, tennis; *Jones fracture:* running activities, especially with prolonged or intense training schedules.

II. Mechanism of Injury[10]
Avulsion fracture: inversion with or without plantar flexion in association with a forceful contraction of the peroneus brevis (eversion and dorsiflexion); *Jones fracture:* repetitive microtrauma without acute inversion.

III. Anatomy
Avulsion of the styloid process of the fifth metatarsal by the peroneus brevis; Jones fracture of the fifth metatarsal shaft within 1.5 cm of the tuberosity (area of relative avascularity).

IV. Symptoms
Avulsion Fracture: pain and swelling at the base of the fifth metatarsal; *Jones fracture:* progressive aching and discomfort over the lateral foot.

V. Signs
Avulsion fracture: point tenderness at base of the metatarsal; *Jones fracture:* palpable tenderness over base of fifth metatarsal, positive tuning fork test.

VI. Radiology
Avulsion fracture: avulsed fragment of fifth metatarsal tuberosity (Figure 54.3); *Jones fracture:* radiolucent fracture line, periosteal reaction, excessive callus on the lateral cortical margin, and intramedullary sclerosis (Figure 54.4A).

VII. Special Studies
Avulsion fracture: none; *Jones fracture:* Tc99m bone scan is positive (Figure 54.4B).

VIII. Diagnosis
Avulsion fracture of the fifth metatarsal; fifth metatarsal fracture (Jones fracture).

IX. Differential Diagnoses
Ankle sprain, avulsion fracture of the fifth metatarsal, epiphyseal injury, irritation of an os vesalius, Jones fracture, metatarsalgia.

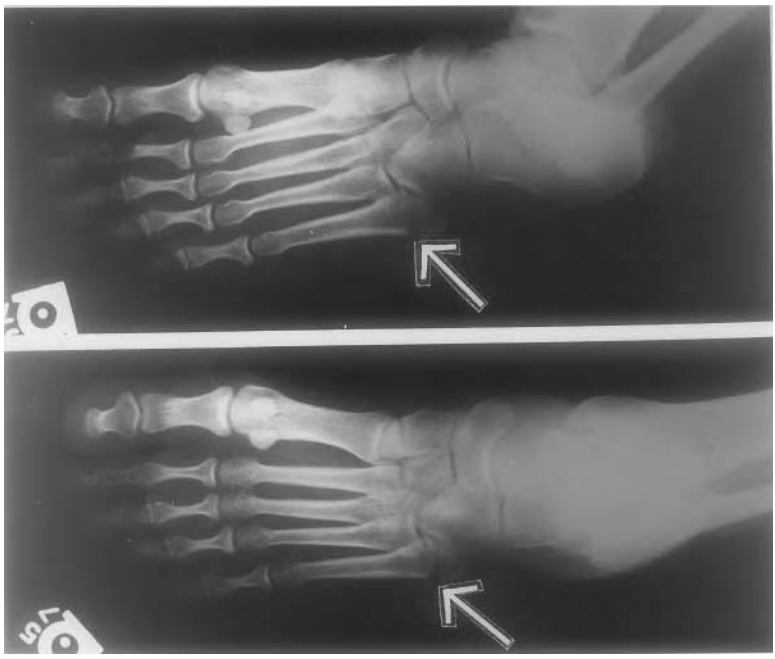

Figure 54.3 An avulsion fracture of the fifth metatarsal (arrows).

X. Treatment/Rehabilitation[10]

Avulsion Fracture

A. Initially: Treat with RICE and analgesics, as needed.
B. Long-term: Because this fracture occurs at a relatively vascular portion of the bone, a short leg cast is usually sufficient (4 to 6 weeks). Eversion strapping or a wooden shoe (to dissipate force) is recommended for a nondisplaced or hairline fracture, followed by physical therapy (ROM, PREs). Return to play is allowed after patient is after pain free and after adequate ankle rehabilitation.

Consultation: Recurrence

Jones Fracture

A. Initially: Non-weight-bearing with crutches.
B. Long-term: Non-weight-bearing leg cast for 6 to 8 weeks; an additional 2 to 4 months for delayed union. Because of prolonged periods of immobilization and attendant disability most competitive athletes or active individuals should be offered operative management (curettage, grafting, or screw fixation); most delayed unions and all nonunion fractures should be managed operatively. Return to play after radiographic demonstration of healing (6 to 8 weeks) and appropriate physical therapy (7 to 14 weeks) (ROM + PRE).

Consultation: should be offered for all

XI. Complications

Avulsion fracture: nonunion (rare); *Jones fracture:* delayed or nonunion fractures (20 to 50%).

XII. Prevention[6]

Orthotics or strapping/taping ankle joint to reduce inversion stress; progressive training schedules, proper technique.

OS CALCIS STRESS FRACTURE

I. Epidemiology

Dancing, running, aerobics (high-impact).[5,6]

II. Mechanism of Injury

Repetitive microtrauma.[8]

III. Anatomy

Stress fracture of long, thin os calcis.

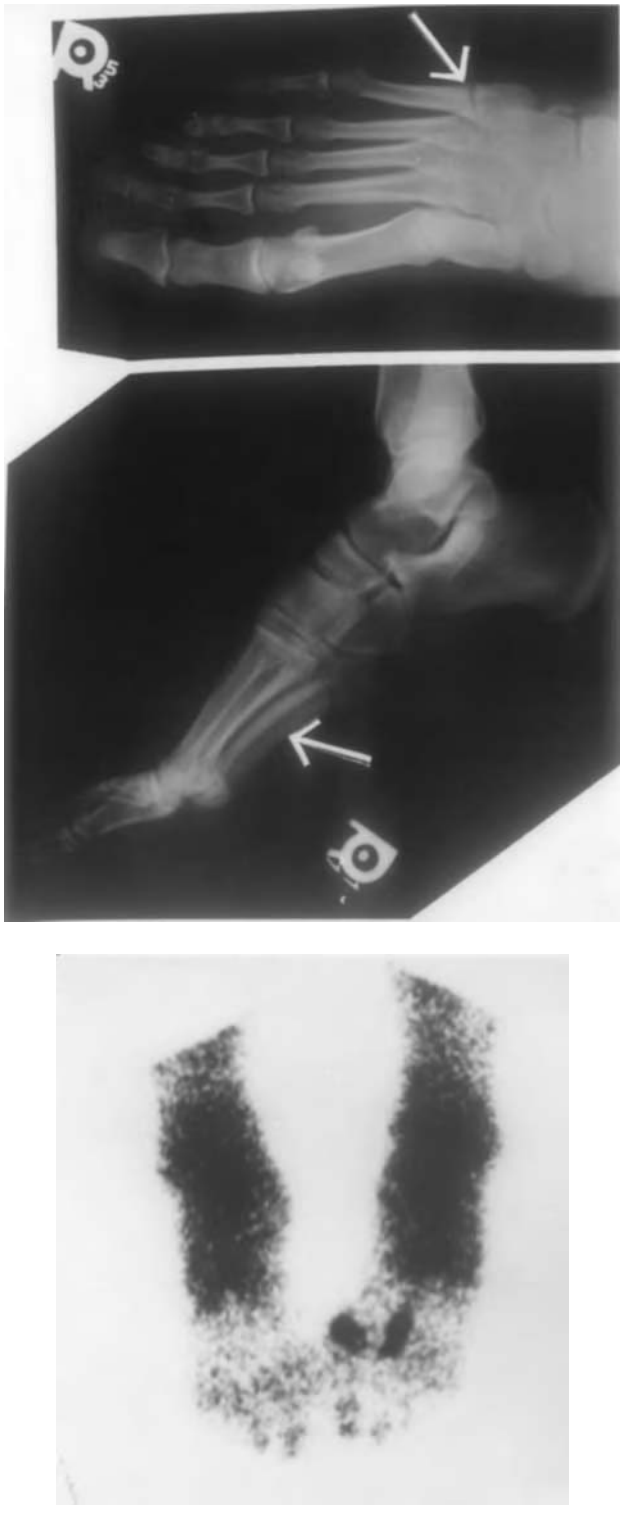

Figure 54.4 (Top) Radiographs showing stress fracture of the fifth metatarsal (Jones fracture; arrows). (Bottom) Positive bone scan revealing stress fracture of distal second metatarsal (6 days after onset of pain). A coincidental inflamed first-toe metatarsalphalangeal joint (MTPJ) bursa (bunion) is also evident.

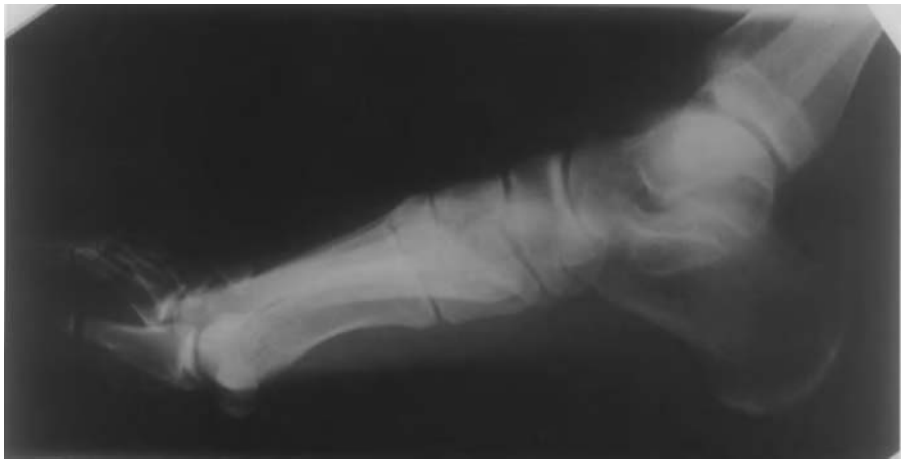

Figure 54.5 Calcaneal stress fracture.

IV. Symptoms
Gradually worsening heel pain.

V. Signs
Calcaneal tenderness, as opposed to surrounding soft tissue (e.g., Achilles tendon); positive tuning-fork sign.

VI. Radiology
X-rays are usually negative for 1 to 2 weeks; thereafter, they may show a fracture line at the posterosuperior margin of the os calcis just anterior to the apophyseal plate at a right angle to the trabeculae (Figure 54.5).

VII. Special Studies
Tc99m bone scan is positive after 2 weeks of symptoms.

VIII. Diagnosis
Os calcis stress fracture.

IX. Differential Diagnoses
Achilles tendinitis, "bone bruise."

X. Treatment/Rehabilitation[6]
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Rest and immobilization with a weight-bearing cast (3 to 6 weeks) or orthotic fitted with extra heel cushioning. A gradual return to play is allowed when patient is pain free. Modify exercise prescription.

Consultation: none

XI. Complications
Reinjury, complete fracture.

XII. Prevention
Adequate cushioning, avoidance of repetitive high-impact training.

METATARSAL STRESS FRACTURE: MARCH FRACTURE

I. Epidemiology[7,8]
Running, marching, volleyball, basketball, dancing; found in post-pubescent athletes of both sexes; accounts for 20% of all athletic stress fractures. Predisposing factors include anemia, anorexia nervosa, short first ray syndrome, cavus foot, and anterior ankle impingement syndrome.

II. Mechanism of Injury
Repetitive microtrauma, especially in the hyperpronated foot, or sudden increase in intensity or duration of training.

III. Anatomy
Stress fracture of metatarsal diaphysis.

IV. Symptoms
Gradual worsening pain in the forefoot.

V. Signs
Palpable tenderness of involved metatarsal; may note some swelling, hyperpronation; tuning fork test is positive.

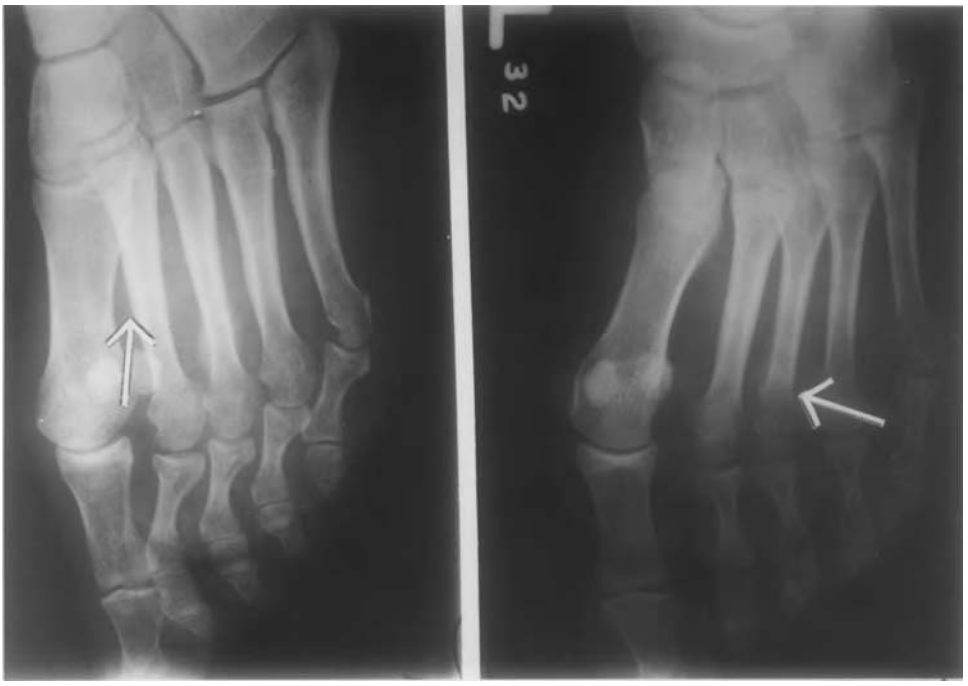

Figure 54.6 Stress fracture of third metatarsal (arrow).

VI. Radiology
X-rays initially are negative but will show callus formation in 2 weeks (Figure 54.6).

VII. Special Studies
Tc99m bone scan is positive after 2 weeks.

VIII. Diagnosis
Metatarsal stress fracture (March fracture, Deutschländers disease).

IX. Differential Diagnoses[10]
Metatarsalgia, Morton's neuroma, interdigital neuritis, dancer's fracture, osteoid osteoma, enchondroma.

X. Treatment/Rehabilitation[6]
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Treatment depends on amount of pain and disability. Adhesive strapping, rest, and good forefoot support may be adequate (6 to 8 weeks). A short leg cast may be needed. Correct tendency to hyperpronate with orthotics or footwear. Return to play is allowed after patient is pain free.

Consultation: none

XI. Complications
Recurrence, complete fracture.

XII. Prevention
Progressive increase in training time and intensity.

METATARSALGIA
I. Epidemiology[10]
Running, aerobics; found in early adulthood (females > males); associated with shin splints, patellofemoral tracking abnormalities, and wearing of high heels.

II. Mechanism of Injury
Altered biomechanics of weight bearing (i.e., hyperpronation) with repetitive stress shifted to metatarsal heads.

III. Anatomy
Short hypermobile first metatarsal, first ray insufficiency syndrome, posteriorly displaced sesamoids, and thickened second metatarsal shaft (D.J. Morton's syndrome or Morton's foot); equinus or cavus foot deformities, central-ray overload or insufficiency.

IV. Symptoms
Pain at base of second metatarsal and heads of second and third metatarsal (D.J. Morton's syndrome); any or all metatarsals may cause symptoms at times.

V. Signs
Large callus formation under second and third metatarsal heads; palpable tenderness at base of second metatarsal and heads of second and third metatarsal; short great toe; hyperpronated foot.

VI. Radiology
X-rays reveal short first metatarsal, thickened shaft of second metatarsal, posteriorly displaced sesamoids.

VII. Special Studies
Tc99m bone scan is sometimes positive, secondary to periostitis after 2 weeks of symptoms.

VIII. Diagnosis
Metatarsalgia (Morton's foot, D.J. Morton's syndrome).

IX. Differential Diagnoses
Metatarsal stress fracture.

X. Treatment/Rehabilitation[6,10]
A. Initially: Treat with NSAID and analgesics, as needed.
B. Long-term: Callus paring; orthotics. For D.J. Morton's syndrome or Morton's foot, use an extension pad (longer pad under first metatarsal); correct tendency to hyperpronate, if appropriate. For metatarsalgia, place metatarsal bar proximal to heads. Return to play is allowed when patient is pain free.

Consultation: intractable pain and dysfunction

XI. Complications
Chronic pain.

XII. Prevention[6]
Orthotics, gradual increase of training intensity and duration.

METATARSAL FRACTURE: DANCER'S FRACTURE

I. Epidemiology[5,6]
Ballet dancers (most common fracture in using demi-pointe technique); found in post-pubescent athletes (females > males).

II. Mechanism of Injury
Loss of balance while on the ball of the point, plus rolling over the outer foot border.

III. Anatomy
Spiral fracture of the distal third of the fifth metatarsal.

IV. Symptoms
Acute pain and swelling on the lateral aspect of the foot.

V. Signs
Palpable tenderness over the distal fifth metatarsal; positive tuning fork test.

VI. Radiology
Spiral fracture.

VII. Special Studies
None indicated.

VIII. Diagnosis
Spiral metatarsal (fifth) fracture (Dancer's fracture).

IX. Differential Diagnoses
Stone bruise, tendinitis.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term. Minimal displacement — restricted activities and comfortable running shoes for 4 to 6 weeks. Moderate displacement — weight-bearing leg cast for 4 to 6 weeks. Marked displacement and comminution — internal fixation and weight-bearing leg cast for 5 to 7 weeks. Return to play is allowed when patient is pain free and physical therapy is completed.

Consultation: marked displacement and comminution

XI. Complications
Recurrence.

XII. Prevention
Proper technique, conditioning, and equipment; strapping/taping to prevent inversion.

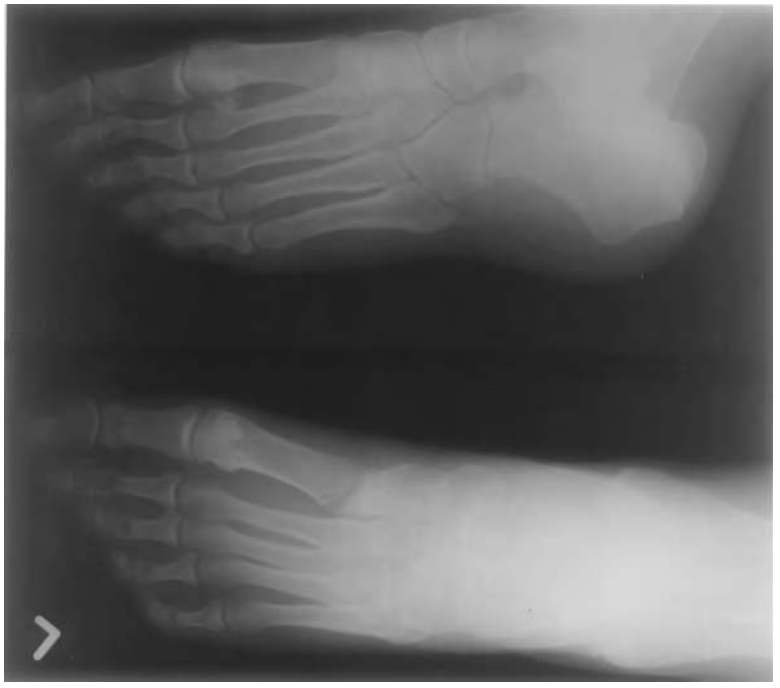

Figure 54.7 Fracture of lateral halluceal sesamoid.

SESAMOID STRESS FRACTURES VS. SESAMOIDITIS OF THE FIRST TOE

I. Epidemiology[11,12]

Floor sports (e.g., dancing, basketball); found in post-pubescent athletes; accounts for 1.2% of all running injuries; 40% of sesamoid injuries are stress fractures and 30% are sesamoiditis.

II. Mechanism of Injury

Repetitive microtrauma, especially in the high-arch/cavus foot or with hallux valgus.

III. Anatomy

Chronic inflammation and stress of two small isolated bones (medial and lateral) located on the plantar surface of the first metatarsophalangeal joint within the tendinous expansion of the flexor hallucis brevis and abductor and adductor hallucis muscles. The medial or tibial sesamoid is larger and bears more weight than the smaller, lateral, or fibula sesamoid.

IV. Symptoms

Painful area under great toe (particularly during push off).

V. Signs

Palpable tenderness, medial and lateral to first metatarsal head, amplified with passive dorsiflexion; swelling; positive tuning fork test with stress fractures. Differential lidocaine block (extraarticular) produces relief if a stress fracture or sesamoiditis is present. Intraarticular block does not completely neutralize the pain.

VI. Radiology

Routine laminagrams and axial sesamoid views initially may be negative for 2 to 3 weeks (Figure 54.7); bipartite and multipartite sesamoids may be observed (normal variant in 10 to 30% of cases).

VII. Special Studies

Bone scan and CT are positive for stress fracture but are not diagnostically specific; help to localize a medial or lateral sesamoid.

VIII. Diagnosis

Sesamoiditis of the first toe, sesamoid stress fracture of the first toe.

IX. Differential Diagnoses

Metatarsalgia, osteoarthritis, bursitis, acute fracture, osteochondritis.

X. Treatment/Rehabilitation[6,11,12]

A. Initially: Treat with PRICEMM (prevention/protection, rest, ice, compression, elevation, modalities, and medications), NSAIDs, and analgesics, as needed.
B. Long-term. For sesamoiditis — semirigid orthotics (plus contralateral neutral companion orthosis) with a metatarsal bar; a built-in J or C pad may also be helpful. Duration is normally 4 to 6 weeks. For stress fracture, use a short leg, weight-bearing cast for 4 to 6 weeks followed by protective padding (vide supra) for an additional 6 weeks. Injection therapy is not recommended. Return to play is allowed when patient is pain free.

XI. Complications

Aseptic necrosis; osteochondrosis, nonunion.

XII. Prevention

Shock-absorbent footwear (wide toe box plus inswung or straight last, depending on foot anatomy); orthotics.

NEUROPATHIES

Although not as common as osseous and soft-tissue problems of the foot, neuropathies are diagnosed most commonly in joggers or runners (60%) and in dancers (20%).[5–7] They are probably under-diagnosed because many athletes work through vague pains and sensations prior to their becoming dysfunctional, and they may be associated with the more commonly seen musculoskeletal maladies. Also, these problems may be quiescent during a static evaluation and may not be demonstrated until a dynamic test is used (running, jumping, etc.). Electromyography (EMG) and nerve conduction velocity (NCV) tests are usually only diagnostic with long-standing conditions. The differential diagnosis should include a more proximal nerve problem (e.g., lumbar disc compression). The concept of the "double crush" phenomenon may explain the altered threshold for a more distal nerve entrapment to become problematic.[13] Compartment syndromes can also produce similar neurologic symptoms and signs. In general, conservative therapy is helpful; however, surgical management may be necessary. A review of the more detailed anatomy of the area is beyond the scope of this chapter.

INTERDIGITAL NEUROMA (MORTON'S NEUROMA, MORTON'S METATARSALGIA)

I. Epidemiology

Running, dancing, aerobics; occurs in more women than men.

II. Mechanism of Injury

Repetitive microtrauma; hypermobile foot with excessive pronation; associated with high-heeled, narrow shoes.

III. Anatomy

Endarteritis, hyalinization, demyelination, fibrosis of interdigital sensory nerve, usually in third web space at leading edge of transverse metatarsal ligament.

IV. Symptoms

Poorly localized, shock-like pain; may radiate into toes or proximally during ambulation; relieved with rest or removal of shoe.

V. Signs

May be able to palpate swelling between metatarsals (approach from plantar surface); lateral compression of metatarsals reproduces pain.

VI. Radiology

Negative.

VII. Special Studies

Bone scans are negative.

VIII. Diagnosis

Interdigital neuroma (Morton's neuroma, Morton's metatarsalgia).

IX. Differential Diagnoses

Interdigital neuritis; fracture.

X. Treatment/Rehabilitation

A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Injection; wide toe boxes, lower heels, metatarsal pads; surgical resection (80% success).

XI. Complications

Intractable pain.

XII. Prevention

Proper footwear, control of excessive pronation.

ENTRAPMENT OF FIRST BRANCH, LATERAL PLANTAR NERVE

I. Epidemiology
Running, soccer, dance, tennis; occurs in 5 to 10% of athletes with chronic, unresolving heel pain.

II. Mechanism of Injury
Inflammation from chronic pressure on nerve; hypermobile foot with excessive pronation; hypertrophy of abductor hallucis or quadratus plantae muscles.

III. Anatomy
Entrapment occurs between deep fascia of the abductor hallucis and medial caudal margin of quadratus plantae muscle or plantar side of plantar ligament, or in osteomuscular canal between calcaneus and flexor digitorum brevis.

IV. Symptoms
Chronic heel pain is intensified by walking and especially running. It may be dull, achy, or a sharp pain; occasionally pain radiates into ankle. Less commonly, numbness of heel/foot is noted.

V. Signs
Exclude more proximal lesion first; exquisitely tender over first branch of lateral plantar nerve deep to abductor hallucis muscle; infrequently, positive Tinel's sign.

VI. Radiology
Possible heel spur.

VII. Special Studies
None indicated.

VIII. Diagnosis
Entrapment of first branch, lateral plantar nerve.

IX. Differential Diagnoses
Plantar fasciitis.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Injection, surgical release; orthotics to control hyperpronation.

XI. Complications
None.

XII. Prevention
Control of hyperpronation.

SURAL NERVE ENTRAPMENT

I. Epidemiology
Running.

II. Mechanism of Injury
Acute twisting injury after recurrent ankle sprains. Ganglions of the peroneal sheath or of calcaneal cuboid joint are sometimes associated with fractures of the base of the fifth metatarsal.

III. Anatomy
Medial sural nerve courses between the gastrocnemius, goes deep halfway up the leg, joins with the peroneal communicating nerve, and travels along the border of the Achilles. It gives off branches 2 cm above the ankle: one to the lateral heel and one that anastomoses with the superficial peroneal nerve and travels to the fifth metatarsal. Entrapment sites include: (1) along Achilles tendon, (2) by fifth metatarsal, and (3) calcaneal/cuboid joint.

IV. Symptoms
Shooting pains and paresthesia over lateral heel, ankle, and/or foot.

V. Signs
Positive Tinel's sign; occasionally, numbness.

VI. Radiology
Possibly healing fifth metatarsal fracture; osteophytes off calcaneal-cuboid area.

VII. Special Studies
Electromyography can reveal terminal latency (seen only in chronic cases); NCV demonstrates delayed conduction (seen only in chronic cases).

VIII. Diagnosis
Sural nerve entrapment.

IX. Differential Diagnoses
Recurrent ankle sprain.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Injection, surgical release.

XI. Complications
Intractable pain.

XII. Prevention
Appropriate ankle rehabilitation.

MEDIAL PLANTAR NERVE ENTRAPMENT OR NEURAPRAXIA (JOGGER'S FOOT)

I. Epidemiology
Running, gymnastics; found in post-pubescent athletes with equal occurrence in men and women.

II. Mechanism of Injury
Repetitive foot eversion, excessive heel valgus, and/or hyperpronation of feet.

III. Anatomy
Compressive irritation of medial plantar nerve as it travels underneath the flexor retinaculum or curves deep to the abductor hallucis muscle. The nerve continues on both the medial and lateral side of the flexor hallucis longus tendon.

IV. Symptoms
Aching, burning, numbness, and tingling of the medial sole and small portion of medial aspect of heel. It is not uncommon to have onset after use of a new orthotic. Pain may be worse with running on level ground or stair workouts.

V. Signs
Positive Tinel's sign over nerve; hypesthesia and tenderness along medial aspect of sole and arch in region of navicular tuberosity.

VI. Radiology
Negative.

VII. Special Studies
Electromyography can show terminal latency (seen only in chronic cases); NCV demonstrates delayed conduction (seen only in chronic cases).

VIII. Diagnosis
Medial plantar nerve entrapment or neurapraxia (jogger's foot).

IX. Differential Diagnoses
Tarsal tunnel syndrome (more proximal involvement).

X. Treatment/Rehabilitation
A. Initially: Treat with NSAIDs and analgesics, as needed.
B. Long-term: Injection; medial heel wedge.

XI. Complications
Muscle atrophy.

XII. Prevention
Correct excessive valgus position with orthotic.

TARSAL TUNNEL SYNDROMES (JOGGER'S/RUNNER'S FOOT, MEDIAL PLANTAR NEURAPRAXIA, CALCANEAL BRANCH NEURODYNIA)[14]

I. Epidemiology
Uncommon in athletes; possibly gymnastics, football, running.

II. Mechanism of Injury
Tumors: lipomas, neuromas, synovial cysts; trauma: fractures, contusions, spasm, laceration, tight cast, edema, adhesions; anatomic factor: valgus alignment; inflammatory condition: arthritis, thrombophlebitis, spondylitis, tenosynovitis. Also, overuse, aging, poorly fitting footwear, fluid retention, poorly graduated training schedule, uneven surfaces, repetitive foot eversion.

III. Anatomy
Posterior tibial nerve and branches, medial and lateral plantar nerves, and medial calcaneal nerve within tunnel (Figure 54.1B). Entrapment sites include:.
A. Proximal — beneath the laciniate ligament (superior flexor retinaculum) involving the posterior tibial nerve and all branches.
B. Distal — deep to the abductor hallucis muscle involving medial and/or lateral plantar nerves.

IV. Symptoms
Insidious onset of burning, aching pain from posterior aspect of heel, continuing medially below the malleolus to the mid-tarsal zone, sometimes worse at night; aggravated by weight bearing and standing. Decreased sensation (in advanced cases) in the sole, arch, and heel.

V. Signs
Positive Tinel's sign over tunnel; palpation of involved nerve often causes pain to radiate both

proximally and distally. Forcing the heel into eversion while palpating the nerve may bring out subtle cases. In advanced cases, look for interosseous muscle weakness. Sensory findings include hypesthesia to pinpoint discrimination; positive tourniquet test.

VI. Radiology
X-rays are usually normal but may reveal exostoses or callus from healing fracture (e.g., calcaneal ankle).

VII. Special Studies
Electromyography results depend on location of entrapment (e.g., entrapment of posterior tibial nerve will attack more muscles); may reveal terminal latency to abductor hallucis (medial plantar nerve) or abductor digiti minimi (lateral plantar nerve), or both. NCV demonstrates delayed conduction with 1% lidocaine into tunnel but cannot distinguish more distal lesions.

VIII. Diagnosis
Tarsal tunnel syndrome(s) (jogger's/runner's foot, medial plantar neurapraxia, calcaneal branch neurodynia).

IX. Differential Diagnoses
Plantar fasciitis, lipomas, ganglions (actual cause of pressure within tunnel).

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Injection; medial arch support pad or heel wedge; surgical release (75% success rate).

XI. Complications
Chronic pain, may be disabling.

XII. Prevention
Progressive increase in training; smooth, level training surface; avoid excessively tight lacing and lateral heel wear with orthotic.

DEEP PERONEAL NERVE ENTRAPMENT (SKI BOOT SYNDROME, ANTERIOR TARSAL TUNNEL SYNDROME)

I. Epidemiology
Running, soccer, hockey, skiing, dancing.

II. Mechanism of Injury
Occurs in soccer (frequent blows to dorsum of foot), skiing (tight-fitting boots), running (either tight-fitting shoes or after recurrent ankle sprains); often associated with recurrent ankle sprains.

III. Anatomy
Entrapment sites include under inferior extensor retinaculum, under tendon of extensor hallucis brevis, superior edge of extensor retinaculum where extensor hallucis longus tendon crosses, talonavicular joint (osteophytes), and between first and second metatarsal bases.

IV. Symptoms
Pain in dorsum of foot with occasional radiation into first web space. Running on curves exacerbates the pain. Pain may be spasmodic dull, aching, or sharp and may occur at night.

V. Signs
Rule out more proximal lesions (e.g., common peroneal nerve). Tenderness in anterior compartment after exercise suggests exertional compartment syndrome.

VI. Radiology
Possibly only osteophytes.

VII. Special Studies
Electromyography can show terminal latency (seen only in chronic cases); NCV demonstrates delayed conduction (seen only in chronic cases).

VIII. Diagnosis
Deep peroneal nerve entrapment (ski boot syndrome, anterior tarsal tunnel syndrome).

IX. Differential Diagnoses
Anterior compartment syndrome.

X. Treatment/Rehabilitation
A. Initially: Treat with RICE, NSAIDs, and analgesics, as needed.
B. Long-term: Injection, surgical release.

XI. Complications
Intractable pain.

XII. Prevention
Avoid tight-fitting footwear; complete appropriate rehabilitation for ankle sprains; use padding to avoid excessive trauma to area.

SUPERFICIAL PERONEAL NERVE ENTRAPMENT

I. Epidemiology
Running, soccer, hockey, tennis, racquetball, dancing; equal occurrence in men and women.

II. Mechanism of Injury
Chronic ankle sprains subject nerve to recurrent stretching.

III. Anatomy
The superficial peroneal nerve (a branch of the common peroneal nerve) runs through the anterolateral compartment between the anterior intermuscular septum and the fascia of the lateral compartment, which it pierces (site of entrapment) 10.5 to 12.5 cm above the tip of the lateral malleolus. The nerve becomes subcutaneous approximately 6.5 cm above the lateral malleolus and divides into the intermediate and medial dorsal cutaneous nerves.

IV. Symptoms
Chronic pain (e.g., several years) along the outer border of the distal calf and the dorsum of the foot and ankle. Complaints of numbness and paresthesia may be noted. Pain is worse with activity. Condition is frequently associated with previous trauma to the extremity (e.g., ankle sprain) or past history of fasciotomy for compartment syndrome.

V. Signs
Rule out more proximal lesions (e.g., sciatica, common peroneal nerve). Point tenderness can be observed approximately 10.5 to 12.5 cm above distal tip of fibula. Pain may radiate both proximally and distally; may elicit numbness. Fascial defect may occur in 75%. Among other tests, have patient dorsiflex and evert foot against resistance; while palpating, plantar flex and invert foot, then repeat the maneuver and apply pressure to nerve.

VI. Radiology
None.

VII. Special Studies
Electromyography can show terminal latency (seen only in chronic cases); NCV demonstrates delayed conduction (seen only in chronic cases).

VIII. Diagnosis
Superficial peroneal nerve entrapment.

IX. Differential Diagnoses
Common peroneal nerve entrapment, sciatica.

X. Treatment/Rehabilitation
A. Initially: Treatment with NSAIDs is not usually helpful.
B. Long-term: Muscle rehabilitation of peroneal muscles; injection.

XI. Complications
Intractable pain.

XII. Prevention
Effective rehabilitation of ankle sprains.

MISCELLANEOUS

Black heel is a symptomatic discoloration of skin on the heel that is actually from multiple layers of punctate specks of dried blood.[12] Other items in the differential diagnosis include plantar warts and malignant melanoma. Paring with a scalpel both makes the diagnosis and may provide short-term treatment. Proper-fitting, well-cushioned footwear may help prevent recurrences.

Calcaneal apophysitis or *Sever's disease* (Figure 54.8) occurs in children ages 7 to 13 and is more prevalent in short, stocky males. They will complain of progressive heel pain and may even have a limp. The condition is usually unilateral but may be bilateral. The posterior calcaneal may be tender and the tuning fork sign positive, but the Achilles tendon is not tender. The heel cord is usually tight and reduced dorsiflexion may be noted. The apophysis of the calcaneus becomes inflamed via traction by the Achilles tendon or by direct trauma from running and jumping (e.g., soccer, gymnastics). X-rays after 2 to 4 weeks of symptoms may show sclerosis and fragmentation. A bone scan will light up in the same area. Initially, rest, NSAIDs, and a heel lift (1/4-inch) will help, but recalcitrant cases may require short leg casting for 2 to 4 weeks. A heel lift can reduce the traction by the Achilles tendon. Removal or trimming of the shoe's heel counter may also be helpful. Aseptic necrosis is a bothersome complication. Heel cord stretching and proper training techniques may prevent its occurrence. Recovery may take months to a year.

Aseptic necrosis of the tarsal navicular bone or *Kohler's disease* is believed to be caused by compression at the time of rapid bone growth (age 5 to 10). Symptoms can vary from vague discomfort to disabling pain. In addition to point tenderness over the navicular bone, the tuning fork test is positive. An x-ray will reveal sclerotic,

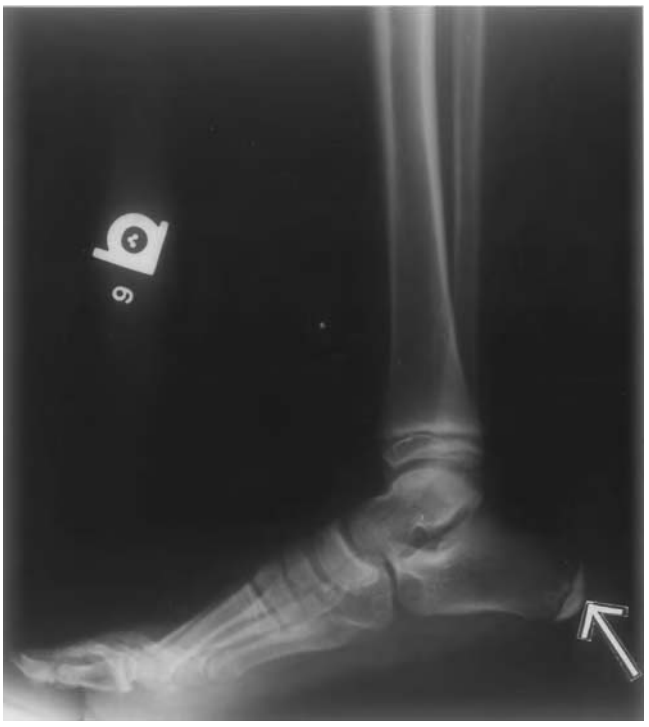

Figure 54.8 Calcaneal apophysitis (Sever's disease; arrow).

flattened bone (resembling a coin or pancake). A bone scan would obviously have a hot spot by the navicular. It is rare to have this condition in adulthood. It can be treated with partial weight bearing and analgesics. The athlete can return to competition when pain free. Consultation should be sought in cases of prolonged pain (e.g., 4 to 6 weeks).

Occasionally an accessory bone, the os supranavicularis (Figure 54.9), can be irritated by footwear or be traumatized by a torsional injury. Tenderness to deep palpation on the dorsal–medial aspect of the foot, with x-ray evidence of the accessory bone, leads to a diagnosis of *os supranavicularis pinch syndrome*. Initially, padding is helpful, but short leg casting for 2 to 4 weeks may be necessary. Intractable pain is an indication for surgery.

Aseptic necrosis of the metatarsal head (usually the second) is known as *Freiberg's disease* (Figure 54.10). It usually affects 11 to 19 year olds, who will complain of pain and have thickening, tenderness, and a positive tuning fork test over the involved metatarsal head. An x-ray at 1 to 2 weeks will show osteosclerosis; at 1 to 2 months, osteolysis. Initially, the RICE regimen will help. An anterior arch pad is useful in the early stages. Some cases will require 3 to 6 weeks of immobilization in a weight-bearing cast. Persistent pain and/or excessive regrowth of the bone are indications for consultation.

The foot may develop "lumps" or "bumps."[15] When one occurs at the back of the heel, it may be either a *pump bump* or a *runner's bump*. Both are tender, but the pump bump may form an adventitious bursa. It is caused by repetitive trauma to the area by a shoe. An x-ray will show an exostosis. Runner's bump may be caused by "tugging" by the Achilles tendon on the adult calcaneus (without an apophyseal plate). An x-ray will show a bone spur extending upward into the Achilles tendon. Both conditions benefit from the use of a heel lift or padded heel counters. If chronic pain develops, surgical excision may be necessary.

A *surfer's knot* is a hyperkeratotic nodule overlying the metatarsal–phalangeal joints.[15] It results from chronic irritation caused by a surfer paddling out to a wave. It usually is painless and freely movable. Only rarely will a cellulitis occur there. Changing technique can alleviate the problem.

On the sole of the foot, a *plantar fibromatosis* or *dancer's bump* may present as a painful, moveable subcutaneous nodule.[5,6] It is a proliferation of fibrous tissue of the plantar aponeurosis. If adjacent nerves are compressed or entrapped,

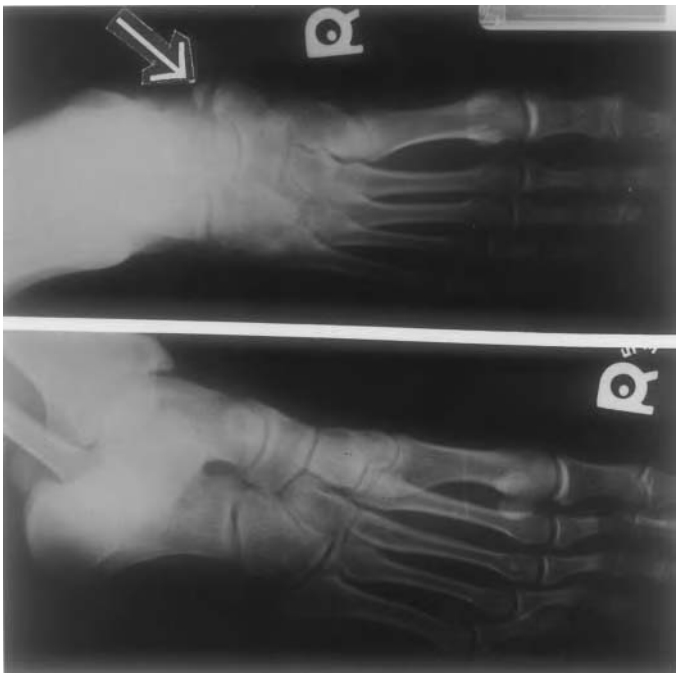

Figure 54.9 Os supranavicularis (arrow).

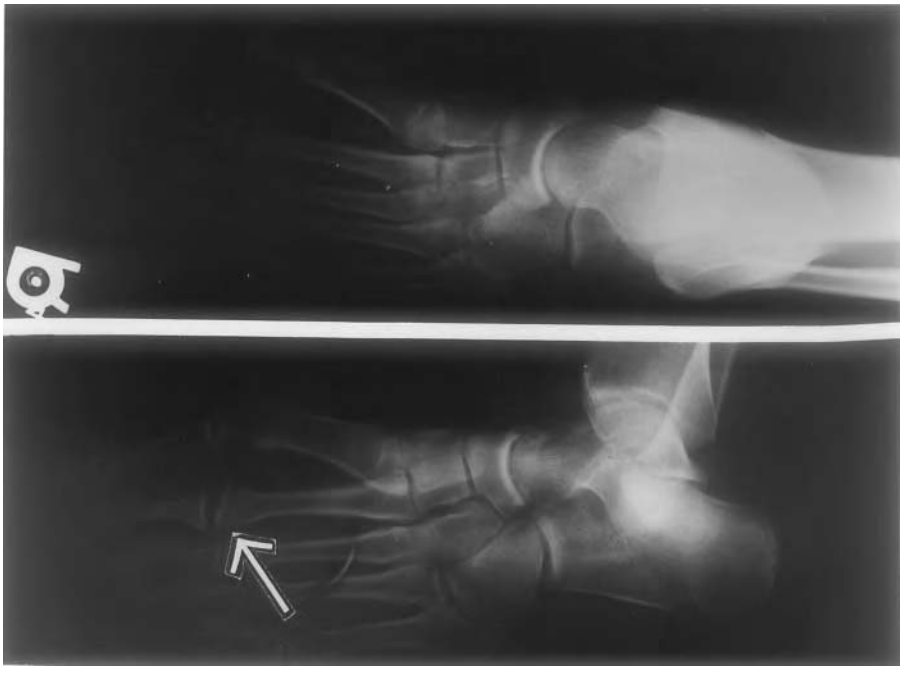

Figure 54.10 Avascular necrosis of the second metatarsal head (Freiberg's infraction; arrow).

surgical excision may be necessary. In children, the pathology has been mistaken for a malignant fibrosarcoma.

The great toe serves a critical role of balance for the foot. It is also subject to many maladies. Some are because of a genetic predisposition, such as hallux valgus (lateral displacement) or varus (medial displacement) deformities. *Hallus rigidus* (fixed first MTP joint) may either be congenital or secondary to trauma. It does not allow for normal dissipation of force. When these conditions become symptomatic, orthotics or padding can be tried, but surgical intervention may be necessary. *Bunions* can form in individuals with hallux valgus deformity. The chronic irritation brought on by tight shoes, dancing, or significant deformity may also result in the formation of an adventitious bursa. Bunion pads and a wider toe box help initially. Correcting the tendency toward overpronation can also help. Osteotomy is necessary for patients with disabling pain. A tailor's bunion represents inflammation of the lateral aspect of the fifth metatarsal from ill-fitting footwear. Treatment consists of RICE, NSAIDs, padding, and appropriate footwear.

Turf toe is a sprain injury to the joint capsule and surrounding ligaments of the first MTP joint brought on by severe hyperextension (dorsiflexion) of the great toe.[12] Football players (e.g., linemen) and other athletes doing tire and spider drills who play in fairly flexible artificial turf shoes are often the ones affected by the problem. The pain is over the first MTP joint and is worsened by passive dorsiflexion. Initially, RICE, NSAIDs, and buddy taping help. An athlete can return to play when able to push off adequately. A steel or tin insert in the toe box can provide additional support to the toe, which also should be taped. Untreated, 50% of players lose range of motion and develop some degree of arthritis.

Ingrown toenails can probably be avoided. It is well worth spending 1 minute at the beginning of the season to instruct players to trim their nails straight across without going into the cuticle region. Some feel a genetic predisposition may exist. Initially, warm soaks and an antibiotic-soaked cotton pledget may help. Oral antibiotics may be necessary. Granulomas can form. Local excision may be required.

A *subungual hematoma*, (black nail or runner's toe) may form secondary to repetitive or direct trauma to the toe because of poorly fitting shoes, or acutely when stepped on or crushed. A hot, sterile needle can relieve the pressure for those with acute pain. Chronic cases need to be managed by correcting the predisposing problem (tight toe box, etc.).

Dynamic instability caused by ligamentous laxity, soft-tissue and bony abnormalities or trauma, and systemic disease (e.g., arthritis, diabetes) can produce a *flat foot* (pes planus). Symptoms include postural arch fatigue and pain on ambulation, running, or jumping. Pain is diffuse over the metatarsal and plantar surface. Shoe modification with wedges and soft or hard orthotics allow most flat-footed individuals an opportunity to participate in sports and recreational activities. Surgery may be necessary.

SUMMARY

Foot injuries represent a significant threat to both the competitive and the recreational athlete. Despite its relatively small size in comparison to the rest of the body, the foot is an amazingly complex structure. A better understanding of the kinesiology of the foot enables the practitioner to diagnose and treat these injuries more accurately and effectively.

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PART VI
SPORTS INJURIES: MEDICAL PROBLEMS IN ATHLETES

55 INFECTIOUS DISEASE AND THE ATHLETE

John P. Metz

INTRODUCTION

Most athletes agree that exercise helps maintain overall health and decreases one's susceptibility to common infections. In a survey of 170 marathon runners, 90% reported they definitely or mostly agreed they "rarely got sick."[1] Ninety percent of non-elite athletes who engaged in regular, moderate exercise reported that they rarely got sick.[2–4] In a 1989 *Runner's World* survey, 60.7% of subscribers reported catching fewer colds since starting running, while only 4.9% reported catching more.[5] Elite athletes, however, feel that intense training lowers their immunity and increases their vulnerability to illness.[4] This chapter briefly discusses how exercise affects the immune system and the risk of infections and then details the management of common infections in athletes, the management of the HIV-positive athlete, and guidelines for return to play decisions.

IMMUNOLOGY, INFECTIONS, AND EXERCISE

A detailed discussion of the interaction between the immune system, exercise, and infections, is too long for this chapter. Briefly, exercise has known effects on the innate[3,4,7–12] and acquired[3,4,9,13–15] portions of the immune system. A brief period of measurable immunosuppression that occurs after acute, intense, physical activity characterized by decreased respiratory mucosal ciliary action, salivary immunoglobulin A levels, natural killer cell count and activity, T-lymphocyte count, and CD4/CD8 ratio is known as the immunologic "open window."[4,8,9,13] During this time, infections are theoretically more likely to invade the host and cause infections (Level of Evidence C, consensus/expert opinion). This seems to correlate with studies of marathon runners and elite orienteers who show increased risk of infection with strenuous physical activity (Level of Evidence B, clinical cohort and epidemiologic studies).[16–18] Studies of moderate exercise have had variable results but seem to indicate a decreased risk of infection (Level of Evidence B, clinical cohort and epidemiologic studies).[19–22] Studies looking at the relation between immune markers and infection risk, however, have found no consistent correlations between the two.[23–29] Currently, the most popular theory of the relation between exercise intensity and infection risk is the J-curve hypothesis proposed by Nieman (Figure 55.1). According to this theory, moderate exercise lowers risk of infection to below that of being sedentary, while strenuous exercise imposes the highest risk of all (Level of Evidence C, consensus/expert opinion).[29] More study is needed, however,[8] as the link between moderate exercise and infection is less clear. Most studies of infection and exercise are relatively small and rely on patient recall and self-reporting for diagnosis. Also, other factors such as pathogen exposure,

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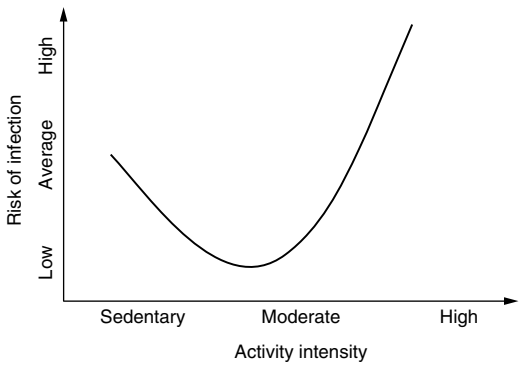

Figure 55.1 J curve of exercise and susceptibility to infection. (From Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw–Hill, New York, 2001, 357–370. With permission.)

stress, sleep, nutrition, and environment may play a confounding role.[8]

INFECTION AND TRAINING

Fever

Fever impairs concentric muscle strength, mental cognition, and pulmonary perfusion. It also increases overall systemic metabolism and insensible fluid loss, resulting in increased caloric, oxygen, and fluid requirements. These factors decrease exercise capacity and increase risk of injury. Treatment of fever should not be based solely on the degree of temperature elevation but rather on the level of patient discomfort. Options include acetaminophen (650 to 1000 mg every 4 to 6 hours, maximum of 4000 mg in a day) and nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (800 mg every 8 hours).[30] One should caution athletes that, when they are dehydrated, using NSAIDs may reduce renal blood flow and precipitate acute renal failure.[31]

Rhinorrhea and Nasal Congestion

The most common complaints related to infectious diseases in athletes are rhinorrhea and nasal congestion most commonly seen with upper respiratory infections (URIs) and acute sinusitis. URIs are quite common, with adolescents and adults averaging 2 to 4 per year. Typical symptoms include rhinorrhea, congestion, sneezing, sore throat, cough, hoarseness, malaise, and headache. Typical findings include nasal mucosa edema and erythema, clear to cloudy rhinorrhea, oropharyngeal erythema, and occasionally cervical lymphadenopathy. Temperature greater than 100°F (37.7°C) is unusual and should prompt a search for other causes.[1] Treatment is straightforward and is aimed at symptom relief until resolution, usually within 7 to 10 days. Rest and good hydration are paramount. Oral or nasal decongestants can help relieve congestion, but side effects can include nervousness, insomnia, tachycardia, and increased blood pressure. If rhinorrhea and sneezing are the predominant symptoms, sedating antihistamines are good choices as their anticholinergic action dries the nasal mucosa and increases mucous viscosity. They can cause intolerable sedation, dry mouth, and constipation, however.[32] Athletes in warm climates should use these medications with caution as they impair sweat production and increase the risk of heat exhaustion or heat stroke.[33] Nasal ipratropium will provide

TABLE 55.1
Common URI Medications and Doses

Medication Type	Medication Name	Dose
Decongestant, oral	Pseudoephedrine	30–60 mg QID
Decongestant, nasal	Phenylephrine (0.5%, 1.0%)	2–3 sprays q4h p.r.n., maximum 3 days
	Oxymetazoline (0.05%)	1–2 sprays BID, maximum 3 days
Antihistamine, sedating	Chlorpheniramine	4 mg q4–6h
	Brompheniramine	4 mg q4–6h
	Diphenhydramine	25-50 mg q4–6h
	Clemastine	0.5-1.0 mg BID
Antihistamine, nasal	Azelastine	2 sprays BID
Anticholinergic, nasal	Ipatroprium 0.06%	2 sprays TID-QID

Source: Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, 357–370. With permission.

TABLE 55.2
Symptoms of Common Infectious Causes of Acute Cough

	Fever	Shortness of Breath	Mucous Production	Nasal Symptoms	Symptom Duration
URI	–	–	±	+	<2 weeks
Sinusitis	±	–	+	+	>2 weeks
Bronchitis	±	±	±	–	Variable
Pneumonia	+	+	+[a]	–	<2 weeks

[a] Typical pneumonia; atypical pneumonia typically has a severe dry cough.

Note: +, usually present; ±, variably present; –, not commonly present;

Source: Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, 357–370. With permission.

the anticholinergic effect of the non-sedating antihistamines without the systemic side effects.[34] See Table 55.1 for names and doses of specific medications.

In the United States, acute sinusitis accounts for over 16 million office visits a year, and is the fifth most common reason why doctors prescribe antibiotics. Typically, diagnosis relies on a constellation of signs and symptoms combined into a clinical impression. Common indicators are unilateral sinus pain and tenderness, purulent rhinorrhea, lack of response to standard URI therapy, sinus pain increased with leaning forward, maxillary toothache, and "double sickening" (which refers to the patient who has a URI that starts to improve but then gets acutely worse). Fever and other constitutional symptoms may or may not be present. Trans-illumination of the sinuses is not always reliable to diagnose sinusitis. Radiographs of the sinuses are generally not useful.[35]

Treatment should include analgesics and decongestants for symptomatic relief. Nasal saline rinses, available over the counter or by mixing 1/4 teaspoon of table salt in 8 ounces of warm water, can give short-term relief and help remove mucous. Placing a warm washcloth over the affected sinus and its corresponding nostril may also help. Sedating antihistamines are not recommended because they increase mucous viscosity and may thus impede sinus drainage. Antibiotics should cover the most common causative pathogens: *Streptococcus pneumoniae*, *Haemophilus influenza*, and *Moraxella catarrhalis*. Appropriate first-line choices include a 10- to 14-day regimens of amoxicillin (500 mg TID), and trimethoprim–sulfamethoxazole DS (one pill BID). Second-line choices include cefuroxime (250 to 500 mg BID), amoxicillin–clavulanate (875 mg BID), doxycycline (100 mg BID), and clarithromycin (500 mg BID).[35]

Cough

Cough is among the top five reasons why patients see physicians, resulting in approximately 30 million office visits per year. Americans spend roughly $600 million dollars a year on cough medications.[36] The most common infections that cause acute cough are URIs, sinusitis, bronchitis, and pneumonia.[37] The signs and symptoms of common causes of cough are listed in Table 55.2. The treatment of cough associated with URI and acute sinusitis must first focus on treating the underlying cause.[38] Second, patients who are smokers should be advised to quit. Third, adequate hydration reduces mucous viscosity and eases expectoration. Frequently, these measures alone may provide significant relief.[38] If the cough is especially irritating, however, it may be treated with medicines. In addition to comforting the patient, they may decrease the risk of complications of repetitive coughing: pneumothorax, chest wall muscle strains, rib fractures, and post-tussive syncope. The most effective cough suppressant is a narcotic such as codeine (10 to 30 mg q3–4h). In addition to cough suppression, it will provide sedation to help the patient sleep. Several nonnarcotic options are available. The cough suppressant dextromethorphan is available in a number of different cough and cold combination medicines. Benzonatate (100 mg TID) is another choice that comes in pill form. The expectorant guaifenesin (600 to 1200 mg BID) is available alone and in combination with other cold medications. Because many of these cough medicines are available in combination with other cold medicines (decongestants, antihistamines, analgesics, etc.), the provider must ensure that the patient taking multiple cold medications is not receiving a potential overdose.[38]

Symptoms of acute bronchitis include the symptoms associated with URIs, but cough is typically the most predominant feature. It may be

productive or not, and fever may or may not be present.[32] Most cases, in the absence of underlying lung disease, are viral. Atypical bacteria such as *Mycoplasma pneumonia* and *Chlamydia trachomatis* may also cause bronchitis in a small percentage of cases. Pulmonary findings are variable and can range from normal to diffuse rhonchi, and/or wheezing. Chest x-rays are usually normal and are more useful to exclude other diseases such as pneumonia.[37]

The treatment of acute bronchitis follows the same general strategy as in URIs and acute sinusitis, with rest and hydration being paramount. Bronchodilators such as albuterol (1 to 2 puffs q4–6 h) may be useful, especially in patients with wheezing or cough that increases with activity. Antibiotics are often not indicated in the first 2 weeks as most cases are viral. In a cough lasting more than 2 weeks, however, *Bordetella pertussis* should be suspected, even in adults immunized as children.[39] The decision to prescribe antibiotics may involve nonmedical factors. For example, one may prescribe antibiotics more quickly to an elite athlete with an upcoming major competition rather than waiting and risking deconditioning or poor performance. In recreational athletes, however, watchful waiting and giving antibiotics when clearly indicated are a reasonable approach. Antibiotic treatment should primarily target *Bordetella* species.[40] The first-line choice is erythromycin estolate (500 mg QID for 14 days). Second-line choices include trimethoprim–sulfamethoxazole DS (one tab BID for 14 days) or clarithromycin (500 mg BID for 7 days). Household contacts should be treated with the same regimens as well.

While recovering from acute bronchitis, the healing bronchi are more sensitive to exercise-induced changes such as increased minute ventilation, increased inhalation of antigens and irritants, and drying and cooling of inspired air. These can trigger bronchospasm that can impede training. The clinician must provide considerable reassurance as complete symptom resolution may take 4 to 5 weeks.[37] Management relies on avoiding irritant stimuli and using bronchodilators such as albuterol (1 to 2 puffs q4–6h). Inhaled corticosteroids such as fluticasone (88 to 440 μ g BID), beclomethasone (2 to 4 puffs QID), flunisolide (2 to 4 puffs BID), or triamcinolone (2 to 4 puffs BID-QID) may also be useful.

Acute cough with a history of fever, sputum production, myalgias, pleuritic chest pain, and shortness of breath and physical findings that include hypoxia, tachypnea, and localized pulmonary rales or rhonchi suggest the diagnosis of community-acquired pneumonia. Pneumonia is the sixth leading cause of death in the United States and the leading cause of death among infectious diseases. Community-acquired pneumonia affects about 3 million persons in the United States per year, and about 1/6 of this group requires hospitalization.[41]

Chest x-rays often show localized or diffuse infiltrates. Early in the course of the illness, the chest x-ray may be normal as radiologic findings may lag behind clinical findings. Sputum Gram stain and culture may provide clues to the causative organism.[41]

In healthy subjects treatment can usually be done on an outpatient basis.[32] Proper rest, hydration, and nutrition are critical. Antibiotics should cover the common bacterial pathogens (*Streptococcus pneumoniae*, *Mycoplasma pneumoniae*, *Legionella pneumoniae*, *Chlamydia pneumoniae*, *Haemophilus influenzae*). First-line therapy includes azithromycin (500 mg for one day then 250 mg a day for 4 days) or clarithromycin (500 mg BID × 7 to 14 days). One may also consider a fluoroquinolone with increased *S. pneumoniae* activity such as levafloxacin (500 mg QD for 7 to 14 days), an oral second-generation cephalosporin such as cefuroxime (250 to 500 mg BID for 7 to 14 days), amoxicillin and clavulanate (875 mg BID for 7 to 14 days), or doxycycline (100 mg BID for 7 to 14 days).[40]

Pneumonia patients, by virtue of their damaged pulmonary parenchyma, will require more time to recover and return to full training. Absolute rest while the patient is symptomatic is critical to avoid prolonged illness, pulmonary abscess, and empyema. Absolute rest lasts at least several days but may last as long as several weeks. Chest x-rays should not be used to guide return-to-play decisions as findings may persist for up to 6 weeks.[31]

Sore Throat

Sore throat is another common complaint that affects athletes. Common infectious causes (Table 55.3) include viral URIs, group A beta-hemolytic streptococcal (GABHS) pharyngitis, infectious mononucleosis (IM), and enterovirus infections, such as coxsackievirus, which have been linked to infectious myocarditis.[42,43] History should target time of onset, ill contacts, time of day when the pain is the worst, presence of cough and/or fever, difficulty swallowing, and difficulty speaking. The exam should look for tonsillar erythema or exudates, asymmetric tonsillar swelling, ulcerations, palatal petichiae, fever, cervical adenopathy, and

TABLE 55.3
Symptoms and Signs Seen with Common Forms of Acute Pharyngitis

	Fever	Adenopathy	Splenomegaly	Rhinorrhea	Ulcerations	Exudate	Fatigue
Cold viruses	–	±	–	+	–	±	±
GABHS[a]	+	+[c]	–	–	–	+	±
IM[b]	+	+[d]	±	–	–	+	+
Enterovirus	±	±	–	–	+	–	±

[a] GABHS = group A beta-hemolytic strep.

[b] IM = infectious mononucleosis.

[c] More typically in the anterior cervical chains.

[d] More typically in posterior cervical chains.

Note: +, usually present; ±, variably present; –, not commonly present.

Source: Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, 357–370. With permission.

splenomegaly. The most common infectious causes of adult pharyngitis are cold viruses;[42] therefore, the treatment of choice in most cases is symptomatic. Warm saltwater gargles, humidified air, throat lozenges, and acetaminophen or ibuprofen are often effective.

To help diagnose strep throat, rapid strep tests should be done if available. If positive, the patient should be treated. If negative, then a throat culture should be done and the patient treated if positive.[42] The presence of a sore throat, fever, cervical adenopathy, and tonsillar exudates with the absence of cough suggests a greater than 50% likelihood of strep throat and warrants empiric treatment.[42] Penicillin (500 mg BID for 10 days) remains the drug of choice. Second-line choices include azithromycin (500 mg QD for 1 day, then 250 mg a day for 4 days) or erythromycin (250 mg QID for 10 days).[42] Antibiotics hasten recovery, render the patient noninfectious after 24 hours, and protect against the rare complication of rheumatic fever.[31] A patient who gets considerably worse despite treatment should be checked for peritonsillar or retropharyngeal abscess, as these demand urgent referral to an otolaryngologist.

Infectious mononucleosis, caused by the Epstein–Barr virus (EBV), occurs most commonly between ages 15 and 24 and affects 1 to 3% of college students each year.[44] Typical symptoms include a 3- to 5-day prodrome of headache, anorexia, and malaise, followed by sore throat, fever, lymphadenopathy, and fatigue that typically lasts about 2 weeks. Anorexia, nausea, fatigue, and malaise are often present and can limit an athlete's return to pre-illness training levels, which can take as long as 3 months.[44] Highly trained athletes are not more susceptible to IM.[45] Physical findings include fever, diffuse posterior lymphadenopathy, and tonsillar erythema and exudates. A morbiliform rash may occur in patients prescribed amoxicillin to treat a presumed strep throat, a finding that can aid in diagnosis.

Diagnostic study results include a lymphocytosis of >50%, >10% atypical lymphocytes on the peripheral smear, and a positive heterophil antibody (monospot) test. Ten percent of IM sufferers will have a negative monospot,[46] in which case EBV serology should be ordered. Approximately one quarter of affected individuals will have a concomitant GABHS pharyngitis, so testing and treatment should follow the above guidelines.[46]

Supportive care should be instituted with attention to rest in the early acute stages as well as oral hydration, saline gargles, and pain and fever relief with acetaminophen or ibuprofen. Aspirin should not be used due to the possibility of inducing Reye's syndrome. Treatment with antivirals such as Acyclovir® is not indicated.[46]

Up to 5% of athletes with IM may suffer one of many other complications affecting several organ systems (see reference 45 for a complete list). Severe tonsillar swelling is not uncommon but responds well to prednisone (40 to 60 mg QD for 5 to 10 days).[46] Patients with IM may develop aplastic or hemolytic anemia or thrombocytopenia, which may be detected with initial diagnostic testing. Also, clinical symptoms of hepatitis may develop which may prompt the physician to obtain liver enzymes. Any abnormalities on laboratory tests noted during the illness should return to normal prior to allowing the athlete to return to activity.[45]

The most feared complication of IM is splenic rupture, estimated to occur in 0.1 to 0.2% of all cases. Ruptures almost always occur between day 4 and 21 of the illness in patients with enlarged spleens, of which only 50% may be clinically palpable. A correlation does not appear to exist

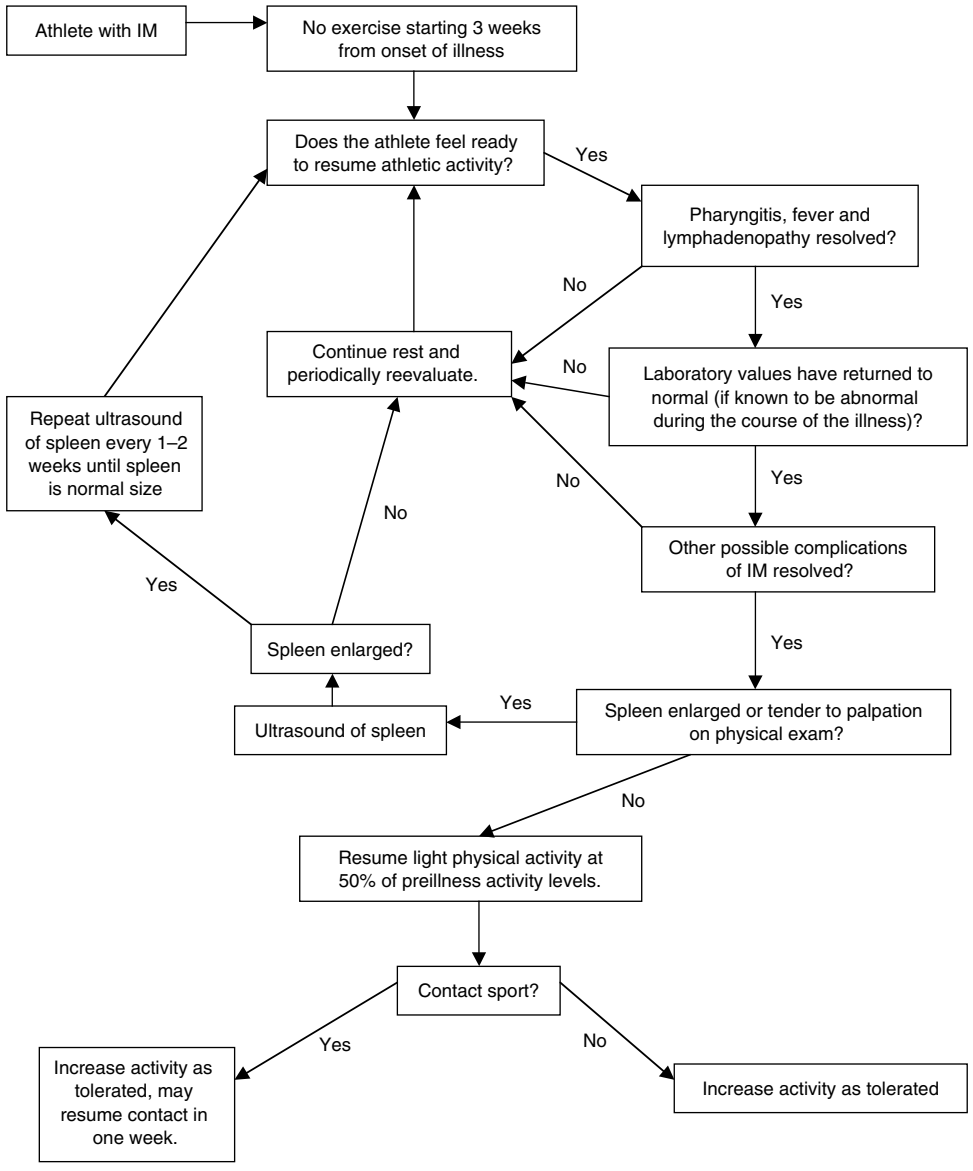

Figure 55.2 Algorithm for return-to-play guidelines for athletes with infectious mononucleosis. (From Metz, J.P., Upper respiratory infections: who plays, who sits?, *Curr. Conc. Sports Med.,* 2(2), 84–90, 2003. With permission.)

between the severity of the illness and the susceptibility to splenic rupture. Patients must be made aware that left upper quadrant pain that radiates to the left shoulder (Kehr's sign) demands immediate medical attention.[31] Some splenic ruptures occur in the absence of significant physical exertion, trauma, or strain and may be the presenting clinical feature.[44]

With the potential for splenic rupture in mind, the following guidelines (Figure 55.2) will help guide the return-to-play decisions for athletes with IM. Exercise for the first 21 days of the illness should be avoided. After that, noncontact exercise at 50% of pre-illness level may be initiated if the athlete feels ready; has no fever, pharyngitis, or lymphadenopathy; has normal labs (if labs obtained during the acute illness were abnormal); has resolution of other complications of IM; and does not have an enlarged or tender spleen. For noncontact athletes, activity can be increased as

TABLE 55.4
Symptoms and Signs Seen with Common Forms of Acute Diarrhea

	Fever	Abdominal Cramps	Weight Loss (chronic)	Foreign Travel	Ill Contacts	Antibiotic Use	Blood/mucous in Stools
Viral	–	±	–	–	+	–	–
Bacterial	+	+	±	+	–	–	+
Inflammatory bowel disease	+	+	+	–	–	–	+
Endocrine	–	–	+	–	–	–	–
C. difficile	+	+	–	–	–	+	+

Note: +, usually present; ±, variably present; –, not commonly present.

Source: Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, 357–370. With permission.

tolerated. Athletes in contact sports can resume contact at 1 month after the onset of symptoms if the spleen is not clinically enlarged or tender. Any athlete with an enlarged or tender spleen 4 weeks after illness onset should have an ultrasound measurement. The athlete may return to activity when the spleen has achieved normal size (Level of Evidence C, consensus/expert opinion).[44,45] Ultrasound measurement of the spleen to left kidney ratio[47] every two weeks can be useful, especially in athletes at the extremes of body habitus. A ratio of less than 1.25 is normal.

Acute Diarrhea

Diarrhea is defined as 3 or more loose stools a day for up to 7 days.[48] Acute diarrhea is most often due to viral infections, but other causes to consider include endocrine disorders such as hyperthyroidism, inflammatory bowel disease, bacterial colitis, and antibiotic-induced colitis. Infectious viral diarrhea is best managed by supportive measures and time. A detailed history can often narrow the cause to one of the above types. Helpful clues include travel history, hobbies, animal contacts, antibiotic usage, dietary habits, and ill contacts. The review of systems should cover stool appearance (mucous, bloody, or watery), fever, weight loss (acute and chronic), and abdominal pain.[48] In most cases, the morbidity from diarrhea is related to fluid losses and subsequent dehydration and electrolyte imbalances. Further dehydration from exercise plus enteric losses can thus increase the risk of adverse outcomes related to diarrhea.

Stool examination for fecal leukocytes, ova and parasites, and occult blood, as well as *Clostridium difficile* toxin assay in the patient with antecedent antibiotic use, are helpful in identifying the cause. Greater than 5 leukocytes per high-powered field strongly suggests the presence of a bacterial infection.[48] Clues to differentiating among types of acute diarrhea are shown in Table 55.4.

As mentioned earlier, treatment in most cases of acute diarrhea focuses on rehydration, which can usually be done orally but may need to be done intravenously in severe cases.[48] Antidiarrheal agents are generally not needed, but if they are needed then one can use the antisecretory agent bismuth subsalicylate (262 mg, 2 QID p.r.n.), or the antimotility agent loperamide (4 mg after the first loose stool, then 2 mg after each subsequent loose stool to a maximum of 16 mg a day). Loperamide should be avoided in patients who appear toxic, who are febrile, or who are having bloody diarrhea. Lomotil, which contains atropine, is not a good choice due to its anticholinergic side effects.[49]

The decision to use antibiotics empirically remains in the hands of the provider. Bacterial colitis (*Salmonella*, *Shigella*, *Escherichia coli*, *Campylobacter*) can be treated with ciprofloxacin (500 mg BID) or trimethoprim–sulfamethoxazole DS (1 pill BID for 3 to 5 days). *Salmonella* should not be treated unless the illness is severe or the patient is immunocompromised, as antibiotic treatment may prolong the carrier state. *C. difficile* is treated with metronidazole (500 mg TID) or vancomycin (125 mg orally QID for 10 to 14 days).[40]

HIV Infection

The athlete with human immunodeficiency virus (HIV) infection raises two important questions: Is exercise good for the athlete, and does the athlete's participation put other athletes at risk? In HIV-positive patients, moderate and high-intensity aerobic exercise programs do not change leukocyte, lymphocyte, CD4, and CD8 counts or

the CD4:CD8 ratio from baseline, but cardiovascular fitness improves.[50] Progressive resistance training increases lean body mass and physical functioning in patients with HIV-associated wasting.[51] Regular exercise is also associated with slower progression to acquired immune deficiency syndrome (AIDS) and decreased short-term risk of death from AIDS.[52] In general, regular exercise does not increase risk of immunosuppression, infection, or death. HIV-positive patients can and should be encouraged to exercise to improve functional capacity and quality of life. Patients with a CD4 count of <200 or an AIDS-defining infection, however, should be limited to moderate exercise and discouraged from heavy exercise.[53]

If HIV-positive individuals can and should exercise, can they do so without putting anyone else at increased risk for infection? Documented sports transmission of HIV is exceedingly rare. Extrapolation of HIV epidemiology and transmission data led to an estimate that the risk of HIV transmission in professional football was 1 in 85 million game contacts.[54] The American Medical Society for Sports Medicine (AMSSM) and the American Academy of Sports Medicine (AASM), in a 1995 joint statement, stated that mandatory HIV testing should not be a requirement for competitive sports participation, although they strongly encourage counseling and voluntary testing in individuals at high risk.[55] The National Collegiate Athletic Association (NCAA) mandates removal of bloody uniforms and covering open wounds prior to returning to competition. Universal precautions are the norm in dealing with any blood or body fluid.[54]

RETURN TO PLAY

When an athlete with an infection can return to play can be difficult to answer and requires weighing concerns about deconditioning and missing competition vs. worsening or prolonging an illness. An important question to consider is will exercise during an illness affect athletic performance or illness duration and severity? Experimental rhinovirus infection does not decrease pulmonary function tests, VO_{2max}, or submaximal exercise testing.[56] Exercise during such an infection does not alter its course or severity, either.[57] Other respiratory viruses, such as influenza virus,[58,59] have been shown to impair pulmonary function, so the results of the above study cannot be generalized to all URIs.

The above rhinovirus studies support the "neck check" method of determining return to play, as described by Eichner[3] and Primos.[60] If symptoms are above the neck (e.g., runny nose, nasal congestion, sore throat, or sneezing) and not associated with below-the-neck symptoms (e.g., fever, myalgias, arthralgias, severe cough, gastrointestinal symptoms), then the athlete may train at half intensity for 10 minutes. If the symptoms do not worsen, then the workout may be continued as tolerated. If the symptoms worsen in the initial 10-minute period, the workout should end and the athlete should rest until symptoms improve. Exercise should be delayed until all below-the-neck symptoms have resolved.[3] When resuming training after recovering from an illness, the runner should start at a 50% intensity and gradually increase to pre-illness training levels over 1 to 2 days for every training day missed (Level of Evidence C, consensus/expert opinion).[60]

Training delay serves three purposes. First, training with below-the-neck symptoms hampers the workout and limits desired training effects. Second, without a medical evaluation an athlete may not know the potential severity of his or her illness.[3] Third, not training may prevent the spread of disease to other athletes[60] as demonstrated by the documented transmission of Norwalk virus during a college football game.[61]

TABLE 55.5
The Athlete Who Fails to Recover

Overtraining
Thyroid disorders
Human immunodeficiency virus (HIV)
Hepatic disease
Depression
Eating disorders
Diabetes
Cancer
Pregnancy
Malnutrition/malabsorption
Anemia
Autoimmune disorders
Alcohol/drug abuse
Parasitic/spirochete infections
Myocarditis
Asthma

Source: Metz, J.P., Deitche, W.S., and Howard, T.M., Infectious disease in the runner, in *Textbook of Running Medicine,* 1st ed., O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, 357–370. With permission.

Finally, the physician should be wary of the athlete that does not recover from illness. The differential diagnosis (Table 55.5) of these athletes is long and should be considered in those individuals who do not recover in a reasonable period of time.

CONCLUSION

Moderate exercise seems to be an immune stimulant while intense exercise may cause immune suppression. No reliable immune markers, however, are available to help the physician make return-to-play decisions. When, despite their best efforts, athletes become ill it is important to properly care for them to minimize lost training time and maximize recovery. Athletes with HIV can safely exercise without fear of affecting their own illness or increasing the risk of transmission to other athletes during competition. Correct diagnosis, treatment, and slowing or suspending training according to the "neck check" protocol are critical to facilitating rapid return to full training.

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56 DERMATOLOGICAL DISORDERS

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INTRODUCTION

The skin serves as a protective barrier against mechanical, environmental and infective forces. The athlete may experience delays in training, a decrement in performance, or an inability to compete once the skin has been injured. Common dermatological conditions affect athletes of all ages and competitive levels, but sport-specific dermatoses may incapacitate an athlete or expose teammates to a potential infection placing them at risk for disqualification. The athletic healthcare team must promptly recognize and accurately assess the skin during training and prior to competition to formulate a definitive treatment plan.

MECHANICAL INJURY

Abrasions

Abrasions, commonly known as rug burn, strawberries, or road rash, occur on artificial turf, floor mats, synthetic courts, and asphalt roads. Treatment of superficial abrasions consists of cleaning and debriding the tissue with warm, soapy water and applying a topical antibacterial ointment. Denuded skin and foreign debris should be removed from the wound to promote healing and prevent tattooing of the skin. Abrasive wounds will heal rapidly when kept moist with either a thin covering of antibacterial ointment (mupirocin) or an adhesive dressing (e.g., DuoDerm®, Op-Site®). Depending on the extent of skin damage, wound coverage may only require a simple occlusive dressing over the abrasion. Due to the risk of bloodborne pathogens and subsequent disease transmission, all wounds should be covered with an occlusive dressing during participation. The NCAA mandates that an athlete be removed from competition if active bleeding exists and that universal precautions should be practiced by the healthcare team.[1] The athlete may resume competition after medical personnel have entirely stopped the bleeding and an appropriate dressing has been applied to withstand the rigors of competition.[1]

Blisters

Blisters are vesicles or bulla filled with either serosanguinous fluid or blood. They result from shearing forces across the epidermal layer of skin. Repeated pressure or friction over boney prominences associated with excessive perspiration and improperly fitted equipment leads to the formation of blisters. Small blisters and hot spots can be treated early with moleskin donuts and nylon foot stockings to decrease friction, talcum powder to absorb perspiration, and benzoin to harden the epidermis.[2] If the blisters are bullous and uncomfortable, they should be drained at the edge with a small needle, leaving the roof of the blister as a protective layer. Ruptured and deroofed blisters may require the application of a hydrocolloidal dressing (DuoDerm®) or an adhesive polyurethane dressing (Op-Site®) as a second skin layer to reduce discomfort and enhance healing.[3] Always emphasize to athletes the importance of primary prevention by wearing properly fitted and broken-in footwear, wearing absorbent socks, and applying petrolatum jelly over boney prominences.

Chafing

Chafing is the effect of repeated irritation to the epidermis from continual rubbing of synthetic fabrics across skin folds. The abraded, red skin is usually found in the inguinal or axillary folds due to the inappropriate use of tight-fitting sports apparel with an elasticized inner undergarment. Chafing can occur from repeated exposure to extreme environmental conditions, leading to a hardened, kornified layer of skin. Treatment involves moisturization of the keratinized skin with lotions (Lac-Hydrin®, Keri® Lotion, or Aquaphor®) and utilizing appropriate cotton clothing.

Corns and Calluses

Corns are small soft or hard, deep painful conical lesions with a translucent central core. Soft corns

usually appear in the web spaces of the toes and hard corns over the plantar surface of a malaligned distal metatarsal head. Calluses tend to be larger, hyperkeratotic non-painful lesions that serve as a protective skin layer and are considered an advantage in gymnasts, racquet sports, golfing and rowing. Each is a result of chronic friction or pressure over a bony prominence of the foot or hand and must be differentiated from plantar warts. Calluses can be distinguished from plantar warts by scraping the base of the lesion with a scalpel blade. The development of small black dots representing thrombosed capillaries implies the presence of plantar warts, compared to calluses that display a thickened epidermis with intact dermatoglyphics. After careful evaluation of foot biomechanics for rearfoot varus and a rigid first ray, the most important factor for successful recovery and prevention of the condition is redistributing the source of pressure.[4] The shaping of a metatarsal pad to the plantar surface, creating a wider shoe toebox, adding cotton or foam padding between the toes, and applying moleskin will all aid in decreasing the pressure over the existing lesion and prevent further injury. Palliative treatment options for corns and calluses include shaving with a scalpel blade after soaking the lesion in warm water. To eliminate the lesions, keratolytic agents such as 5 to 10% salicylic acid in collodian, 40% salicylic acid plaster, and 12% lactic acid are carefully applied at night in such a way as not to affect the surrounding skin.[5]

Black Heel

Black heel, or *talor noir*, refers to a bluish-black plaque formation of horizontally arranged dots or calcaneal petechiae within the stratum corneum on the posterior or posterolateral aspect of the heel.[6] Repetitive rapid impact and changes in direction lead to a shearing of intraepithelial capillaries on the heel. The condition occurs more frequently in adolescent and younger adult athletes playing basketball and tennis and competing in track and field events. A similar condition, mogul's palm, has been described on the hypothenar eminence of mogul skiers' palms due to the skiers constantly planting their poles and shifting direction.[7] Each of these two conditions is self-limiting and will resolve spontaneously once the season ends. Although asymptomatic, the dark discoloration may resemble melanoma. Gently paring the skin and immersing the scrapings in water can confirm the presence of the condition with a positive occult blood-screening test.[8] The use of heel cups, felt pads, cushioned athletic socks, and properly fitted footwear may help to prevent black heel formation.

Black Toenail

Rapid deceleration of the forefoot against the shoe toebox may produce subungual hemorrhages of the first and second toe nailbeds. Often referred to as tennis toe, jogger's toe, or skier's toe, the great toenail becomes dystrophic after repeated impact to the shoe toe box. The condition occurs with greater frequency in sports requiring quick stops such as tennis, skiing, hiking, and rock climbing.[8] Toenail pressure from improperly fitted shoes or boots and frequent dorsiflexion of the toes will predispose the athlete to subungual hemorrhages. The hematomas usually resolve spontaneously and do not require further treatment. If acute pain from elevation of the nail from the nailbed develops, then the hematoma can be drained by carefully boring a hole through the nail with an 18-gauge needle or electrocautery unit. To avoid this condition, footwear must be appropriately fitted to allow for adequate toe space (2 cm from the longest toe to the end of the shoe).[9] Appropriate running shoes and properly trimming the distal nail to its shortest length in a straight-cut line will reduce the likelihood of developing this condition.[10] Notable exceptions are the persistence of a linear black band or streak running the entire length of the nail representing a melanocytic nevus or the more serious involvement of the proximal nail fold in a malignant melanoma.[11] These conditions should be referred to a dermatologist.

Ingrown Toenail

Ingrown toenails commonly occur at the margins of the great toenail. The condition is due to nailbed pressure forcing the lateral edge of the nailplate into the lateral nail fold. The condition arises from not properly trimming the toenails or from inadequate toe space in improperly fitted shoes. The distal nail should be trimmed straight across and at least one thumbnail in distance should exist from the longest toenail to the end of the shoe to prevent reoccurrences. Acute treatment options include soaking the toes in an Epsom saltwater bath, gentle manual nail elevation, wearing shoes with a larger toebox, and placing a small piece of cotton under the corner of the nail to elevate the lateral margin to alleviate inflammation.[9] The toenail may require oral antibiotics to prevent secondary infection. If these measures are ineffective, the affected lateral one third of the nail may be removed after digital anesthesia.

Jogger's Nipples

Constant irritation and friction between coarse, cotton fabrics and the unprotected nipple and areola lead to painful, bleeding nipples.[12] The majority of jogger's nipples occur in male athletes, especially long-distance runners and triathletes.[13] Preventive measures include wearing soft, natural, silk-fiber shirts and the application of breast padding, electrocardiographic lead pads, adhesive bandages, or a double coat of fingernail polish over the nipples prior to running. Symptomatic treatment with soothing topical creams decreases the pain and promotes healing.

Runner's Rump

Runner's rump refers to a collection of ecchymotic lesions on the superior portion of the gluteal cleft of long-distance runners.[13] It is thought to result from constant friction between the gluteal folds with each running stride. The hyperpigmentation will spontaneously resolve with rest.

Rower's Rump

Rower's rump develops in the gluteal cleft of rowers training on small scull seats and metal rowing machines.[14] Repeated friction with unpadded seats produces a lichen simplex chronicus of the buttocks. Treatment consists of padding the rowing seat and the use of potent, fluorinated topical steroids. A properly fitted and padded seat will help prevent the plaques from forming.[14]

Acne Mechanica

Acne mechanica is an occlusive obstruction of the follicular pilosebaceous units usually seen in football and hockey players.[15] The condition occurs during the season while the athlete is wearing equipment over the bony prominences of the head. The papulopustular eruption repeatedly arises where friction and perspiration develop under padded garments, headgear, and chin-straps. The commonly affected areas include the forehead, cheek, and chin. Occasionally, pads will produce the same condition over the shoulders, back, and hips. Preventive measures include wearing a clean, cotton T-shirt against the skin to absorb the perspiration and prevent follicular occlusion.[13] Fitting a piece of soft fabric between the equipment and skin will reduce both friction and heat. The equipment should be routinely cleaned with soap and water or an alcohol solution to prevent bacterial formation. Acne mechanica in dark-skinned athletes may evolve into acne keloidalis on the nape of the neck.[8] The athlete can treat the condition with various topical acne keratolytics with astringents (3% salicylic acid, 70% resorcinol) and antibiotics (tetracycline, clindamycin).[13] Prior to the use of isotretinoin for severe pustular acne, athletes should be well informed and educated about the possible development of side effects such as muscle soreness, joint pain, and lethargy.[13]

Follicular Keloiditis

Keloids are the inflammatory proliferation of fibrous tissue. The condition is usually painless, is more prevalent in dark-skinned African athletes, and resolves at the end of the season. Multiple, small keloids commonly develop where the headgear comes in contact with the forehead, cheeks, and posterior neck or where the undergarment pads cover the thighs, knees, and shoulders.[16] The disfiguring appearance can hinder athletic performance depending upon the site. Treatment at the end of the season involves gradual reduction of the lesion with intralesional injections of steroids or topical application of a steroid-impregnated adhesive tape.[17] Surgery can often lead to even larger keloid formation.

Piezogenic Papules

Piezogenic papules represent herniations of subdermal fat into the dermis on the lateral or medial surface of the heel.[18] They are flesh-colored papules noticeable only upon weight-bearing. The pain associated with the papules is thought to represent ischemia of the herniated fatty tissue caused by compressive compromise of the blood supply.[19] These lesions may be found in up to 20% of the general population upon standing.[17] The papules are more common in endurance athletes, especially long-distance runners, and may be so painful as to preclude further training and racing. Padding, taping for support, and/or heel cups may help reduce the pain.

Athletic Nodules

Sports-specific nodules are fibrotic, connective tissue (collagenomas) found on various skin locations due to repetitive pressure, friction, and trauma.[20] These asymptomatic nodules usually represent a badge of distinction to the sport and convey a sense of accomplishment.[13] Multiple sports have described distinct locations for nodules. Surfer nodules, ulcers, and knots located on the dorsal aspect of feet and infrapatella bursa have decreased in the last several years due to wearing neoprene wetsuits, using proper paddling techniques, and using improved surfboard wax.[21] Boxers and football players develop nodules over

their knuckles and hockey players develop nodules over the dorsum of the feet known as "skate bites."[8] Runners and hikers will also develop nodules over the dorsum of their feet due to increased pressure from tightening the shoelaces too tight. Each of these nodules will resolve upon discontinuing the activity or diminish after the season. Treatment options include intralesional steroids to reduce the size of the nodule and surgical excision.[20]

ENVIRONMENTAL INJURY

Sunburn

Acute and chronic skin damage from sun exposure is the most preventable dermatitis. Athletic participation in water sports, snow skiing, and high-altitude endeavors are more prone to sunburn due to the intensity and reflectance of ultraviolet B (UVB) light.[16] Exposure to UVB light (290 to 320 nm) during the hours of 10 a.m. and 2 p.m. for 2 to 6 hours will produce mild erythema to intense blistering, edema, and pain.[5] A rise in altitude from sea level to 5000 feet intensifies sunlight by 20%.[2] Although the ultraviolet A (UVA) light range of 320 to 400 nm is 1000-fold less burning to the skin than UVB, UVA is more penetrating and produces chronic damage to the skin.[4] The majority of premalignant and malignant neoplasms occur in fair-skin athletes with repeated sun exposure early in childhood.[10] Preventive measures include avoiding exercise between 10 a.m. and 2 p.m., applying sun protection factor (SPF) 15 or greater sunscreens with *para*-aminobenzoic acid ester (PABA) at least 20 minutes prior to sun exposure, and recoating after several hours or after water exposure.[2] Whenever possible, athletes should protect their head with a broad-brim hat, wear white clothing over their torso and arms, and consider applying zinc ozide to the nose, lips, and ears.[4] Symptomatic treatment options include cool compresses, topical lotions with vitamin E, aspirin, oral nonsteroidal anti-inflammatory agents (NSAIDs) and low-potency, topical corticosteroids to reduce the inflammation. All athletes should avoid further skin exposure to allow for adequate healing. More severe forms of sunburn may require tepid showers, oatmeal baths, hydrocolloid dressings, silver sulfadiazine, oral corticosteroids for 5 days, and pain medication.

Miliaria

Miliaria rubra, or prickly heat, occurs in athletes, particularly football players, participating in the hot, humid summer environment.[3] The eccrine sweat ducts become occluded and rupture into the surrounding tissue. A fine, diffuse erythematous vesiculopapular rash develops over the epidermis, producing a prickly skin sensation. Sparing of the palms and soles due to the lack of eccrine sweat glands is helpful diagnostically.[22] Symptomatic relief involves limiting further activity and moving the athlete to a cool, dry area to minimize further perspiration. The application of hydrophilic ointments (Eucerin®) and mild topical corticosteroids can help open occluded ducts.[3] Participation may have to be reduced for several weeks to allow for the body's homeostatic mechanism to gradually acclimatize and reduce the risk of subsequent heat injury.

Solar Urticaria

Solar urticaria is an uncommon cause of urticaria in athletes.[5] The reaction is not due to a delayed photosensitivity reaction to an antigen as polymorphous light eruption, but manifests within minutes after exposure to UVA and UVB wavelengths.[23] Initially itching and burning occur, followed by erythema and wheal formation that clears within 1 hour after exposure.[8] Normally unexposed skin areas of the trunk will be more prone to develop an urticarial reaction than the previously exposed face or distal extremities. Phototesting is recommended to determine the type and treatment of solar urticaria. Desensitization and psoralen, long-wave ultraviolet light (PUVA) have been successful in minimizing symptoms.[24] The use of antihistamines has not been found to be effective. Oral betacarotene, topical sunscreens, antimalarials, and gradual tanning while not training have been found effective in reducing symptoms.[23]

Cholinergic Urticaria

Cholinergic urticaria is an acetylcholine-mediated dermatosis that occurs commonly during exercise or is related to emotional stress.[25] The condition is characterized by the eruption of pinpoint papular wheals with surrounding subcutaneous erythematous flares during and after heat exposure or exercise. The extremely pruritic condition is often found on the chest and back of the athlete. The most reliable and safe test to determine the severity of the urticaria is have the athlete perform exercise for 15 minutes on a treadmill or bike to reproduce the condition. Treatment with H1-antihistamines and danazol has been found to be effective if taken 1 hour prior to exercise.[26] A hot shower the night before may deplete histamine and provide a refractory

period for the athlete to compete.[27] The condition can be exacerbated with the use of aspirin and is often resistant to therapy. The best treatment is to avoid the usual triggers and consider attempting exercise in a climatized environment.

Cold Urticaria

Cold urticaria is rare but is the most common form of acquired physical urticaria in winter athletics and cold water swimming.[23] Wheals or hives are usually confined to the exposed area. Athletes who demonstrate cold urticaria are likely to have recurrent, severe episodes during similar circumstances due to the antigen–antibody reaction resulting in the release of histamine.[28] To confirm the diagnosis, provocative testing of the forearm with ice cubes for 5 minutes or submerging the forearm in cold water for 5 to 15 minutes will produce wheals.[8] Preventive measures include avoiding prolonged exposure to cold conditions, wearing protective clothing, the use of cyproheptadine (Periactin®), 2 mg once or twice a day, or doxepin (Sinequan®), 10 mg two or three times daily.[9]

Chilblain

Chilblain or pernio is the mildest form of cold injury and develops on the feet, hands and face. It has been described in children who wear unventilated, plastic boots with inadequate boot lining to absorb perspiration.[28] Athletes participating in winter sports are initially unaware of the injury but later complain of reddish-blue patches that burn and itch. The exposed skin is cool to the touch due to vasoconstriction and may later develop blisters.[5] The injured area should be rewarmed, massaged gently to increase circulation, and protected from further environmental exposure. Topical corticosteroids or a short burst of oral corticosteroids may be utilized to minimize the painful, inflammatory skin lesions. The key to preventing this condition is the use of properly insulated and appropriately fitted footwear and gloves. The use of moisture-wicking socks and gloves and frequent sock changes will aid in preventing this injury.

Frostnip

Frostnip or superficial frostbite occurs as temperatures drop below 50°F (10°C) with a significant windchill factor.[9] The skin and superficial subcutaneous tissue of the fingertips, toes, nose, cheeks, and ears will blanche or turn a grayish-white, develop paresthesias, and finally lose sensation. Penile frostnip has been reported in a jogger wearing polyester trousers and cotton undershorts.[29] Frostnip can be reversed with immediate self-rewarming to return blood flow and sensation to the exposed area. Blisters may form in 1 to 3 days after treatment.[16] Paresthesias and a burning sensation may persist for several months after the injury. Prevention is adequate insulation and skin protection from both wind and cold weather by not shaving prior to participation to preserve the natural skin oils and application of a sunscreen cream.[28]

Frostbite

Frostbite occurs as living tissue ceases cellular metabolism from exposure to temperatures below 28°F (–2°C).[28] The degree of frostbite clearly depends on multiple factors, including environmental conditions (cold, wind, altitude), skin perspiration, depth of tissue involvement and the potential for repeat exposure. Muscles, nerves, and blood vessels are damaged earlier than connective tissue, tendons, and bone.[28] The tissue appears cold, white, and hard and will not exhibit pain or sensation to tactile stimulation until thawing occurs. Rewarming of the tissue should be attempted only when the environment can be controlled and risk of refreezing has been eliminated. Until this is possible, the affected area should be padded to prevent further mechanical trauma. Frozen areas should be rewarmed as rapidly as possible in a warm-water, circulating bath of 110 to 112°F (38 to 44°C) to prevent mechanical trauma.[28] Analgesic pain medication is needed during the thawing phase while sensation to pain returns. After the tissue is rewarmed, the affected area will exhibit swelling, blistering, and pain for weeks. Tissue necrosis may occur for weeks to months as reepithelialization replaces the denuded areas.[2] In all but the most superficial cases, patients need to be evaluated by a surgeon experienced in tissue debridement for frostbite. Prevention includes wearing multiple layers of lightweight winter clothing and the application of petrolatum or zinc oxide paste to exposed skin surfaces.[28]

INFECTIOUS INJURY

Folliculitis

Folliculitis is a common pustular infection of the hair follicle in athletes due to occlusive padding, synthetic tight-fitting garments, and sharing whirlpools for rehabilitation. Multiple, pinpoint, nontender, pruritic, erythematous pustules develop at the hair shafts. The most common folliculitis is produced by *Staphylococcus*. A follicular pustular

dermatitis known as keratosis pilaris may develop over the anterior thighs or lateral upper arms from the rubbing of pads and stretch garments. Another entity, "hot tub" folliculitis, has been well documented with athletes using whirlpool baths for rehabilitation.[30] A pruritic, papulopustular rash with vesicles develops in 48 hours, usually over the skin covered by the bathing suit. The dermatitis is caused by *Pseudomonas aeruginosa*. The disease is self-limiting and will resolve in 7 to 10 days. For athletes requiring treatment, ciprofloxacin, 250 mg twice a day, is recommended.[10] Routine cleaning of the equipment and maintaining proper pH and chlorination of the water are necessary preventive measures to suppress the bacteria.[30]

Furunculosis

Furuncles are erythematous, nodular abscesses found in the hairy areas of the axilla, torso, buttocks, and groin where friction and perspiration are common. Furuncles are highly contagious, and known outbreaks have occurred in team sports, implicating close contact, prior skin injury, and poor hygiene practices.[31] Staphyloccocci are the most common pathogen. The anterior nares should be cultured due to the predisposition of the nares to harbor the *Staphylococcus* species.[10] Acute treatment consists of warm compresses and a 10-day course of a cephalosporin, erythromycin, or a penicillinase-resistant penicillin derivative.[32] A prophylactic dose of rifampin, 600 mg for 7 to 10 days, has been utilized in resistant cases.[9] Bacitracin or mupirocin ointment can be applied to both the involved tissue and nares to prevent further transmission. To prevent autoinnoculation, the area should be covered with a sterile dressing. When either a furuncle or a carbuncle becomes fluctuant, incision and drainage are necessary due to the poor hematogenous tissue penetration of the antibiotic into the fluctuant area. Proper showering and washing with antibacterial soaps are imperative preventive measures. The NCAA requires all wrestlers to be without new lesions for 48 hours before a meet, to have completed 72 hours of antibiotic therapy, and to have no moist or draining lesions prior to competition.[1] Athletes should not be allowed to participate in contact sports and swimming if lesions exist.[33]

Impetigo

Impetigo is a bacterial infection characterized by serosanguinous, honey-crusted pustules on an erythematous base. Beta-hemolytic streptococci more commonly produce impetigo, but staphylococcal species have been isolated from cultured wounds. The lesions are commonly found on the face but can occur over the extremities. The diagnosis is confirmed by culture. If streptococcal species are isolated, the healthcare provider needs to be aware of the possibility of poststreptococcal glomerulonephritis. Athletes participating in contact sports or swimming are highly contagious and should not participate in competition until all lesions have resolved.[3] Less extensive lesions may respond to twice-daily applications of mupirocin ointment with aluminum acetate compresses (Burow's solution) in a 1:40 dilution three times daily.[32] A 10-day course of an oral cephalosporin or penicillinase-resistant penicillin may be appropriate for more extensive lesions to promote rapid healing. The NCAA guidelines for participation of wrestlers, as previously described for furunculosis, pertain to all bacterial infections.[1]

Erythrasma

Erythrasma is a chronic, bacterial infection affecting the intertriginous areas of the inguinal folds and axilla. The causative organism is *Corynebacterium minutissimum*. Predisposing factors include occlusion or maceration of the skin in a humid, warm environment. The lesions can last for years and are often asymptomatic. The sharply demarcated, reddish-brown plaques are similar in appearance to tinea cruris.[33] Under a Wood's lamp (black light) the lesions will fluoresce coral-red, while tinea cruris does not fluoresce.[3] Gram stain reveals Gram-positive rods. Treatment options include a topical erythromycin cream or gel and oral erythromycin, 250 mg four times a day for 14 days. The areas need to be covered for athletes to participate in close-contact drills or events.

Pitted Keratolysis

Pitted keratolysis occurs primarily in basketball and tennis players on the keratinized sole of the foot.[25] A scalloped-bordered plaque with sculpted pits of variable depth forms on the weight-bearing plantar surfaces (heel and toes). Hyperhidrosis and Gram-positive bacteria, most commonly *Corynebacterium* and *Micrococcus* species, found in the stratum corneum have been implicated in producing the pungent foot odor.[34] The condition is often misdiagnosed as tinea pedis. Application of topical antibiotics for 2 to 4 weeks (5% erythromycin in 10% benzoyl peroxide) will reduce the bacterial inflammatory component and result in clearing.[5] Prophylactic therapy includes washing with benzoyl peroxide soap and adding topical foot powders with 20% aluminum chloride

(Drysol®) to control hyperhidrosis.[34] Proper hygiene measures and frequent change of absorbent socks will improve the condition.

Otitis Externa

Acute otitis externa, or "swimmer's ear," is a bacterial infection more common to aquatic activities.[35] The otic canal becomes pruritic, red, edematous, and painful and exhibits an intermittent purulent discharge and loss of hearing. A gentle tug on the pinna can reproduce the painful symptoms leading to the diagnosis. Continual exposure of water to the external canal removes the protective acid pH bacteriostatic seal created by the cerumen.[5] The most common pathogens are Gram-negative bacteria, particularly *Pseudomonas*.[36] Treatment involves gentle removal of canal debris and keeping the canal open and dry with astringent agents (Burow's solution) or a wick.[37] If the tympanic membrane cannot be visualized on exam or is possibly perforated, antibiotic suspensions are required for therapy. A local anesthetic (Auralgan®) can be utilized if the eardrum is not perforated. Common antibiotic regimens include fluoroquinolone/steroid or polymixin/neomycin/steroid drops (Cortisporin®) instilled in the ear for 7 to 10 days.[2] Oral antibiotics are recommended in more severe infections. A 2% acetic acid solution in propylene glycol (Vosol®) is particularly effective in preventing recurrent infections.[35] Earplugs may be harboring the infection, and all devices must be replaced or routinely sterilized. Activity may need to be suspended for 5 to 10 days while the infection is resolving.

Onychomycosis

Onychomycosis is a common fungal infection known as tinea unguium and can be attributed to either of two dermataphytes, trichophyton rubrum or trytophytum mentagrophytes, in 80% of cases.[38] Candida albicans and various molds can also contribute to onychomycoses. The toenail displays an irregularly, thickened nail matrix with discoloration. Occlusive footwear and communal showers in locker rooms harbor the fungi. Given the difficulty in attaining adequate penetration of medications into the nail and nail bed, topical antifungals have been ineffective. Oral griseofulvin has been replaced by itraconazole (Sporanox®) and terbinafine (Lamisil®) as the therapeutic agents of choice for both toenail and fingernail therapy in adults.[39] Itraconazole can be given in either a continuous dose of 200 mg daily for 12 weeks for toenails and 6 weeks for fingernails or a pulsed dose of 400 mg daily for the first full week of 3 to 4 successive months for toenails or 2 successive months for fingernails.[38,39] The second agent, terbinafine (250 mg), is taken daily for 12 weeks for toenails and 6 weeks for fingernails.[38,39] Oral antifungal medications are expensive and have reported side effects, including hepatotoxicity and pancytopenia. Laboratory analysis performed prior to and during therapy is required. Recently, ciclopirox nail lacquer topical solution 8% has been found to be efficacious in the treatment of mild to moderate onychomycosis and is a therapeutic option for patients who cannot tolerate oral agents or in whom oral agents have failed.[40] Preventive measures include proper change of absorbent socks, wearing ventilated footwear, wearing shower shoes, and the use of antifungal powders.

Tinea Corporis

Tinea corporis is often referred to as "ringworm" for its characteristic annular lesion, which has a sharply demarcated, reddened border with central clearing. The dermatophyte infection is limited to the torso, extremities, and face. Each lesion will expand and produce concentric rings along its annular margins. The lesions develop a mild pruritus and are diagnosed by demonstrating hyphal elements either on a KOH slide preparation or in a fungal culture. A subset of tinea corporis, tinea corporis gladiatorum, occurs frequently in wrestlers on their head, neck, and upper arms.[41] In the majority of cases, *Trichophyton tonsurans* is the causative fungus.[41] Recent studies found that oral fluconazole (200 mg, taken once weekly for 4 weeks) resulted in negative cultures after 7 days in 60% of the wrestlers.[41] Terbinafine cream has been shown to have favorable results in children.[41] The NCAA requires a minimum of 72 hours of topical terbinafine or naftifine applied to skin lesions, a minimum of 2 weeks of oral therapy for scalp lesions, and all lesions to be adequately covered with an antifungal cream, gas-permeable dressing, and ProWrap® with stretch tape prior to wrestling.[1] Regular skin inspections by the athletes and coaches will improve awareness and prevent a potential epidemic.

Tinea Cruris

Tinea cruris is an erythematous, pruritic plaque with well-demarcated, scaly borders that may extend to the groin, upper thighs, abdomen, and perineum.[42] The dermatophytic infection is commonly referred to as "jock itch." Maceration of the stratum corneum created by persistent perspiration and irritation in

the inguinal folds allows for penetration of the fungal hyphae. The scrotum is commonly spared due to the fungistatic sebum produced by the scrotal skin.[16] The appearance of an inflammatory, red rash with satellite lesions involving the scrotum is candidiasis and requires treatment with imadazole (clotrimazole, miconazole, ketoconazole) creams. Diagnosis can be confirmed by the presence of hyphae on a KOH slide preparation. Topical antifungal creams applied at least twice a day for 2 to 4 weeks provide adequate therapy. Oral antifungal agents may be required in recalcitrant cases if the hair roots are involved. Tinea cruris must also be differentiated from candida intertrigo (scrotal involvement, satellite lesions), erythrasma (brown and scaly, fluoresces coral red), psoriasis (silvery scale, pitted nails, scalp lesions), folliculitis (punctate pustules), or a chronic irritant dermatitis from elasticized undergarments.[43] Allowing the skin to air dry, wearing loose-fitting cotton undergarments, and the application of drying agents (Domeboro®) with mild topical corticosteroids can decrease the inflammatory process.

Tinea Pedis

Tinea pedis, or "athlete's foot," is a papulosquamous fungal infection found on the lateral soles of the feet and between the toes. The superficial dermatophytic fungal infection is caused by *Trichophyton rubrum*, *Trichophyton mentagrophytes*, or *Epidermophyton floccusum*.[36] The skin is pruritic, red, and scaly with peeling and fissuring of the toe webs. A vesiculubullous form and a moccasin-like pattern have been described on the plantar surface of the feet.[44] Hyphal elements on a KOH slide preparation or in a fungal culture confirm the diagnosis. The majority of cases respond promptly to topical antifungal creams, such as miconazole, clotrimazole, and econazole. Nail involvement, onychomycosis, is often the source in recalcitrant cases and will require oral terbinafine or itraconazole. Crusting, erythema, and serosanguinous exudates involving the feet and web spaces may indicate a secondary Gram-negative or Gram-positive infection requiring antibiotic coverage.[43] Frequent sock changes, use of shower shoes in locker rooms, and applying over-the-counter antifungal powders are effective measures to decrease the reoccurrence of infection.

Tinea Versicolor

Tinea versicolor is a chronic, asymptomatic scaling dermatosis associated with overgrowth of the active fungal form of *Pityrosporum orbiculare* known as *Malassezia furfur*.[5] Various pigmented macules commonly occur on the chest and back but occasionally involve the lateral neck, upper extremities, and abdomen. Wood's lamp reveals a yellow-green fluorescence of the skin scales.[4] Tinea versicolor is treated with topical 2.5% selenium sulfide shampoo (Selsun®) for 15 to 30 minutes for 5 to 10 days or by applying the lotion overnight (6 to 12 hours) from the neck down to the thighs and rinsing off the next morning.[32] During warm, humid summer months, the athlete may need to reapply selenium sulfide weekly over the next 4 weeks to suppress recurrent infections. In extensive disease, oral ketoconazole, 200 mg daily for 5 days or 400 mg once a month, has been shown to be an effective alternative therapy.[4] With oral therapy, the athlete needs to continue to exercise and perspire for at least 1 hour after taking ketoconazole to promote absorption into the hair root.[3] One should be mindful of the hepatotoxicity of ketonconazole. Griseofulvin is not an effective treatment.

Herpes Gladiatorum

Herpes gladiatorum or rugbeiorum refers to a herpes simplex virus (HSV-1) outbreak on the face of wrestlers or rugby players during "lock-up" or in a scrum. The lesions may appear suddenly on the right side of the face, trunk, and extremities 1 to 2 weeks following close contact.[45] Classic lesions appear as a cluster of painful vesicles on an erythematous base and resolve in 10 to 14 days. The virus is passed by direct face-to-face transmission between athletes and headgear does not decrease the risk of transmission.[45] Facial lesions can easily be mistaken in their later stages for impetigo and therefore lead to significant morbidity involving the conjunctiva and cornea if diagnosis is delayed. The clinical appearance of this disorder can be variable, and all suspicious skin and eye lesions should be cultured for HSV-1. A Tzanck smear of the base of the vesicle may reveal multinucleated giant cells. Treatment with topical acyclovir is ineffective in decreasing viral shedding. Famciclovir, 250 mg three times a day for 5 days, and valaciclovir, 1 g twice a day for 5 days, are recommended for initial therapy.[10] Neither famciclovir or valaciclovir have been approved for use in children less than 18 years of age. Valaciclovir, 500 mg once daily, has been prescribed for prophylaxis to prevent recurrence during the season.[10] In the pediatric population, 40 to 80 mg/kg/day of Acyclovir® in three or four doses for 7 to 10 days remains the standard of care.[36] Primary prevention to avoid horizontal

transmission of the herpes infection consists of disinfecting floor mats and equipment, as well as inspection of all athletes prior to participation. The NCAA will allow wrestlers to participate if they are free of systemic systems and have not developed new lesions during the last 72 hours, if all lesions have a firm adherent crust, and if the wrestler has been on antiviral therapy for 120 hours.[1] The healthcare team must educate athletes regarding the seriousness of the infection and examine athletes engaging in close contact prior to each practice and competition.

Molluscum Contagiosum

Molluscum contagiosum is a common viral skin disease characterized by small umbilicated, flesh-colored, dome-shaped papules. This poxvirus is highly contagious and spread by direct skin transmission from person to person, auto-innoculation, water transmission, and gymnastic equipment.[5] The papules are self-limiting and resolve over weeks to months. For the athlete to continue competition, the lesions must be removed by sharp curettage or liquid nitrogen, and any solitary lesions must be covered with a gas-permeable dressing (Op-Site®, Bioclusive®), ProWrap®, and stretch tape.[1] Liquid nitrogen, cantharidin (0.7% in collodion), topical tretinoin (Retin-A®), electrodessication, and the use of imiquimod 5% cream have been successful but may require multiple treatments.[5,36]

Verruca

The human papilloma virus induces warts, or verrucae vulgaris. The average incubation period for the virus is 6 months.[33] Plantar warts disrupt the normal dermatoglyphics of the pressure points of the feet and often coalesce to form a gyrate or mosaic pattern. Small black dots representing thrombosed capillaries within a hyperkeratotic plaque confirm the diagnosis.[10] Warts can be transmitted on swimming pool decks and gymnasium weights.[26] The NCAA requires wrestlers to be able to cover multiple digitate verrucae of the face with a mask, and verrucae plana or vulgaris must be adequately covered or the athlete will be disqualified.[1] Healthcare providers need to be cautious in treating children as most of these lesions will resolve spontaneously; however, due to their painful, contagious, and unsightly character, multiple treatment modalities have been utilized. Warts are typically very resistant to therapy. Therapy consists of a combination of keratolytic agents and physical debulking. Salicylic acid solutions (Duofilm®, Compound W®) and 40% plaster compounds (Mediplast®) can be applied overnight with an occlusion wrap during the season.[3] Excess tissue can be pared the next day. The process can be repeated on a daily basis until a satisfactory result is achieved. Liquid nitrogen cryotherapy can be done concurrently or separately every 2 weeks. Cryotherapy is effective in devitalizing and debulking the affected tissue but is a painful process requiring multiple applications and is not recommended during the season.[3] The use of lasers to destroy the vascular supply of the wart has been utilized in recalcitrant cases. Surgery should be discouraged due to the formation of chronic, painful scar tissue.

MISCELLANEOUS

Contact Dermatitis

Athletes are exposed to multiple irritants and allergens within the environment, on equipment, and through ingestion of medications and application of compounds. Each irritant or allergen may produce a characteristic contact dermatitis dependent upon its concentration, duration of exposure, and condition of the skin. Primary irritant dermatitis is a nonallergic reaction that leads to symptoms within minutes of the exposure.[33] The dermatitis is localized to the contact site and exhibits erythema and a burning sensation. Common irritants are detergents and soaps found in swimming pools and locker rooms, adhesive pretape sprays, sunscreens, and fiberglass.[10]

Allergic contact dermatitis is an acquired immune response that develops hours to days after recurrent exposure to an allergen. The dermatitis exhibits patches of erythema, edema, vesicle formation, and extreme pruritus. Equipment with protective rubber coverings (golf clubs) and black rubber seals (swim gear), tanned leather straps, latex products, iodine preparations, topical antibiotic ointments, adhesive tape, shoe dyes, and poison ivy or oak have all produced allergic reactions.[32]

After a careful exposure history and a high index of suspicion, initial treatment includes avoidance and washing with water in an attempt to physically remove the irritant and prevent further systemic progression. Alternative equipment has been manufactured using polyurethane, neoprene, and silicone to alleviate allergic reactions. Calamine lotion, Burow's wet dressings, and topical corticosteroids can be applied to reduce inflammation. Antihistamines, corticosteroids, analgesics, and H2-antagonists are commonly utilized for moderate to severe systemic hypersensitivity reactions by either oral or intravenous

routes. Patch testing is helpful in proper identification of the allergan triggering the dermatitis.

Swimmer's Itch

Swimmer's itch is a parasitic dermatitis produced by the cercariae form of the schistosomes commonly found in freshwater lakes of the United States.[46] The dermatitis is usually found on exposed areas, not under the bathing suit. The cercariae penetrate the skin but die beneath the epidermis, leaving a protein residue.[47] Initially, pruritus occurs and lasts for about 1 hour followed by the appearance of 1- to 2-mm macules at each site of penetration.[46] The macules may disappear 3 to 4 hours later, and papules with surrounding erythema develop and may last for a week.[35] Vesicles may form after the second day, and a brownish pigmentation may persist for months at the site of the lesions.[46] Treatment consists of avoidance of known freshwater where the parasite has been isolated. Antihistamines, calamine lotion, and topical steroids have all been utilized for symptomatic relief.[35]

Seabather's Eruptions

Seabather's eruption has been reported off the East coast from Long Island, NY, to Bermuda, along the west coat of Florida, and down into the Caribbean.[48] The pruritic papules and wheals primarily occur in scuba divers and swimmers on parts of the body covered by the bathing suit. Free-swimming, saltwater larval forms of *Edwardsiella lineata* and *Linuche unguiculata* contain nematocysts that discharge a toxin onto the skin when trapped between the skin and bathing suit.[35] Prolonged wearing of the bathing suit, washing off with fresh water, and strenuous exercise can activate the nematocysts.[48] The dermatitis usually will persist for 3 to 7 days but has been known to recur 2 weeks after the initial eruption when the suit was worn again.[48] Meat tenderizers, baking soda, warm saltwater, vinegar solution, and shaving cream are recommended to denature any undischarged nematocysts on the skin.[47]

Green Hair

Regular swimmers with natural or tinted blonde, gray, or white hair may develop a green discoloration to their hair from the uptake of copper ions in swimming pool water.[13] The green discoloration was originally thought to be due to chlorine, but chlorine now is considered to be only a bleach. The copper may occur naturally in the water or from copper pipes and algicides.[35] Immediately washing the hair and maintaining the pool pH between 7.4 and 7.6 will prevent this condition.[8] Washing the hair with copper-chelating shampoos (Ultraswim®) for 30 minutes or soaking in 3% hydrogen peroxide for 3 hours will return the hair to its previous color.[13]

Envenomation

Insects, especially hymenoptera (bees, wasps, hornets, yellow jackets, fire ants), can cause very dangerous allergic reactions.[49] Hymenoptera venom contains various components producing localized pain, erythema, edema, and pruritus. A systemic toxic reaction usually occurs after multiple stings or with a participant having a known allergic reaction. Symptoms include nausea, vomiting, diarrhea, lightheadedness, headache, angioedema, syncope, and ultimately anaphylaxis with respiratory collapse or cardiac death. The majority of these reactions occur within the first 15 minutes. In general, severe reactions occur when the interval between stings and onset of symptoms is short.[49] Definitive identification of the insect species is unnecessary because the signs and symptoms of envenomations are similar in hymenoptera. The stinger should be removed and the site thoroughly washed with soap and water. The venom can be neutralized with meat tenderizer or baking soda at the site. Ice packs will reduce the swelling and slow the absorption of the venom. Antihistamines and NSAIDs are commonly prescribed. Participants with known allergic reactions to hymenoptera should be advised to use sunscreens with insect repellent formulas. Athletic trainers or physicians covering events should have on hand an EpiPen® kit for participants with known allergic reactions and instruct athletes to wear medical warning tags.[49]

Exercised-Induced Anaphylaxis

The most severe form of urticaria is exercised-induced anaphylaxis (EIA).[8] Pruritus with large wheals may progress to systemic symptoms of wheezing, nausea, diarrhea, angioedema, hypotension, and shock. Running has been found to be the most common exercise predisposed to EIA.[10] Three distinct patterns of cutaneous involvement may be seen: cholinergic urticaria, giant urticaria, and angioedema without urticaria.[50] In contrast to cholinergic urticaria, EIA lesions are large and are not produced by hot showers, pyrexia, or anxiety.[27] Plasma histamine levels are elevated in all forms of EIA. Preventive measures include not exercising in extremes of either hot or cold weather and the use of nonsedating antihistamines 1 hour prior to exercise.[51]

Athletes who want to continue vigorous exercise should be instructed to exercise with someone (jogging partner) who has knowledge of their condition and can administer an injectable subcutaneous 1:1000 epinephrine syringe (EpiPen® kit) if they develop symptoms.[3]

CONCLUSION

Our largest organ system serves to protect us from natural and manmade forces that tend to irritate and infect the skin during athletic endeavors. The skin's reaction to physical agents, environmental stimuli, and infective organisms may predispose an athlete to a decrement in performance, impede training, or disqualify the athlete from participation. The athlete deserves an accurate diagnosis and optimal treatment plan by the healthcare team to compete successfully and achieve peak performance.

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57 GASTROINTESTINAL PROBLEMS IN TRAINING AND COMPETITION

David L. Brown

INTRODUCTION

The athlete in training and competition is no stranger to disorders of the gastrointestinal (GI) system. In fact, the body's visceral functions can often be the nemesis of high-demand exercise. Both psychological and physiological factors impact GI function. Knowledge of these factors will help the primary care physician understand and manage their active patients.

EPIDEMIOLOGY

Gastrointestinal symptoms are highly prevalent in athletes, particularly those involved in demanding endurance activities. Upper GI symptoms include nausea, vomiting, belching, heartburn, and abdominal pain. Lower GI symptoms reported include bloating, cramps, fecal urgency, diarrhea, and fecal incontinence. In addition to subjective symptoms, abnormalities on laboratory evaluation have been observed, including elevations in liver associated enzymes. Long-distance runners have been the most studied group from a gastrointestinal perspective; however, recent studies have also looked at long distance walkers, cyclists, triathletes, and weightlifters. Upper and lower GI tract symptoms occur with equal prevalence in cyclists (67 and 64%, respectively). Lower GI symptoms predominate over upper GI symptoms in endurance runners (71% vs. 36%). Whether running or riding, these same patterns also hold true among triathletes.[1] In low-intensity, long-distance walking, the overall occurrence of gastrointestinal symptoms has been found to be much lower than in other sports studied. During one event, only 24% of walkers surveyed reported symptoms. The most common symptoms were flatulence and nausea, each of which occurred in only 5% of walkers.[2]

Symptomatic gastroesophageal reflux is extremely common in athletes. Collings et al.,[3] using esophageal pH monitoring, found that cyclists had the lowest esophageal acid exposure, followed closely by runners. In their study, weightlifters had the highest rates of reflux, occurring for over 18% of their exercise period. All groups had increased reflux when exercising in the post-prandial period. Cyclists had a modest increase in reflux after eating; however, weightlifters nearly doubled their amount of reflux to 35% and runners tripled their reflux (from 8 to 26%).

When peptic ulcer disease (PUD) is observed, it is associated with the primary risk factors of *Helicobacter pylori* infection and, of particular

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importance in athletes, use of nonsteroidal anti-inflammatory drugs (NSAIDs). *H. pylori* is associated with 65 to 95% of gastric ulcers and 75% of duodenal ulcers.[4] The U.S. Food and Drug Administration has estimated the risk of a clinically significant, NSAID-induced event, including bleeding and perforation, to be 1 to 4% per year for non-selective NSAIDS. Either of these risk factors alone increases the risk of ulcer 20-fold. When these two factors combine, an individual is 61 times more likely to develop ulcer disease. The risk of ulcer bleeding goes up 1.8 times with *H. pylori* infection, 4.9 times with NSAID use, and 6 times in the presence of both factors.[5]

The primary lower gastrointestinal condition affecting athletes is runner's diarrhea. Studies indicate that this syndrome affects up to 26% of marathon runners.[6] Runner's diarrhea is not typically associated with bleeding; however, McCabe et al.[7] showed that 20% of runners completing a marathon had occult blood in their stools and another 6% had bloody diarrhea. Further, 17% had noted frank hematochezia while in training.

GASTROINTESTINAL HISTORY AND PHYSICAL

History

The organized evaluation of any GI complaint begins with a thorough history. This includes delineating the exact nature and chronicity of the problem, specifying exacerbating and relieving factors, and assessing for red flag symptoms. Individuals should be questioned about the location, quality, and radiation of any pain as well as the relationship of symptoms to food ingestion. Symptoms related to the upper GI tract include nausea, vomiting, bloating, and excessive belching. Lower tract symptoms include crampy/spasmodic abdominal pain, constipation, and diarrhea. The athlete's symptoms should be correlated with their sporting activity and mode of training. Providers should take a careful dietary history to assess pre-training and pre-competition food, fluid, and supplement intake. Any dietary changes that pre-date the onset of symptoms could identify the culprit. It is also important to inquire about the relationship of symptoms to transitions in training, especially any escalation in training volume and intensity.

The presence of red flag symptoms should prompt a gastroenterology referral. Upper tract alarm symptoms include dysphagia, odynophagia, hematemesis, melena, and early satiety. For the lower GI tract, a diminution of stool caliber and hematochezia should cause concern. Unremitting pain, especially pain that awakens an individual from sleep, and any systemic symptoms such as fevers, night sweats, and unplanned weight loss are especially concerning. Additionally, even more moderate symptoms that are failing maximal conservative management should trigger further evaluation.

The review of systems should key on symptoms that indicate involvement of a specific portion of the digestive system or involvement of organ systems masquerading as a gastrointestinal condition. The middle-aged athlete with apparent exertional GI complaints such as heartburn, epigastric pain, and nausea, may actually be having angina. Providers need to have a high index of suspicion for coronary artery disease in these individuals and should thoroughly explore their cardiac risk factors. The athlete with chronic constipation or diarrhea should be asked about environmental temperature intolerance, palpitations, tremor, skin and hair changes, and menstrual irregularities that could point to hyper- or hypothyroidism. The presence of right upper quadrant or epigastric pain that is specifically triggered by fatty foods could indicate symptomatic cholelithiasis or recurrent pancreatitis. If the athlete is having flank or lower quadrant abdominal pain associated with gross or microscopic hematuria, then nephrolithiasis or other urinary tract pathology should be considered. Female athletes with abdominal complaints should be questioned about the relationship of their symptoms to their menstrual cycle, looking for ovarian and uterine pathology or pregnancy. Orthopedic issues such as sacral, pelvic, and femoral neck stress fractures can also present with lower abdominal symptoms. If a stress fracture is in the differential, providers should assess for risk factors such as rapid progression in training, poor nutritional status, and the presence of the female athlete triad.

Physical Examination

The physical exam, while fairly insensitive, should be used to provide evidence to corroborate or refute what is found in the history. While the physical is often tailored to upper vs. lower tract complaints, it should be kept in mind that some conditions, such as inflammatory bowel disease, may affect anywhere from the mouth to the anus. Working top to bottom, the oral cavity should be assessed for signs of ulceration or posterior pharyngeal inflammation. The tooth enamel should be examined for deterioration that can occur with chronic acid reflux or forced vomiting

in conjunction with an eating disorder. It is important to examine the neck for lymphadenopathy, thyroid nodules, and thyromegaly.

The abdominal exam should start with inspection, focusing on distension or overt organomegaly. To avoid altering the bowel sound pattern, auscultation for hypo- or hyperactive bowel sounds should be accomplished prior to percussion or palpation. Percussion is useful in evaluating for hyperresonance, which would indicate bowel distension or obstruction. Dullness to percussion can be seen focally when hepatosplenomegaly or a mass lesion is present. Shifting dullness with positional changes is seen with accumulation of ascites fluid. Palpation should start in areas remote from symptoms and then conclude by focusing over the area of concern. The provider should note any rigidity, organomegaly, involuntary guarding, or rebound tenderness. In the acute setting, special tests can also be helpful. A positive Murphy's sign is suggestive of acute cholecystitis. Classically, focal pain over McBurney's point indicates an acute appendicitis. Obturator, psoas, and "heel tap" signs are all tests that can be performed to document peritoneal irritation.

The exam should conclude with at least a visual inspection of the anus, looking for perianal disease. When lower gastrointestinal symptoms are present, however, providers should perform a digital rectal exam and assess for occult blood. In the female athlete with lower quadrant tenderness, no exam would be complete without performing a bimanual pelvic exam to assess the uterus and adnexa.

UPPER GASTROINTESTINAL DISEASES

Gastroesophageal Reflux Disease

Presentation

As in the non-athletic population, the most common presenting complaints in athletes with gastroesophageal reflux disease (GERD) are heartburn and acid regurgitation. When an individual describes the classic presentation of retrosternal burning, exacerbated by meals, intense workouts, and recumbency with resolution on antacids, the diagnosis is clear; however, many athletes present with more atypical symptoms, including nausea, excessive salivation (water brash), bloating, and belching.[8] Still others may present only with extraintestinal complaints, such as sore throat, exertional dyspnea, cough, or wheezing (see Table 57.1).

TABLE 57.1
GERD Symptom Patterns

Classic Symptoms
Heartburn
Acid regurgitation
Non-Specific Symptoms
Nausea
Dyspepsia
Bloating
Belching
Indigestion
Hypersalivation/water brash
Atypical Symptoms/Signs
Pulmonary
Asthma/wheezing
Chronic cough
Ear, nose, and throat
Dental erosions
Halitosis
Lingual sensitivity
Chronic pharyngitis
Hoarseness
Rhinitis/Sinusitis
Globus
Cardiac
Atypical chest pain
Red Flag Symptoms
Chronic untreated symptoms
Dysphagia
Weight loss
Hemetemesis
Melena
Odynophagia
Vomiting
Early satiety

Pathophysiology

To understand the pathophysiology of acid reflux, one must first be familiar with the tiered defense system of the esophagus. The primary mechanical barriers to reflux are the lower esophageal sphincter (LES) and the diaphragm. The tonic contraction of the LES, when not swallowing, prevents the free reflux of gastric contents. The diaphragm encircles the LES and acts as a mechanical support, especially during physical exertion. This mechanical barrier is reinforced by luminal acid

clearance. Large bolus clearance is accomplished by gravity and swallow-induced peristalsis. Salivary and esophageal gland secretions, rich in bicarbonate, clear the residual acidity. The final barrier is the esophageal epithelium itself. Intercellular junctions limit hydrochloric acid diffusion between cells. Intracellular buffering is reinforced by transmembrane channels that exchange hydrogen ions for sodium and chloride for bicarbonate. The esophageal blood supply delivers bicarbonate and removes H^+ and CO_2 to restore cellular buffering capacity and maintain normal tissue acid balance.[9]

The pathophysiology of GERD involves the movement of gastric contents from the stomach into the esophagus. Symptoms develop when substances in the refluxate, including hydrochloric acid and the proteolytic enzyme pepsin, cause irritation of the esophageal epithelium. Reflux alone is insufficient to explain why individuals become symptomatic because healthy individuals have a physiologic amount of acid reflux. In fact, GERD patients have rates of gastric acid and pepsin reflux similar to healthy individuals.[10] The critical factor in symptom development appears to be that the contact time between refluxed material and the epithelium is so excessive that the normal gastric contents overwhelm the epithelial protective mechanisms. Alternatively, symptoms may develop when normal contact time occurs in the face of insufficient protective mechanisms.

Symptomatic reflux episodes during exercise are likely multifactorial but appear to correlate with transient LES relaxations (TLESRs). This vagally mediated reflex facilitates LES relaxation and gas venting in response to gaseous stomach distention. When reflux events coincide with TSLESRs, the decrease in LES tone lasts longer and is not accompanied by a swallow-induced peristaltic sweep, leading to prolonged acid exposure. Prolonged acid exposure may also occur due to delayed acid clearance from body positioning. Supine or forward-flexed posture during particular modes of exercise increases intraabdominal pressure and overcomes the mechanical protection of the LES; this posture also negates the bolus acid clearance achieved by gravity. Nevertheless, delayed acid clearance correlates best with impaired esophageal motility during exercise. Studies in trained athletes show that, as exercise intensity increases, a progressive decrease occurs in the frequency, duration, and amplitude of esophageal contractions. Increasing exercise intensity is also associated with increased reflux episodes and duration of acid exposure.[11] High-intensity exercise also reduces splanchnic blood flow, which may inhibit restoration of acid base balance and deprive the epithelium of the oxygen and nutrients needed for damage repair.

Evaluation and Management

If the history and physical raise red flags, the athlete should be referred for evaluation by a gastroenterologist (see Figure 57.1) Likewise, if an individual's symptoms are particularly severe or if the diagnosis is unclear, a GI referral is warranted. For patients with extraintestinal manifestations or atypical GERD symptoms, providers can consider an initial therapeutic trial; however, if empiric therapy fails, it is important to consult not only gastroenterology but also the specialty that would evaluate for any extraintestinal complications. In the face of a classic history and normal physical exam, it is reasonable to institute empiric therapy in a stepwise fashion (see Figure 57.2), starting with addressing any modifiable risk factors such as food and medication triggers, exercising immediately after meals, and wearing tight-fitting workout apparel.

Persistent symptoms despite behavioral interventions warrant medical therapy. If an athlete's complaints are episodic, over-the-counter (OTC) antacids or an H-2 receptor antagonist (H2RA) can be employed on an as-needed basis. This can be advanced to prescription-strength H2RA therapy if control is insufficient. Should symptoms continue after 6 weeks of H2RA therapy, neither continuing therapy nor increasing the dose is likely to achieve control (Level of Evidence A, randomized controlled trial).[12] At this point, it was previously common practice to consider add-on therapy with a pro-kinetic agent to improve LES tone, gastric emptying, and peristalsis. These agents all have side effects that make them undesirable for use in athletes. Bethanechol has generalized cholinergic effects. Metoclopramide has a high incidence of fatigue, restlessness, tremor, and tardive dyskinesia, making it a poor choice for anything more than sporadic use. Cisapride, formerly the pro-kinetic agent of choice, was found to be associated with arrhythmia development, especially with concomitant use of macrolides, imidazoles, or protease inhibitors.[13] This discovery led to severe prescribing restrictions in the United States, and it is currently available only by directly petitioning the manufacturer.

Thus, in individuals who fail to respond to H2RAs, standard-dose proton pump inhibitors (PPIs) are the treatment of choice. PPIs have been

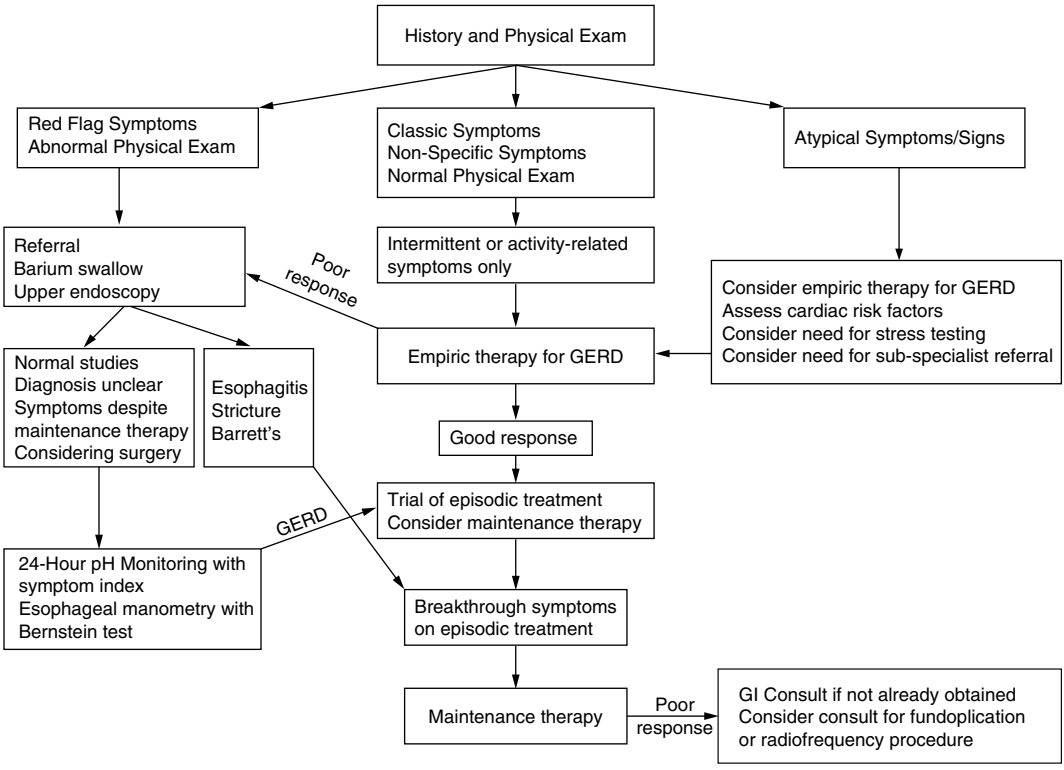

Figure 57.1 Evaluation of GERD. (Adapted from O'Connor, F.G., in *Textbook of Running Medicine*, 1st ed., McGraw-Hill, New York, 2001. With permission.)

shown to provide more rapid relief of symptoms (Level of Evidence A, randomized controlled trial)[14] and are more likely than H2RAs to heal and prevent recurrence of erosive esophagitis (Level of Evidence A, meta-analysis).[15] The results of a systematic review of PPI therapy show that the agents in this class are equally efficacious in controlling heartburn and had similar healing and relapse rates.[16] Because reported differences in initial bioavailability and antisecretory potency are not clinically significant with long-standing use, one PPI cannot be recommended over another (Level of Evidence B, non-randomized clinical trial).[17] If the response to episodic treatment is generally favorable but symptoms are occurring on a more chronic basis, maintenance therapy is beneficial. Because their efficacy is dose dependent, PPI therapy can be stepped up to control symptoms. Failure to respond to high-dose PPI therapy requires gastroenterologist evaluation to rule out complications of GERD. In the absence of findings consistent with reflux disease, further GI testing will be necessary to confirm GERD and assess for other esophageal disorders.

More invasive treatments are available for patients with an established diagnosis of GERD who respond poorly to PPIs, who are intolerant of medical therapy, or who desire a permanent solution to potentially eliminate their need for medication. Laparoscopic anti-reflux surgery has been shown to provide a 96% improvement in primary symptoms and 96% long-term satisfaction rate; however, 2% of patients were worse after surgery and 14% still required medication.[18] While the potential exists for excellent results, the best outcomes occur in carefully selected patients under the care of an experienced surgeon. Other endoscopic therapies, including suturing, radiofrequency ablation, injection therapy, and bulking therapy are currently being investigated. To date, these procedures have not been adequately studied. Their particular niche in GERD management and potential for widespread use have yet to be determined.

Peptic Ulcer Disease

Presentation

Epigastric pain is the hallmark of peptic ulcer disease. Both gastric and duodenal ulcers typically

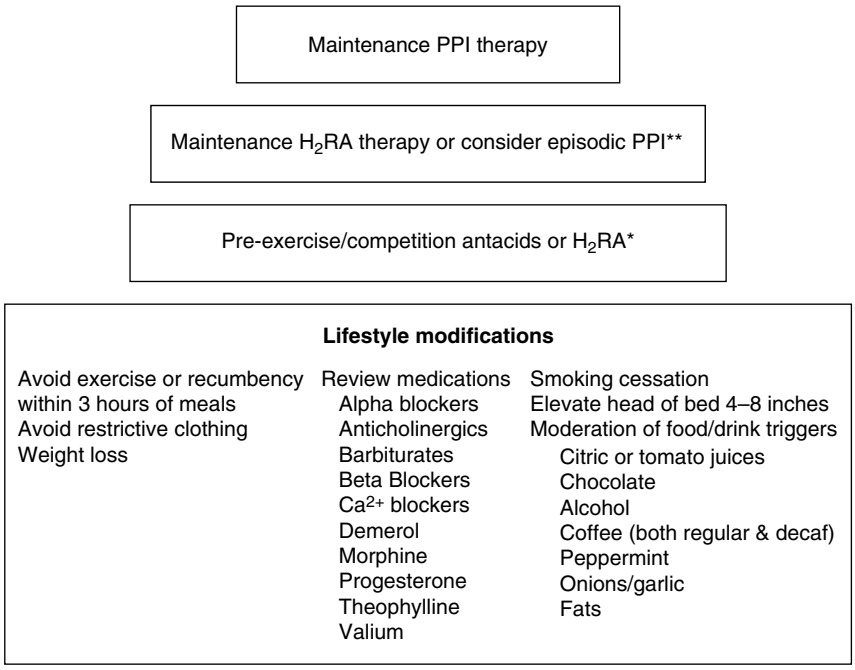

Figure 57.2 Therapeutic pyramid for exercise-related GERD. (Adapted from O'Connor, F.G., in *Textbook of Running Medicine*, 1st ed., McGraw-Hill, New York, 2001. With permission.)

present with deep burning or gnawing pain, sometimes with radiation to the back. With duodenal ulceration, symptoms usually develop 2 to 3 hours after meals and are relieved with food or antacid ingestion. The symptoms of gastric ulcers develop sooner after meals and are less consistently relieved with food or antacids. In fact, food ingestion can actually precipitate gastric ulcer pain in some individuals. Most PUD patients have associated anorexia and weight loss. Some patients, particularly with duodenal ulcers, experience hyperphagia and weight gain, presumably due to the mitigating effects of food. Not uncommonly, the initial presentation of PUD can be life-threatening upper GI hemorrhage or perforation.[19]

Pathophysiology

Peptic ulcers are erosions in the surface of the stomach or duodenum that extend down to the muscularis mucosa. *H. pylori* induces ulcers by both direct and indirect mechanisms. Bacterial phospholipases weaken the protective mucus barrier allowing toxic compounds from their breakdown of urea to damage the epithelium directly. The same urease enzyme that promotes this direct cell damage acts as a potent antigenic stimulator of immune cells. By inciting an exuberant host inflammatory response, *H. pylori* produces indirect epithelial damage.[20] NSAID prostaglandin inhibition affects multiple layers of the protective barrier of the GI tract. As organic acids (except nabumetone), they easily penetrate the hydrophobic mucous layer and by decreasing mucosal surface nitric oxide production degrade the ability of the stomach to elaborate its protective mucous layer. NSAIDs decrease acid buffering capacity by inhibiting bicarbonate synthesis and facilitate free radical damage by inhibiting glutathione, a superoxide radical scavenger. With increasing concentration, they diminish mucosal blood flow and penetrate the epithelial cells, eventually leading to mitochondrial oxidative uncoupling and cell death.[21] No studies have directly related NSAID use to upper GI symptoms or bleeding specifically in athletes; nevertheless, the increased mucosal permeability and decreased splanchnic blood flow that occurs with prolonged exercise may serve as a pathophysiologic foundation on which the effects of *H. pylori* and the NSAIDs can build.

Evaluation and Management

Athletes should be questioned regarding any relationship of symptom onset with NSAID use. Laboratory analysis should assess for occult GI bleeding and anemia. If any alarm signs or symptoms

are present or if an individual has new onset dyspepsia after the age of 45 or has a family history of gastric cancer, early gastroenterology referral is recommended.

Initially, NSAID use should be discontinued if possible. If analgesic therapy is crucial, replacing a non-selective NSAID with acetaminophen or a COX-2 inhibitor would be prudent. A systematic review of the upper GI safety and tolerability of one of the COX-2 inhibitors found a 46% lower rate of medication withdrawal for adverse events, a 71% lower risk of ulcers on endoscopy, and a 39% lower incidence of symptoms due to ulcers, perforations, bleeding, or obstruction compared to non-selective NSAIDs (Level of Evidence A, meta-analysis).[22]

If an athlete's symptoms are not predominantly GERD related, if no markers for severe disease are noted, and if medication-induced disease is eliminated, the consensus recommendations in the Maastricht 2-2000 report have adopted a "test and treat" approach to adults under the age of 45 with persistent dyspepsia. These patients should have a noninvasive test for *H. pylori* infection. Urea breath analysis is the favored test, with the stool antigen assay as an alternative. Individuals who are *H. pylori* negative should receive short-term H2RA or PPI therapy (4 to 6 weeks). If they fail empiric antisecretory therapy or if symptoms recur upon cessation of treatment, they should be referred for endoscopy. Symptomatic individuals who test positive for *H. pylori* should have eradication therapy. The Maastricht consensus panel recommends that proton pump inhibitors or ranitidine bismuth citrate along with clarithromycin and amoxicillin be used as first-line therapy. Metronidazole can be substituted for amoxicillin in penicillin-allergic patients. Subsequent second-line therapy should be with a PPI, bismuth, tetracycline, and metronidazole (see Table 57.2 for eradication regimens). All patients should be retested for evidence of a cure after a minimum of 4 weeks of therapy. The athlete should be off any antisecretory medication, especially PPIs, for a minimum of 1 week prior to retesting. Urea breath analysis is the post-treatment diagnostic test of choice. Stool antigen testing can be used if urea breath testing is unavailable. Individuals who fail second-line therapy and those with persistent dyspepsia should be referred to gastroenterology for further evaluation.[23]

TABLE 57.2
Regimens for Treatment of *Helicobacter pylori* Infection

Proton Pump Inhibitor Regimen	Eradication Rate (%)[a]
Amoxicillin, 1000 mg BID	96.4
Clarithromycin, 500 mg BID	
Bismuth subsalicylate, 525 mg QID	85–90
Tetracycline, 500 mg QID	
Metronidazole, 250 mg QID	
Metronidazole, 500 mg BID	89.8
Clarithromycin, 500 mg BID	
Amoxicillin, 1000 mg BID	79.0
Metronidazole, 500 mg BID	

[a] All eradication rates are based on a 7-day regimen. Although European data suggest that 7 days are adequate, this has not been confirmed by U.S. studies; thus, a full 14-day treatment course is recommended.

Source: Adapted from O'Connor, F.G., in *Textbook of Running Medicine*, 1st ed., McGraw-Hill, New York, 2001. With permission.

LOWER GASTROINTESTINAL DISEASES

Runner's Diarrhea

Presentation

Athletes with runner's diarrhea suffer from a syndrome encompassing a spectrum of exertional or immediately post-exertional lower gastrointestinal symptoms. Their presentation ranges from abdominal cramping and fecal urgency to diarrhea and frank incontinence. Often, runner's diarrhea occurs in association with increases in training mileage or with particularly strenuous training sessions and competitions. An individual may be able to endure an episode by transiently reducing their pace; however, when symptoms are more severe, it may be necessary to completely suspend the workout and quickly seek relief.

Pathophysiology

While the true etiology of runner's diarrhea remains unknown, several possible physiologic mechanisms have been proposed. Increased parasympathetic output during moderate exercise may intensify peristalsis, leading to cramping and rapid bowel transit. Heightened sympathetic tone during more intense exercise could lead to increased bowel activity by increasing the release of hormones such as gastrin and motilin.[24] Alternatively, strenuous exercise may lead to rapid shifts in intestinal fluid and electrolytes, causing colonic irritability.[25] Another attractive hypothesis is that

the 70 to 80% reduction in splanchnic blood flow that occurs with vigorous exercise may lead to an ischemic enteropathy. The resulting poor tissue perfusion maintained over the length of the exercise session could cause mucosal ischemia, leading to fluid shifts and diarrhea. This theory could also explain the high prevalence of GI bleeding in marathon runners in whom prolonged ischemia could then lead to mucosal necrosis, superficial erosions, and hemorrhage.[26]

Evaluation and Management

The history should thoroughly detail the onset, severity, and chronicity of symptoms. Documenting any recent travel, unusual food ingestion, or exposure to sick contacts can help distinguish a potential infectious etiology. It is important to inquire about diarrhea not associated with training as well as melena and hematochezia. If available, reviewing the athlete's training log is a crucial part of the evaluation. A detailed diary can help correlate symptoms with changes in exercise mode, frequency, duration, and intensity. A dietary journal, if not already included in the training log, can be helpful in identifying particular replacement fluids, nutritional supplements, or food products that may be triggering the athlete's symptoms. The past medical history should be reviewed, looking for any history of inflammatory bowel disease or previous GI hemorrhage. Other co-morbid diseases should be looked at carefully as several conditions, and potentially the medicine used to treat them, can lead to diarrhea. The individual's family history should be scrutinized for any inflammatory bowel disease or other chronic bowel conditions. A focused lab assessment includes fecal occult blood testing and a complete blood count to look for anemia. In the presence of severe diarrhea, serum electrolytes should be drawn. Liver enzymes and pancreatic enzymes can also be considered. If the history is suggestive of an infectious process, the stool should be examined for leukocytes, ova and parasites, and stool cultures.

For classic runner's diarrhea, treatment starts with a temporary reduction in training intensity and duration for 1 to 2 weeks. In most cases this alone is enough to abolish symptoms.[27] During this time, cross training with low or non-impact activities can be used to maintain the athlete's aerobic capacity. Any dietary or fluid replacement triggers should be eliminated. If a specific trigger is not identified, individuals with ongoing symptoms may benefit from dietary manipulation. A diet low in fiber can be helpful.[28] While not an adequate regimen for the control of chronic symptoms, some individuals may benefit from a complete liquid diet on the day prior to competition or a scheduled intense exercise session. When the diarrhea is under control, a full return to high-intensity exercise can be achieved by gradually increasing training as symptoms tolerate. Antidiarrheal medication should be used sparingly and with great caution. Anstispasmodics, such as loperamide, are generally safe; however, anticholinergic medications such as diphenoxylate with atropine (Lomotil®) are to be avoided due to the potential for increased heat injury risk secondary to their effect on sweating. Consulting gastroenterology is necessary should an individual have difficult-to-control symptoms or if any red flags are found during the history, physical, or laboratory evaluation.

Abdominal Pain (Side Stitch)

Presentation and Proposed Etiology

In the young, active population, abdominal pain with exertion is a common symptom. The conditions previously discussed notwithstanding, the so-called side stitch is the most common cause of abdominal pain in athletes. Typically seen in runners, it presents as a somewhat pleuritic aching sensation, usually in the right upper abdominal quadrant. It is often seen in deconditioned individuals starting an exercise program but can also be observed in athletes intensifying their training. Exercise in the post-prandial period is a frequent exacerbating factor. Side stitches usually stop immediately upon ceasing exercise. As an individual gains aerobic fitness, the frequency and severity of attacks tend to subside. While their true etiology remains elusive, they are most likely caused by hypoxia-induced diaphragmatic muscle spasm.[29] Other potential etiologies include pleural irritation, hepatic capsule irritation, symptomatic abdominal adhesions, and right colonic gas pain.[30]

The management of side stitches involves using the history to rule out not only the other gastrointestinal diseases discussed in this chapter but also other exertional pain syndromes, especially angina. Fortunately, other serious causes of abdominal pain with exercise, such as mesenteric ischemia, bowel infarction, omental infarction, and hepatic vein thrombosis, are rare. However, in the setting of unremitting pain, especially with signs of systemic illness or shock, these conditions need to be considered in the differential and patients referred for potential surgical evaluation.

Athletes with the typical features of a side stitch should be reassured that this is a benign

process and will get better as their conditioning improves. They should be advised against exercise immediately after eating. If an episode of pain does occur, temporarily stopping exercise, stretching the right arm over their head, and exhaling through pursed lips can help abort it quickly.[31]

Elevated Liver Enzymes

Etiology

Liver enzyme elevations have been described in otherwise asymptomatic long-distance runners as well as other athletes. They are usually found as incidental findings in lab studies obtained for reasons other than evaluating for liver disease. The suspected etiology is an ischemic insult secondary to reduced splanchnic blood flow and oxygen tension during vigorous exercise.[32] Increases in alanine aminotransferase (ALT), aspartate aminotransfease (AST), alkaline phosphatase, creatinine phosphatase, and lactate dehydrogenase that have been observed are confounded by the fact that these enzymes can also be elevated in response to musculoskeletal injury. However, measurement of glutamate dehdydrogenase and gamma-glutamyl-transferase, enzymes more specific to the liver, have confirmed hepatocellular injury.[33]

Management

Because these asymptomatic enzyme abnormalities are often discovered in the convalescent setting, the history and physical should focus on recent training sessions and environmental exposure, evaluating for evidence of a missed heat injury or episode of exertional rhabdomyolysis. The athlete should be questioned regarding any history of chronic liver disease or alcohol dependence and their medication list reviewed for any potentially hepatotoxic agents. With the nearly ubiquitous use of nutritional supplements, it is crucial to investigate this often-overlooked area.

The majority of athletes can be reassured that this appears to be a benign process and the enzyme abnormalities usually revert to normal within just 1 week after abstaining from exercise. Thus, the first step in the laboratory evaluation is to obtain a repeat liver enzyme panel after abstaining from NSAIDs, alcohol and exercise for 1 week. If the liver enzymes have not reached normal levels in that time, the athlete can be rechecked in 1 month. If the liver enzymes remain elevated on serial examinations, the athlete needs further evaluation, starting with an iron panel, total iron-binding capacity (TIBC), and hepatitis serologies. If these are unrevealing, antinuclear antibody (ANA) titer, anti-smooth-muscle antibody, ceruloplasmin, alpha-1-antitrypsin, and serum protein electrophoresis should be obtained as second-line tests. It is prudent to obtain a right upper quadrant ultrasound to evaluate for fatty liver, cholelithiasis, or other obstruction. If testing is abnormal, the liver enzymes have been mildly elevated for over 6 months with negative evaluation, if they have been significantly elevated (AST or ALT >150) without improvement for 2 months, or if signs of evolving hepatic insufficiency are present, then the athlete should be referred to gastroenterology.[34]

SUMMARY

Both upper and lower gastrointestinal problems affect the exercising population. Primary care physicians should be ever cognizant that athletes can fall prey to conditions that affect the general population. By being aware of red flag symptoms, the sports physician can expedite the evaluation and treatment of potentially serious conditions. By following an organized evaluation and management process, clinicians can accurately diagnose and offer prompt treatment for gastrointestinal conditions, thereby minimizing their impact on the active lifestyles of our athletes.

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58 GENITOURINARY PROBLEMS IN THE ATHLETE

Nicholas A. Piantanida

INTRODUCTION

Strenuous aerobic exercise creates extraordinary demands on the body. The athlete's genitourinary system must meet the dynamic physiologic demands of exercise without the conditioning benefit that occurs in the musculoskeletal and cardiovascular systems. Furthermore, with the popularity of ultra-endurance events, the genitourinary system is increasingly tasked to function under higher levels of both internal and external demands. This chapter briefly reviews renal anatomy and physiology in an exercising athlete and then details a diagnostic and therapeutic approach to common genitourinary problems in the athlete.

RENAL ANATOMY

The kidneys are retroperitoneal organs aligned obliquely along the borders of the psoas muscles. The liver slightly displaces the right kidney so that it is lower than the left kidney. The kidneys are partially mobile, creating displacement of 4 to 5 cm with the extremes of inspiration. Supporting elements to the kidneys include perirenal fat, the renal vascular pedicle, abdominal muscle tone, and the expansive effects of the abdominal viscera. A shared autonomic innervation with intraperitoneal organs explains, in part, some of the gastrointestinal symptoms that accompany genitourinary disease.[1]

On longitudinal section of the kidney (Figure 58.1), two zones can be identified: an outer cortex and an inner medulla. The medulla is composed of a number of pyramidal shaped structures — renal pyramids. These pyramids have an outer zone along the renal cortex and an inner zone that funnels into the calyx called papillae. The nephron is the functioning unit of the kidney. The greatest function of the entire nephron is perhaps reabsorptive. Urine is formed by filtration, reabsorptive, and secretory processes along the entire length of the nephron.[1]

The blood supply to the kidney is highly specialized. Blood enters the kidney from the renal artery and branches numerous times until forming afferent arterioles. The plasma from the afferent arteriole filters through with the glomerular capillary tuft. At the glomerular capillary tuft, 20% of the plasma water is filtered and the remaining plasma, along with larger solutes, travels along an efferent capillary network surrounding the tubular network of the nephron. The efferent arteriole, in the tubular portions, will capture reabsorbed water and solutes and transition substances to be secreted.[2]

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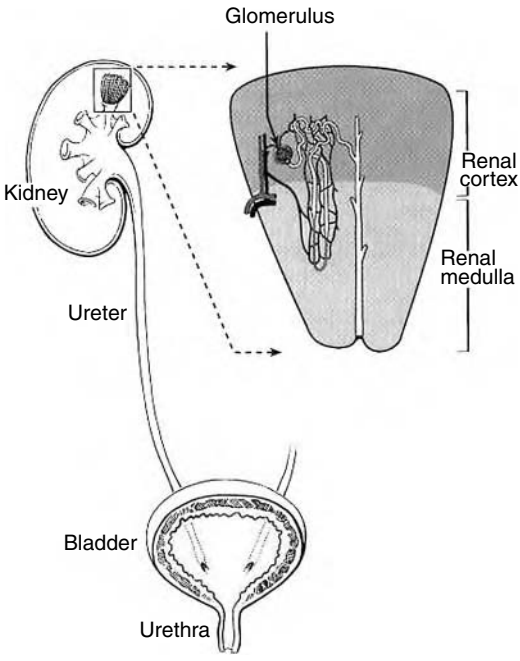


Figure 58.1 Genitourinary system. (From Sokolosky, M.C., *Emerg. Med. Clin. North America,* 19, 3. With permission.)

RENAL PHYSIOLOGY

At rest, 20 to 25% of cardiac output goes to the kidneys. The majority of this blood flow passes into the renal cortex, with the glomerular portion of the nephron filtering an average of 180 liters of plasma each day. The tubular portion of the nephron reabsorbs 99.5% of the filtered water and essential solutes.[2] Renal blood flow (RBF) and glomerular filtration rate (GFR) are renal perfusion indices. RBF is regulated, similar to blood flow in any organ, by the net arteriovenous pressure difference across the vascular bed. GFR is the volume of plasma per minute filtered at the level of the glomerulus. RBF and GFR are linked in differing degrees to intrinsic and extrinsic mechanisms of profusion modulation.

With exercise, RBF diminishes from the resting volume by 25%. A process known as autoregulation is an intrinsic renal mechanism to maintain GFR and RBF relatively constant in the face of changing systemic arterial pressure. Neural and hormonal influences represent extrinsic mechanisms capable of modulating RBF and GFR even in the autoregulatory range of systemic arterial pressure. In either the initial setting of increased sympathetic vascular tone or under conditions of moderate intensity exercise, the renal neural-regulating mechanism will preferentially increase vascular tone in the efferent arteriole, which will decrease RBF to a greater extent than the GFR. Such a process increases hydrostatic pressure at the glomerular tuff and generates an increased filtration fraction (GFR/RBF). As further sympathetic stimulation is generated, as in the setting of severe-intensity exercise, the afferent arterioles will demonstrate higher levels of vasoconstriction, and the decreases in GFR will parallel those of RBF. Several hormonal and other endogenous substances influence renal vasoconstriction or vasodilation. Angiotensin II, a potent vasoconstrictor, is synthesized through the cascade of increased renin release by granular cells reacting to decreased renal profusion. Prostaglandins are potent vasodilators that modulate the vasoconstricting effects of the sympathetic system and angiotensin II.[2,3]

During exercise, the role of antidiuretic hormone (ADH) appears to be protective to reduce urinary water excretion. ADH is produced in the hypothalamus and stored in the posterior pituitary. ADH is released under conditions of high plasma osmolality and/or low plasma volume. Osmoreceptors are the most sensitive measure for increased secretion of ADH, but the plasma volume receptors, once activated, elicit the strongest response. Thirst is triggered by low plasma volume and is a late sign of dehydration, behind increased serum osmolality.[2]

The athlete's body fluid homeostasis is held in a balance by its ability to maintain hydration levels and manage normal levels of total body sodium. Hyperhydration does not appear to significantly influence an athlete's performance or postexercise urine output. In fact, in endurance events, overzealous hydration predisposes the athlete to exercise-induced hyponatremia. Speedy et al.[4] have reported that the incidence of hyponatremia in the New Zealand Ironman triathlon fell from 22% in 1997 race to 3% the following year when participants were encouraged to drink more conservatively and a broader spacing of watering stations was applied.

UROLOGIC LABORATORY EXAMINATION

Patients with urinary tract symptoms or signs should undergo urinalysis. Macroscopic urinalysis (dip strip) can be a useful clinic-based screening tool for symptomatic patients. A normal dip strip is sensitive enough to make microscopic analysis of the urine unnecessary. Abnormalities on dip strip require further investigation by complete microscopic urinalysis to include sediment examination.[5]

TABLE 58.1
Dip Strip Indices

Factor	Description	Modifying Agents
Color and appearance	Assess gross hematuria and pyuria	Drugs, foods, dyes
Specific gravity	Measure of urinary concentration	ADH, glucose, protein
pH	Degree of urine acidity	Uric acid stones or *Proteus* sp.
Protein	Measures urine albumen Trace = 15–29 mg/dL 1+ = 30–99 mg/dL 2+ = 100–299 mg/dL 3+ = 300–999 mg/dL 4+ = >1000 mg/dL	Fever, exercise, dehydration, highly concentrated urine or orthostatic factors
Glucose	Accurate for urinary glucose	Ascorbic acid, cephalosporins
Hemoglobin	Screen for erythrocytes	Myoglobin, ascorbic acid
Nitrite	Positive when number of bacturia >100k	Coagulase-splitting species, p.m. void
Leukocytes	Indicator of pyuria	Glucosuria, ascorbic acid, drugs

Source: Williams, R.D., in *Smith's General Urology*, 15th ed., Tansgho, E.A. and McAninch, J.W., Eds., Lange Medical Publications/McGraw-Hill, Los Altos, CA, 2000, chap. 5. With permission.

Specimen collection is a critical step in urinalysis. A properly obtained midstream urine specimen is vital to accurate urinalysis. Refer to Table 58.1 for a review of the dip-strip indices and sensitivities, as well as confounders to dip-strip interpretation.[5] The microscopic exam of urinary sediment allows the provider to accurately define the complete picture of renal or bladder pathology. The morphology and quantity of various elements of the urinary sediment can assist the provider with a diagnosis. For example, red blood cell casts are pathognomonic of intrinsic renal disease in the form of glomerulonephritis or vasculitis. The routine practice of screening urine dip strips as a component of the preparticipation physical examination on healthy athletes is not recommended, as demonstrated by Peggs et al.[6]

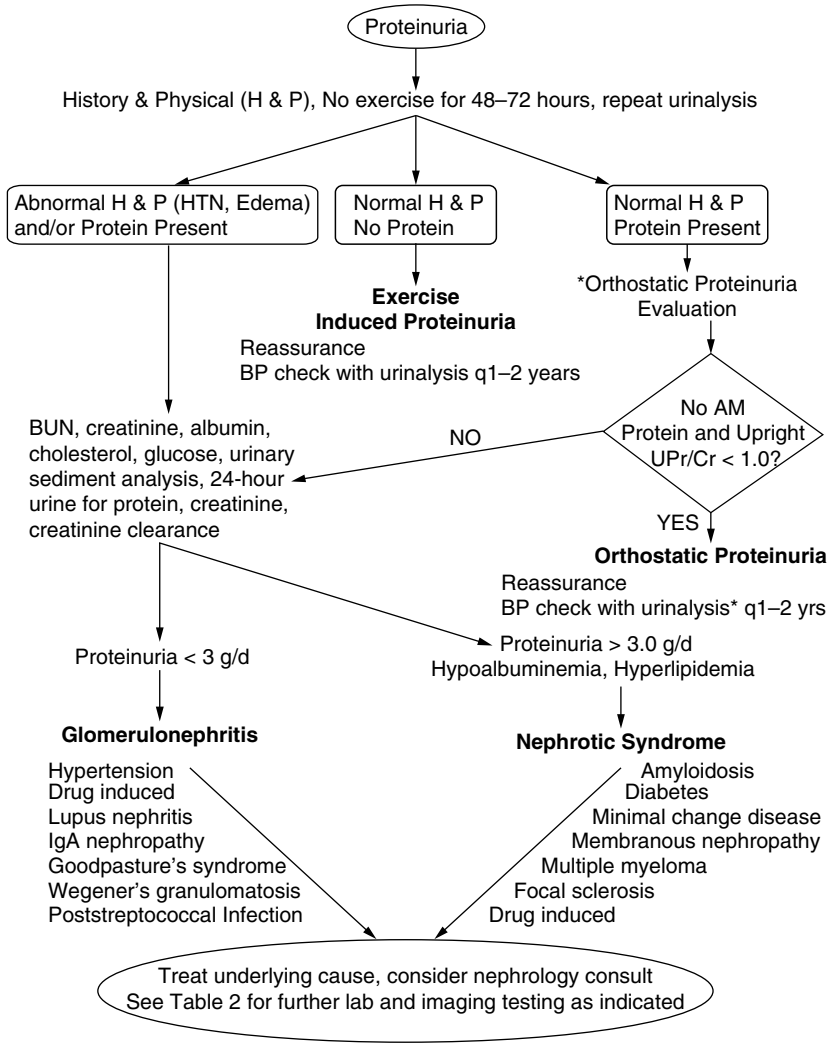
PROTEINURIA

Proteinuria is defined as more than 150 mg of protein per 24 hours. Proteinuria is a common finding on dip strip within 48 hours of exercise, with fewer than 2% of all cases screened having a serious urinary tract disorder.[7] Common causes of benign proteinuria include dehydration, orthostasis, fever, heat or cold injury, emotional stress, vigorous exercise, and acute illnesses. Pathologic causes of proteinuria include chronic disease states, such as hypertension, diabetes, or collagen vascular diseases, which produce end organ renal disease. Also, processes that involve cancer, infection, or autoimmune disease can directly invade or injure the renal structure to cause proteinuria.[7]

Exercise-induced proteinuria is relatively common with several pathophysiologic features depending on the nature and intensity of the exercise. In runners and other endurance athletes undertaking sustained aerobic effort, proteinuria results from increased glomerular capillary permeability to protein. As previously mentioned, moderate exercise reduces RBF with a proportionally milder diminishment of GFR. This proportional difference in RBF and GFR translates to a greater filtration fraction of macromolecules into the tubular lumen. Furthermore, sustained aerobic activity enhances renin formation which increases glomerular membrane permeability.[3]

In sprinters and other athletes engaged in severe, short-term, exhaustive exercise, proteinuria results from a mix of glomerular and tubular contributions. These athletes experience proteinuria from the increased glomerular permeability as described and also have impaired tubular resorption of plasma proteins. Athletic effort at brief maximal intensity produces a macromolecule load and solute demand that overwhelms the ability of the renal tube to reabsorb protein.[3]

Proteinuria is usually detected qualitatitively with the use of a screening urine dip strip (Table 58.1). A correlation with urine specific gravity is important to avoid a false positive result that may be secondary to a high urine concentration (specific gravity levels over 1.025). In cases where the



*Orthostatic proteinuria evaluation: 1) Overnight 1st morning supine void. 2) Next day 12-hour urine collection to measure urine protein (UPr) to urine creatinine (Cr) ratio.

Figure 58.2 Proteinuria algorithm.

urine dip strip for protein is 2+ or greater, urine specific gravity is less important.[3]

In an athlete with proteinuria, urinalysis should be repeated after a period of 48 to 72 hours free from exercise. A more generous period of 1 week should be considered in an athlete recovering from a febrile illness. If this second dip strip is positive, then a detailed evaluation should be pursued. See the proteinuria algorithm in Figure 58.2 and the diagnostic tests in Table 58.2 for a stepwise approach to the athlete with proteinuria (Level of Evidence B, systematic review).[7,8]

A medical history taken from a proteinuric athlete should focus on drug exposure, voiding history, metabolic disease, and recent illnesses. Family and personal history of developmental anomalies should be defined, including hereditary nephritis and polycystic kidney disease. A medication history should include inquiring about the use of ergogenic aids. A review of systems should involve a discussion of fever, weight loss, renal colic pain, and other urinary symptoms. Physical exam should identify hypertension, an infectious source, and search for edema in the extremities.

Treatment for benign proteinuria in the athlete includes correcting the underlying cause, whether it is poor hydration, acclimatization, stress management, or avoiding exercise during periods of

TABLE 58.2
Diagnostic Testing in Proteinuria and Hematuria

Test	Interpretation of Finding
Blood chemistries	Identify any abnormalities following renal disease
Complete blood count	Demonstrate any renal impairment on hematopoiesis
Antinuclear antibody	Elevated in systemic lupus erythematosus
C-reactive protein	Normal rules out an inflammatory cause
Serum and urine electrophoresis	Abnormal in multiple myeloma
HIV, antistrep O titer, hepatitis	Defines infectious causes for glomerular proteinuria
Cryoglobulins	Present in myeloma, lupus, and other autoimmune diseases
Complement C3 and C4	Decreased with increased immune complexes
Renal ultrasound or CT scan	Anatomic survey of renal anatomy
Chest x-ray	Rule out evidence of systemic disease (strep pneumonia, amyloidosis, sarcoidosis)

temperature stress or physical illness. Exercise-induced proteinuria will recur at a specific level of individual exertion. Athletes with exercise-induced proteinuria have no increased risk for chronic renal disease. They should participate to the full capacity of their sport and obtain a medical check-up with urinalysis yearly (Level of Evidence C, consensus opinion).[3,7,8]

HEMATURIA

Hematuria is defined as the abnormal excretion of red blood cells (RBCs) (>3 RBC/high power field [HPF]) in the urine.[9] Hematuria in athletes is known by several names, including sports hematuria, stress hematuria, and 10,000-meter hematuria. The incidence of exercise-induced hematuria is variable and ranges from 11 to 100%.[10] This broad variability follows in order of exercise intensity, type of exercise, and degrees of dehydration. Fortunately, the incidence of serious disease in men and women is low, and episodes of microscopic or gross exercise-induced hematuria resolve in several days of rest.[10]

The etiology of sports hematuria is multifactorial and varies from physiologic to traumatic sources. As previously mentioned, exercise physiologic stress to the kidney diminishes blood flow, while GFR is preserved by autoregulation and neurohormonal factors. In this configuration, two forces are at play that cause hematuria. First, vasoconstriction of the efferent glomerular arteriole creates stasis in the glomerular capillaries favoring passage of red blood cells into the urine. Second, hypoxic injury occurs in the nephron with subsequent increased glomerular permeability, leading to red blood cell loss into the urine. Direct trauma at any site along the genitourinary system can precipitate bleeding. Bladder irritation or microtrauma (also known as "bladder slap") can result in a runner with a bladder nearly empty of urine, causing hematuria. Under these circumstances, hematuria results from the multiple times the anterior and posterior bladder wall impact each other. Heel strike or march hemoglobinuria appears in runners with mechanical destruction of red blood cells in the heels that exceeds the binding capacity of haptoglobin in the blood and is directly excreted as free hemoglobin.[8]

The other causes of hematuria are diverse and include neoplasm, autoimmune disease, infection, nephrolithiasis, hematologic disorders, and polycystic kidney disease, as well as being drug induced. Infection (e.g., cystitis, urethritis, prostatitis) accounts for 25% of all cases of atraumatic hematuria, with stones representing another 20%, and no cause being found in 10%.[9] The algorithm in Figure 58.3 and the diagnostic tests in Table 58.2 provide a sequential approach to evaluating hematuria in the athlete and should simplify testing, diagnosis, and treatment (Level of Evidence B, systematic review).[8,10,12,13] If an evaluation for hematuria demonstrates unstable vital signs, new-onset hypertension, edema, age over 40, or a history of recurrent gross or microscopic hematuria, then an expedited assessment must follow to find a treatable cause for glomerular disease or to identify a possible urologic neoplasm.[8–10]

The medical history should define any traumatic or atraumatic mechanisms. Events of a recent illness or medication profiles should be reviewed. A genitourinary history should define

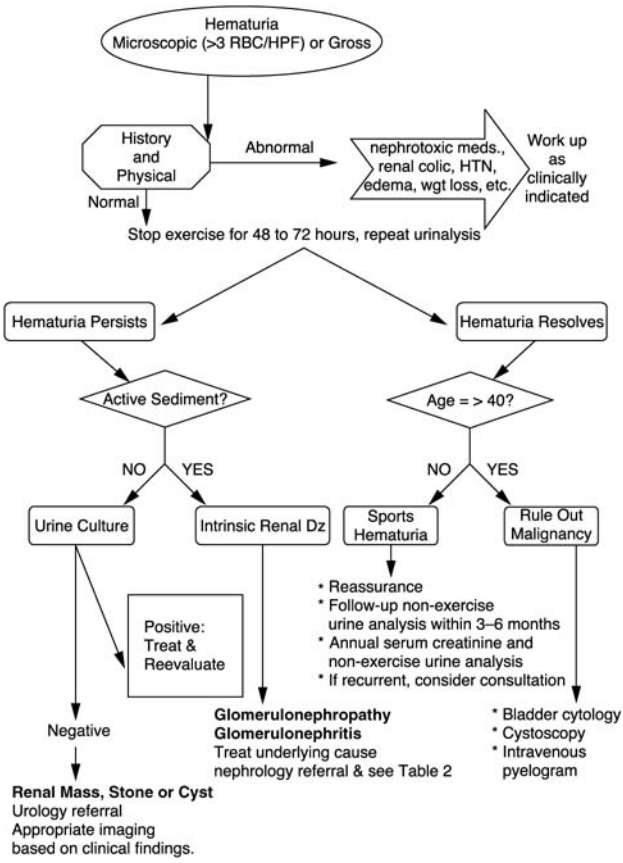


Figure 58.3 Hematuria algorithm.

any associated urologic symptoms and search for evidence of genitourinary tenderness, swelling, or voiding abnormalities. Review of systems should include fever, weight loss, fatigue, menstrual timing, sickle cell status, nephrolithiasis, and connective tissue disease. Family history should be reviewed for bleeding disorders, polycystic kidney disease, and renal disease. Microscopic urinary exam will further assist with red blood cell morphology, and other urinary sediment findings can then suggest the level of genitourinary injury or irritation.

The following circumstances are rarely consistent with benign exercise-induced hematuria, and other causes for the hematuria should be pursued:

- Microscopic hematuria does not resolve with an exercise rest for 72 hours.
- Episodes of hematuria are associated with exertional renal colic pain[11] or hypertension and edema.
- Gross hematuria is present on more than one occasion and not associated with a genitourinary infection or drug interaction.
- The athlete is a man over the age of 40 and has had recurrent episodes of hematuria.

In these instances where the athlete presents with hematuria not consistent with exercise-induced hematuria, the patient should have a preliminary work-up, including a 24-hour urine collection to measure creatinine clearance and urine cytology, urine culture, diagnostic imaging in the form of an intravenous pylogram or renal ultrasound, and a referral to urology for cystoscopy. A renal biopsy to rule out glomerular or renal tubular disorders is best deferred to the nephrologist (Level of Evidence B, systematic review).[8–12]

Treatment for exercise-induced hematuria is based on the suspected pathophysiology of the condition and limited benefits offered by the

TABLE 58.3
Nephrotoxic Medications

Renal Disease	Pathophysiology	Medications
Acute renal failure	Prerenal failure due to decreased renal profusion	Diuretics, NSAIDs, ACE inhibitors, cyclosporine, radiocontrast agents
Renal toxic effect	Acute tubular injury due to a direct toxic effect	Quinolones, aminoglycosides, carbamazepine, tacrolimus
Immune-mediated interstitial nephritis	Acute interstitial nephritis due to immune-mediated inflammation of the interstitium	Penicillins, rifampin, sulfonamides, thiazide, cimetidine, furosemide, NSAIDs, ciprofloxacin
Renal vascular injury	Vascular endothelial injury manifested as hemolytic-uremic syndrome	Conjugated estrogens, tacrolimus, cocaine, quinine
Immune-mediated glomerulopathy	Acute immune mediated inflammation of the glomerulous	Gold, captopril, NSAIDs, penicillamine
Postrenal obstruction, intratubular	Intratubular obstruction due to medication precipitation	Acyclovir, sulfonamides, methotrexate
Postrenal obstruction, ureteral	Ureteral obstruction due to retroperitoneal fibrosis	Atenolol, ergotamine, dihydroergotamine, methyldopa
Chronic renal failure	Chronic interstitial fibrosis with or without papillary necrosis	NSAIDs, acetaminophen, aspirin, lithium

Note: NSAIDs, nonsteroidal anti-inflammatory drugs; ACE, angiotension converting enzyme.
Source: Adapted from Choudhury, D. and Ahmed, Z., *Med. Clin. North Am.*, 81, 705, 1997. With permission.

interventions. No evidence suggests that exercise-induced hematuria causes permanent renal damage. The common medical practice is to return these athletes back to play after the hematuria resolves in 24 to 72 hours. Athletes should be encouraged to maintain adequate hydration before and during exercise. Runners should avoid emptying their bladder completely before exercise to prevent "bladder slap" hematuria and further ensure adequate shoe cushioning to prevent "heel strike" hematuria.[12] Therefore, in the presence of normal renal function and no changes in medical history, an athlete who has recurrent bouts of hematuria that clear with rest may proceed to enjoy the benefits of sport with no limitations. Scheduled monitoring measures should, however, include a microscopic urinary exam, not preceded by exercise, within 3 to 6 months the first year and then repeated annually thereafter with a serum creatinine. Carson and colleagues[13] found that 20% of patients over age 50 with an initial negative evaluation for exercise-induced hematuria had significant lesions within 2 years. (Level of Evidence B, systematic review).[8,10,12]

ATRAUMATIC RENAL INJURY

Nephrotoxicity

Drug-induced renal dysfunction is caused by many common medications. During exercise, the renal endothelial surface is a vulnerable target for drugs and their metabolites. As described earlier, exercise couples diminished renal profusion with a concentrated filtrate that can be further impaired by drug-associated injury to tubular or glomerular structures, allergic interstitial inflammation, and/or vascular compromise. Because the clinicopathologic presentation of a drug-induced nephropathy is variable, its true incidence is difficult to describe.[14] Drugs that are associated with acute and chronic renal failure are listed in Table 58.3.

Rhabdomyolysis

Exercise-induced rhabdomyolysis is a relatively common complication of strenuous exercise, as evidenced by military recruit data. Acute renal failure, in the setting of exertional rhabdomyolysis, has a variable incidence ranging from 17 to 40%. Sinert et al.[15] explained, from their investigation, that several nephrotoxic cofactors (significant hypovolemia and/or aciduria) are required to precipitate acute renal failure with exertional rhabdomyolysis. In the presence of acidic urine (pH below 5.6), myoglobin dissociates into globulin and hematin. Hematin produces free hydroxy radicals that render a toxic effect on the cellular tubular level. Furthermore, in urine below pH 5.0, myoglobin forms casts that occlude the renal

tubules and precipitate acute renal failure.[15,16] Treatment for rhabdomyolysis begins with a strong measure of prevention with attention to factors of acclimatization, rest/work cycles under conditions of heat exposure, and hydration. In the presence of rhabdomyolysis, careful monitoring of urinary production and urine pH will assist with decisions to hydrate the athlete and alkinalyze the urine (Level of Evidence A, randomized controlled trials).[15,16]

SCROTAL MASS AND PAIN

Scrotal pain in an athlete represents many diagnostic possibilities and can be challenging, especially given the serious nature of a misdiagnosis. The primary etiologies for acute traumatic scrotal pain include testicular contusion, testicular rupture, testicular torsion, and scrotal hematoma. Common causes of insidious scrotal pain include epididymitis, testicular appendage torsion, inguinal hernia, and testicular tumor.[17] The medical history should focus on the mechanism and time course of presentation. The physical should follow a focused inspection and palpation of the scrotal anatomy. Scrotal masses should be further described by transillumination as cystic or solid. Examination of the abdomen and groin should rule out referred pain. A cremasteric reflex is rarely intact with testicular torsion. A urinalysis should be performed.[17]

The scrotum is often the subject of blunt trauma, with the right testis being more vulnerable to direct blows presumably because of its position. A kick to the groin in soccer or a fall in cycling, for example, may generate enough force to rupture the testicle. More commonly the energy transfer is more moderate and results in a testicular contusion or a scrotal hematoma. A careful manual exam may assist in differentiating a testicular contusion or scrotal hematoma from a testicular rupture but a more definitive evaluation should follow with an ultrasound. In the interim period, these injuries are acutely treated by scrotal support, ice, scrotal elevation, and analgesics. In the setting of an expanding scrotal hematoma, then immediate surgical consultation is indicated.

Testicular torsion has its own diagnostic challenges. The incidence of testicular torsion is highest during early puberty through the teenage years. When trauma has an association with this injury it is more often minor. Pain onset is acute and tenderness is diffuse, affecting a single testicle. Pain is aggravated with testicular elevation above the symphysis pubis. Focal tenderness at the testicular upper pole is more suggestive of appendiceal torsion. When testicular pain is less than 12 hours in duration and a testicular torsion is suspected by history and physical, the patient is taken for urgent surgical intervention. In cases where testicular pain exceeds 12 hours in duration or the diagnosis is unclear, further diagnostic studies may include a nuclear testicular flow study or color Doppler ultrasonography. Galejs and Kass[17] suggest that institutional preference and improved accuracy have advanced the more timely application of color Doppler ultrasound over that of nuclear scanning (Level of Evidence B, systemic review). Treatment for testicular torsion involves a small mid-line incision. A necrotic testis is removed; a viable testis is fixed with nonabsorbable sutures. Torsion of a testicular appendage is managed over several weeks of scrotal elevation, rest, and NSAIDs.[17]

Epididymitis has an occasional association with trauma with more frequent causes being attributed to sexually transmitted diseases and urinary tract infections. Epididymitis is seen from adolescent years into adulthood. Pain is insidious and localizes to the epididymis; the testis is not tender. Chronic epididymitis that accompanies a urinary tract infection should have a renal/bladder sonogram and a voiding cystourethrogram to rule out structural abnormalities. Epididymitis treatment includes NSAIDs, scrotal elevation, and empiric antibiotic therapy until the urine culture yields an infected organism. If no organism is identified, epididymitis treatment duration is extended 1 week beyond the period of scrotal tenderness resolution, typically totaling 2 to 4 weeks.[17]

Testicular cancer may present as a firm testicular mass. The patient or medical provider, on routine physical exam, incidentally finds the mass, which may be associated with a dull scrotal ache. Testicular cancer represents only 1% of all cancers in males, but one of the bimodal incidence peaks during a common age group for athletes, 15 years to 35 years old. Of testicular tumors, 97% are germinal in origin. A testicular ultrasound is a reliable means to initially evaluate a testicular tumor. A urologic consult should be scheduled in a timely fashion.[18]

GENITOURINARY INFECTIONS

The incidence of urinary tract infections has not been demonstrated in any study to have a higher prevalence rate among athletes; however, athletes may, by virtue of physiologic factors, experience a decrease in natural barriers such as thinner urethral tissues in amenorrheic or postmenstrual

TABLE 58.4
Sexually Transmitted Disease Treatment

Disease	Primary Treatment	Alternative Treatment	Recurrent Treatment
Gonococcal urethritis/cervicitis	Ceftriaxone, 125 mg IM Cefixime, 400 mg PO[a]	Ciprofloxacin, 500 mg PO[a] Ofloxacin, 400 mg PO[a]	N/A
Non-gonococcal urethritis/cervicitis or chlamydia	Doxycycline, 100 mg BID × 7 days Azithromycin, 1 g PO[a]	Erythromycin base, 500 mg PO QID × 7 days Levofloxcin, 500 mg PO QD × 7 days	Metronidazole, 2 g PO[a] Erythromycin base, 500 mg QID × 7 days
Herpes virus	Acyclovir®, 400 mg PO TID × 7–10 days	Famciclovir, 250 mg PO TID × 7–10 days Valacyclovir, 1 g PO BID × 7–10 days	Acyclovir, 400 mg PO TID or 800 mg PO BID × 5 days Famciclovir, 125 mg PO BID × 5 days Valacyclovir, 1 g PO QD × 5 days
Herpes virus suppression	Acyclovir®, 400 mg PO BID	Famciclovir, 250 mg PO BID Valacyclovir, 500 mg to 1 g PO BID	N/A
Genital warts or human papillomavirus	Imiquimod or podofilox, 0.5% solution or gel applied 3 days a week for 30 days	Office-based treatments include cryotherapy each week or podophyllin resin, 10–25%	N/A

[a] Single-dose regimen.

Note: PO, by mouth; IM, intramuscular; N/A, nothing applies. Use these regimens in nonpregnant adult patients.

Source: Centers for Disease Control and Prevention, 2002 guidelines for treatment of sexually transmitted diseases, *MMWR*, 51(RR-6), 1, 2002. With permission.

women. Poor hydration practices and voluntary urinary retention in athletes can lead to urinary stasis and/or urinary reflux that can form the nidus of infection. Finally, some sports, such as bicycling, generate recurrent incidental injury to the urethra, prostate, and bladder that can establish an inflammatory process that may be difficult to differentiate from infection.[19]

Sexually transmitted diseases (STDs) comprise a diverse group of infectious diseases that incorporate many pathogens and a varied clinical picture. Athletes are among the young risk takers of the sexually active population. Indiscriminate sexual behavior puts them at greater risk to contract sexually transmitted diseases.[19,20] It is estimated that 1 in 4 Americans will experience an STD. Human papillomavirus has the highest prevalence in the United States followed by genital herpes. Most concerning is the burden of asymptomatic *Chlamydia trachomatis* and *Neisseria gonorrhoeae* infections. Turner et al.[21] reported that, over a 1.5-year period ending in 1998, 7.9% of the urine specimens for a cohort of 579 adults ages 18 to 35 years had either an untreated gonococcal or chlamydial infection. Information such as this reveals the hidden epidemic of STDs in the United States. See Table 58.4 for treatment selections for the above-mentioned STDs (Level of Evidence B, systemic review).[19,22]

SOLITARY KIDNEY

The incidence of a solitary kidney (congenital or acquired) is estimated in the American population to be 1/1100–1800. The results of a questionnaire sent to the membership of the American Medical Society for Sports Medicine in 1994 reported that 237 of the 438 respondents (54.1%) indicated that they would allow full participation in sports for an athlete with a solitary kidney after discussion of the possible risks.[23] In 2001, the American Academy of Pediatrics published a policy statement regarding medical conditions affecting sports participation. This policy statement proposed that an athlete with a solitary kidney could receive a qualified "yes" for sports participation after completing an individual assessment for contact, collision, and limited-contact sports. Renal injuries in sports are uncommon, but not rare; sports are reported as a cause for renal injury in 4 to 28% of such cases.[24] More importantly, the

loss of a kidney from a sports accident has been described as extremely rare. The benefits of sports participation should be examined in relationship to the risks in the decision-making process. It is imperative that an open and full discussion occurs among all the parties involved.[24]

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59 HEMATOLOGIC CONCERNS IN THE ATHLETE

William B. Adams

HEMATOLOGY IN THE ATHLETE

Exercise *per se* does not predispose to hematologic disease states. Although athletes as a group tend to be healthier, they are still susceptible to the same hematologic diseases as non-athletes; however, symptoms from hematologic disturbances may present earlier and at lower severity, often manifesting as impaired physical performance.[1,2] Maximal or prolonged exertion efforts typically cause transient changes in several hematologic indices. Regular endurance and altitude training generally result in more sustained alterations of hematologic parameters.[3] Dietary inadequacies, not uncommon in athletes, may cause hematologic problems due to a deficit of calories or critical nutrients.[4]

HEMATOLOGIC DISORDERS IN THE ATHLETE

Anemia

Anemia is the reduction of total red blood cell (RBC) volume (hematocrit, or Hct) or hemoglobin (Hgb) concentration below normal values. It is a common clinical condition with a multitude of causes.[5] The prevalence of anemia for males in the United States ranges from 6/1000 below age 45 to 18.5/1000 males ages 75 and above. For women of all ages, the prevalence is 30/1000.[6] While athletes tend to be healthier than the general population, they may have a slightly higher prevalence of anemia from certain nutritional deficiencies, particularly those trying to restrict weight or those following special diets that are deficient in iron, vitamins, or calories.[3,7] Anemia arises from either excessive loss or inadequate production of RBCs, or a combination of both. Symptoms and physical manifestations depend upon decrements in RBC volume and oxygen delivered to tissues, the rate at which these changes occur, and the cardiopulmonary compensatory capacity.[5,8]

Athletic Pseudoanemia (Sports Anemia)

Sweat losses and intravascular fluid shifts during sustained aerobic exercise may decrease plasma volume 5 to 20%. Trained endurance athletes tend to have a greater reduction in plasma volume during exercise due to greater sweat losses which

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is offset by a physiologic plasma volume expansion in the resting or pre-exercise state. Red cell production is increased with regular endurance training; however, this increased red cell mass is offset by a greater expansion of plasma volume. Consequently, a slight reduction in Hgb and Hct levels occurs in the resting state. This is not a true anemia but rather a physiologic adaptation that promotes increased cardiac output and enhanced oxygen delivery to tissues and protects against hyperviscosity. Hence, it is termed *athletic pseudoanemia* or is sometimes called *sports anemia*.[3,9,10] Hgb values typically run 0.5 g/dL lower for athletes pursuing moderate-intensity training and 1.0 g/dL lower for elite-level athletes.[10] This hemodilution from conditioning is temporary, however, and may resolve within days of terminating endurance-level training.[3] Diagnosis may be confirmed by testing the athlete after several days of rest from training or can be inferred from complete blood count (CBC) testing revealing normal RBC indices and red cell distribution width (RDW) with a normal reticulocyte count and normal serum ferritin level (Level of Evidence B, clinical cohort study). In athletes, normal ferritin levels may be as low as 12 μ g/L, particularly with high-intensity training.[7] If iron-deficiency anemia is in question, a brief trial of oral iron supplementation with a repeat reticulocyte count at 1 to 2 weeks may provide the answer. A rise in the reticulocyte count confirms iron deficiency as the etiology of the anemia (Level of Evidence C, expert opinion).[11]

Iron-Deficiency Anemia

Iron-deficiency anemia is the most common cause of true anemia in the athlete as in the nonathlete.[9,10] It occurs more often in female athletes mostly due to menstrual losses coupled with inadequate consumption of meat or other sources of iron.[7,9] Laboratory testing reveals a low Hgb and Hct with low mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH). RDW is increased unless iron deficiency is chronic. Peripheral smear reveals hypochromic microcytic cells with a low to normal reticulocyte count. Serum ferritin levels that are low (≤12 μ g/L) better reflect total body iron content, as serum iron levels are unreliable. Total iron binding capacity (TIBC) and transferrin saturation (serum iron × 100/TIBC) more accurately reflect iron status. In isolated iron deficiency, TIBC tends to be elevated, while transferrin saturation tends to be low (particularly <16%).[5,7,8] In evaluation of iron deficiency anemia it is imperative to determine the cause of the deficiency in order to best effect therapy and avoid overlooking potentially serious conditions. Iron replacement should continue until 6 to 12 months after anemia has resolved (Level of Evidence C, expert opinion).[11]

Blood Loss

Acute heavy bleeding may cause anemia before the onset of iron deficiency. Typically, the diagnosis is obvious from history or exam findings of gross blood, melena, or hypovolemia. Bleeding contained within tissues or body cavity may be less obvious, particularly in the retroperitoneal space. In the absence of fluid administration, Hgb and Hct (both concentration values) are initially normal. Platelet counts transiently drop but become elevated within an hour if no ongoing hemorrhage is present. Over the ensuing days, Hgb and Hct values decline with plasma expansion from endogenous reservoirs. RBC indices remain normal until 3 to 5 days later when a reticulocytosis occurs, increasing MCV and RDW. Bilirubin levels are normal unless internal bleeding is present. With internal bleeding, unconjugated bilirubin and lactate dehydrogenase (LDH) rise as in hemolysis; however, no indicators of hemolysis can be found on peripheral smear.[5,12] If blood loss is slow and insidious, anemia may not manifest until iron stores are depleted. Often this occurs with gastrointestinal (GI) bleeding and with menstrual blood loss in women; however, this situation may be revealed by a reticulocytosis with concomitant increase in RDW before iron stores are depleted.

Gastrointestinal Losses

(See Chapter 57.) Gastrointestinal bleeding is a very common and often serious cause of anemia. Accordingly, a stool occult blood test is indicated in initial assessment of any anemia work-up (Level of Evidence C, consensus opinion).[6] GI bleeding may arise from the mucosa due to peptic ulcer disease or medication use (e.g., nonsteroidal anti-inflammatory drugs, or NSAIDs), vascular anomalies, inflammatory bowel diseases, ischemic syndromes, infection, diverticuli, or tumors. In prolonged endurance events, low-grade gastrointestinal bleeding is tremendously common. The source of this bleeding is seldom detectable and is theorized to arise from acute transient ischemia or mechanical contusion (e.g., cecal slap syndrome).[3,4,7,11] Athletes frequently pursuing long-distance running and those using NSAIDs may accrue enough cumulative blood loss to impact RBC mass.[3,10,11] In the absence of

this or other pathology, however, exercise-associated GI bleeding is seldom significant enough to cause anemia.[1] Regardless, any GI bleeding warrants thorough investigation to rule out serious conditions (Level of Evidence C, consensus opinion).

Menstrual Losses

(See Chapter 17.) Blood loss from menstruation may be significant, particularly if a woman is prone to heavy or frequent menses or her diet is inadequate to compensate for cumulative menstrual losses. Characteristics of excessive flow are a requirement for 12 or more pads throughout menses, passage of clots beyond the first day, or duration of flow greater than 7 days.[5] Quantitation of menstrual flow and assessment of adequacy of iron replacement for chronic menstrual losses should be considered in the evaluation of all women athletes with anemia. Treatment focus is on reduction of menstrual flow if excessive and on iron replacement (Level of Evidence C, consensus opinion).

Hematuria/Hemoglobinuria

(See Chapter 58.) Bleeding from the urologic system is not typically of a volume to produce anemia. Some runners experience hematuria that is thought to arise either from increased filtration of RBCs into the urine (as a result of vascular shunting) or from bladder wall contusion during prolonged running. Any gross or microscopic hematuria, however, requires evaluation with particular concern to rule out urologic tumors (Level of Evidence C, consensus opinion).[3] Hemoglobinuria secondary to hemolysis is occasionally seen in long-distance running. It has been attributed to footstrike hemolysis but typically is a not a significant source of blood or iron loss (see exertional hemolysis, below).[7,11]

Exertional Hemolysis (Footstrike Hemolysis)

Exertional hemolysis is a condition of intravascular destruction of RBCs in association with various exertional activities. Originally described as march hemoglobinuria in foot soldiers in the late 1800s, it was thought arise from the footstrike causing compression of capillaries and rupturing RBCs; however, it is also seen in swimmers, rowers, and weightlifters, although usually to a much lesser degree.[3,9,11] It is now hypothesized that intravascular turbulence, acidosis, and elevated temperature in muscle tissues may be causative factors, as well.[10] Typically, hemolysis is not significant enough to affect CBC parameters; however, if enough cumulative hemolysis occurs, the reticulocyte count RDW and MCV may be elevated and haptoglobin levels reduced. Transient hemoglobinuria may occur if hemolysis exceeds capacity of serum haptoglobin to bind up released hemoglobin (approximately 20 cc of blood).[3,9,10] Generally, no treatment is necessary. Reducing impact forces to the feet (e.g., improved shoe cushioning, softer running terrain) may benefit some, particularly elite level runners (Level of Evidence C, consensus opinion).[10]

Sickle Cell Trait

Sickle cell trait (SCT) is a common condition present in 8% of blacks in the United States. It typically does not cause anemia and seems to have little impairment of athletic performance;[1,13] however, SCT may confer heightened risk of complications with exercise at altitude, in heat stress environments, in settings of rapid conditioning, or under conditions of sustained maximal exertion efforts. Individuals with SCT may also manifest mild microscopic hematuria which appears to occur independent of physical exertion.[13,14] This hematuria is rarely significant, but should be attributed as hematuria from SCT only after other etiologies are ruled out (Level of Evidence C, expert opinion).[13,14] Hypoxic environments, particularly altitudes above 10,000 feet, may provoke sickling and cause a clinical picture similar to sickle cell anemia. Exertion at altitudes of 5000 feet or more may produce enough hypoxic and metabolic stress to induce sickling and its sequelae.[13,14]

Individuals with sickle cell trait may be at higher risk of exertion-related rhabdomyolysis, particularly in heat stress conditions. Retrospective studies of recruit training populations indicate that sickle cell trait may confer increased risk of sudden death and exertional rhabdomyolysis. Although the total incidence is low, the occurrence of sudden death and exertional rhabdomyolysis in blacks with sickle cell trait was 30 times higher than in those without and 100 times higher than non-black recruits without sickle trait.[14] While causative factors are difficult to discern, it is suggested that rapidly advanced conditioning training and sustained maximal exertion efforts increase the risk.[13,14]

In light of exertion related risks, it may be prudent to screen individuals for sickle trait who are in higher prevalence groups or those with a family history of sickle cell disease or trait. With known sickle trait, avoidance of hypoxic environments and strict adherence to heat illness prevention are crucial. Also avoidance of rapid accelerated

training and maximal sustained exertion in unconditioned sickle trait individuals may be warranted (Level of Evidence C, expert opinion).

Other Disorders Causing Anemia

Anemia may result from other several other conditions or as a consequence of various disease processes. These may manifest in the form of accelerated red cell destruction or hemolysis or through impaired erythropoeisis. Details regarding diagnosis and evaluation of these may be found in Reference ???.

EVALUATION OF HEMATOLOGIC DISORDERS

In the absence of pathologic indicators, it may be prudent to initially repeat the CBC after the athlete has rested for several days to eliminate acute transient hematologic perturbations as cited above (Level of Evidence C, expert opinion).[5,15] Ideally, blood drawn for hematologic study should be collected when the patient is normally hydrated and in a calm, well-rested state with no recent food intake or use of caffeine, nicotine, or other stimulants. Stress, emotional disturbance, and stimulants may artifactually increase the white cell and platelet counts. Dehydration and overhydration may alter all parameters through hemoconcentration or dilutional effects. The possible influence of these factors should be considered in analyzing results of blood collected under these conditions.[5,15]

Evaluation of Anemia

History

The evaluation of anemia in the athlete should start with a search for historical clues, symptoms, and physical signs that point toward a specific etiology. Historical factors to solicit include character and duration of symptoms and whether onset was abrupt or insidious. Prior history of hematologic problems, malignancy, chronic diseases, or any family history of blood disorders is important as well. Assessment of calorie intake vs. expenditure, endeavors at weight control, and use of exclusionary diets may reveal problems of caloric inadequacy or deficiency of critical nutrients. Use of nutritional aids, supplements, ergogenic agents, medications (particularly NSAIDs and inhibitors of DNA synthesis or folic acid), tobacco, or alcohol may be contributory as well.[5,15] Anemia classically presents with fatigue or malaise, but athletes often complain of decline in performance or endurance or an elevated heart rate.[1] Reports of petechiae, bruising, or bleeding problems; abdominal discomfort; jaundice; alteration of bowel patterns; dyspnea; fever; or pica may suggest particular etiologies. It is important to seek out indicators of GI bleeding as this is a common and oftentimes serious cause of anemia. Menstrual blood loss commonly contributes to anemia in women and should be quantified. Chemical exposure through work or hobbies may have hemolytic or hematopoietic effects as well. [5]

Examination

The physical examination should assess overall health and nutrition as well as hemodynamic status, particularly orthostasis. Pallor and relative or absolute resting tachycardia indicate significant anemia. Findings of scleral icterus, jaundice, and splenomegaly suggest a hemolytic process. Bruising and petechiae may indicate a coagulation or platelet disorder. Certain integument changes may characterize dietary deficiencies or hypothyroidism. Findings of adenopathy, foci of skeletal tenderness in limbs or sternum, and abdominal or pelvic masses may suggest underlying malignancy. Signs of chronic diseases (particularly renal and hepatic), infection, endocrinopathies, and malignancies particularly should be sought out along with stool testing for occult blood (Level of Evidence C, consensus opinion).[5,8]

Studies

Evidence of heavy bleeding, severe hemolysis, malignancy, or profound deficiency in one or more hematologic cell lines necessitates specialized testing and specialist referral early on; otherwise, if the history, physical examination, and testing for occult bleeding do not point toward a specific etiology, a systematic laboratory evaluation should ensue (Level of Evidence C, expert opinion).[5] In analyzing laboratory studies, verify that blood was not collected under conditions that spuriously alter hematologic parameters, as noted above. Repeating studies after several days of rest may preclude much unnecessary work-up and anxiety.[5,15] Stool occult blood testing is indicated early as GI bleeding is a common cause of anemia;[16] otherwise, initial work-up should start with a CBC, differential count, peripheral smear review, and reticulocyte count. These studies allow classification of anemia according to conditions of excessive loss (bleeding or hemolysis) or inadequate production (ineffective erythropoeisis). Using this scheme with subsequent subcategorization according to RBC size (MCV) and hemoglobin content (MCH) allows for a more focused approach to determine the etiology of

the anemia. If initial assessment reveals gross or occult bleeding, the evaluation is directed toward identification of the source and implementation of corrective measures (to include iron and blood replacement as indicated).

The first step in assessing anemia is to determine if the condition is one of excess blood loss or inadequate RBC production. This requires determination of the reticulocyte count and calculation of the reticulocyte production index (RPI). The RPI accounts for expected variance in reticulocyte percentage for different hematocrit values; hence, it is a more reliable parameter. The formula for determination of the RPI is:

Reticulocyte Production Index

$$\text{Patient's Hct RPI} = \frac{\text{Reticulocyte percentage}}{\text{Reticulocyte maturation time (days)}} \times 0.45$$

$$\frac{\text{Reticulocyte maturation time}}{} = \begin{cases} 1.0 \text{ for Hct of } 0.45 \\ 1.5 \text{ for Hct of } 0.35 \\ 2.0 \text{ for Hct of } 0.25 \\ 2.5 \text{ for Hct of } 0.15 \end{cases}$$

RPI values of 3 or more indicate increased erythrocyte production as seen with blood loss (see Figure 59.1).[5]

Elevations of serum bilirubin (particularly unconjugated), LDH, and urobilinogen with decreased haptoglobin indicate hemolysis.[5] In this setting, determine the cause of hemolysis and implement corrective measures (see Reference 17). Normal values for these tests indicate bleeding and should be followed with investigations to identify and treat the bleeding source. Internal bleeding, however, may mimic hemolysis, yielding the same chemistry disturbances as RBCs are broken down and reabsorbed. The difference is distinguished by history, examination findings, and a paucity of fragmented cells on peripheral smear.[5,8,12,17]

Anemia with RPI values less than 2 indicates impaired erythropoeisis (see Figure 59.2). In this setting the next step involves using CBC results to subcategorize anemia according to erythrocyte indices of mean corpuscular volume and mean corpuscular hemoglobin. The MCV allows classification of the anemia as normocytic (normal MCV), microcytic (low MCV), or macrocytic (elevated MCV). Decreased MCH indicates hypochromia as seen in prolonged iron deficiency. It is important to realize that these parameters are averages and may not adequately reflect the clinical state early on when morphologic variation within the RBC population is averaged out. This situation, however, is revealed by an increase in the red cell distribution width, which reflects size variance in the red cell population and can identify acute alterations in RBC morphology long before the MCV is affected (e.g., early iron-deficiency anemia). The peripheral smear also may reveal morphologic characteristics in RBCs or other cell lines indicative of certain pathologic processes (e.g., hematologic malignancies, hemolysis, hemoglobinopathies).[5,8,15]

Another useful study at this stage, particularly in evaluating microcytic anemia, is the serum ferritin level (see Figure 59.3). In the absence of concomitant disease processes, serum ferritin reflects total body iron stores. Serum ferritin tends to be low in iron deficiency (typically, <12 μg/L); however, inflammatory disease processes may elevate ferritin levels into the normal range, masking diagnosis of iron deficiency.[8] Ferritin levels tend to be elevated in thalassemia, anemia of chronic disease, liver disease, and various malignancies. Determination of TIBC and transferrin saturation (serum iron × 100/TIBC) may further aid in determination of etiology. TIBC tends to be elevated in iron deficiency and decreased in anemia of chronic disease. Transferrin saturation tends to be lower in iron deficiency than anemia of chronic disease, particularly for values <16%.[8] Various etiologies of microcytic anemia are listed in Table 59.1.

Macrocytic anemia may be either a megaloblastic or nonmegaloblastic anemia (Figure 59.4). The former typically results from deficiency of B12 or folate, although intrinsic defects in DNA synthesis and use of drugs that inhibit folate or DNA synthesis may be at fault as well. Nonmegaloblastic anemias typically result from alcoholism, liver disease, or hemolytic anemia. Megaloblastic anemia tends to manifest higher MCV values and is often associated with pancytopenia and hypersegmentation of neutrophils and oval macrocytes on peripheral smear. LDH is significantly elevated as well. Serum or erythrocyte B12 or folate assays help differentiate between these diagnoses. In unclear situations, a bone marrow examination may be necessary.[8] Table 59.2 lists many etiologies of macrocytic anemia.

A normocytic anemia may be a mild manifestation of systemic disease, an anemia in transition to becoming macrocytic or microcytic, or a state where concomitant conditions yield mixed microcytic and macrocytic erythrocyte populations with

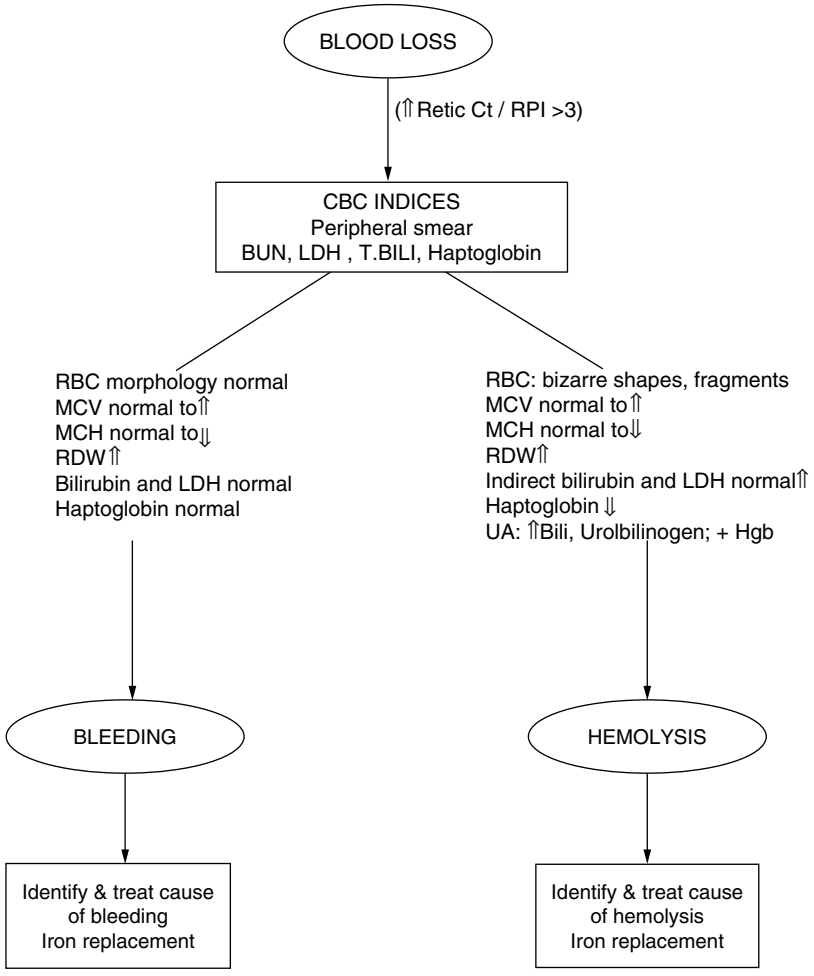

Figure 59.1

normal indices due to averaging. The latter situation would be apparent from evaluation of RDW and review of the peripheral smear. Normocytic anemia may represent early acute hemolysis or bleeding. These states should be distinguishable from clinical exam findings and laboratory results as detailed above (Figure 59.1); otherwise, the remaining etiologies fall into the category of impaired erythropoiesis arising from impaired marrow activity (hypoplastic or aplastic anemia, leukemia and similar marrow infiltrative diseases) or decreased erythropoietin activity, as seen in renal and liver disease, endocrinopathies, and severe malnutrition. Also, anemia of chronic disease often manifests a normocytic anemia. This condition is characterized by elevated ferritin levels. Intrinsic marrow diseases are typically characterized by pancytopenia and immature or bizarre morphologies on peripheral smear. These are confirmed by bone marrow biopsy.[8] Renal, liver, and endocrine diseases should be identified in clinical evaluation but may be missed if symptoms are mild or more insidious in development. Figure 59.5 outlines an approach to evaluating normocytic anemia and Table 59.3 lists common etiologies (Level of Evidence C, expert opinion).[5,8]

Erythrocythemia (Polycythemia) and Erythropoietin

Red blood cell mass may be increased as a physiologic response to hypoxic stress or disease processes or may be induced by drug use. Smoking, carbon monoxide exposure (e.g., ice rinks), and training at altitude may also increase RBC mass in athletes. A spurious erythrocytosis may also arise from transient plasma volume contraction

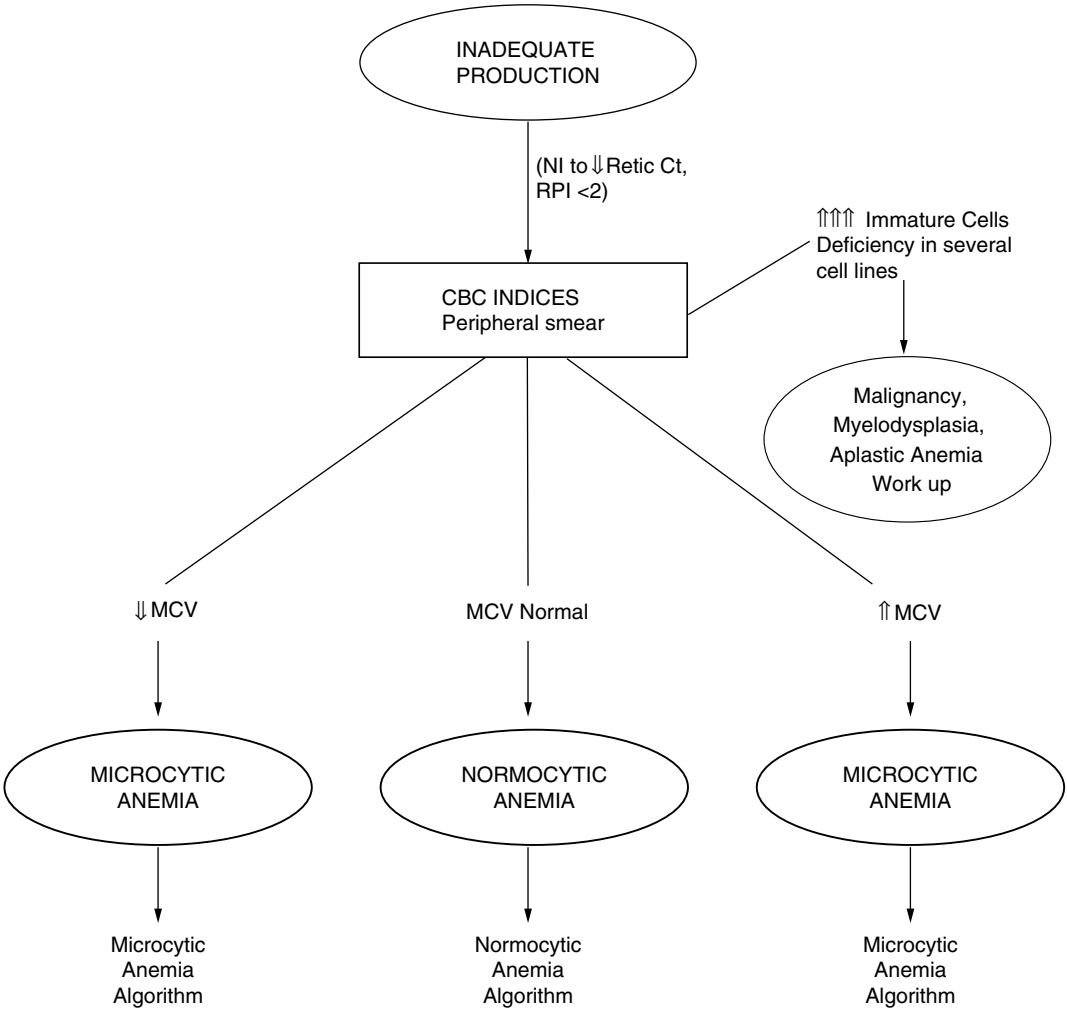

Figure 59.2

(e.g., exercise, dehydrated status).[18] True polycythemia arises from conditions of excess RBC production, either as part of a hyperplastic marrow response (polycythemia vera) or as a secondary response to excess erythropoietin production (secondary polycythemia). Polycythemia vera is a myeloproliferative disorder involving trilineage marrow hyperplasia. Thus, the elevations of RBC mass are often with concomitant leukocytosis and thrombocytosis. It is typically characterized by low erythropoietin levels in the presence of markedly elevated hematocrit. These patients require regular phlebotomy to prevent a hyperviscosity state.[19] Secondary polycythemia results from elevated erythropoietin. This occurs as a response to hypoxia or reduced oxygen delivery to tissues in cases of hemoglobin variants with excessively high oxygen affinity. This is characterized by elevated RBC and erythropoietin levels with normal WBC and platelet counts.

WHITE BLOOD CELL LINE ABNORMALITIES

Strenuous or prolonged vigorous exercise may produce acute profound perturbations of white blood cell (WBC) populations. This effect, however, resolves with rest and is not typically associated with persistent abnormalities of white cell lines. Various drugs may either elevate or depress WBC production, as may infection. Persistent leukopenia may be indicative of human immunodeficiency virus infection or marrow disorders. Some populations (e.g., black males) may manifest a mild neutropenia that is non-pathologic.[15] If blood

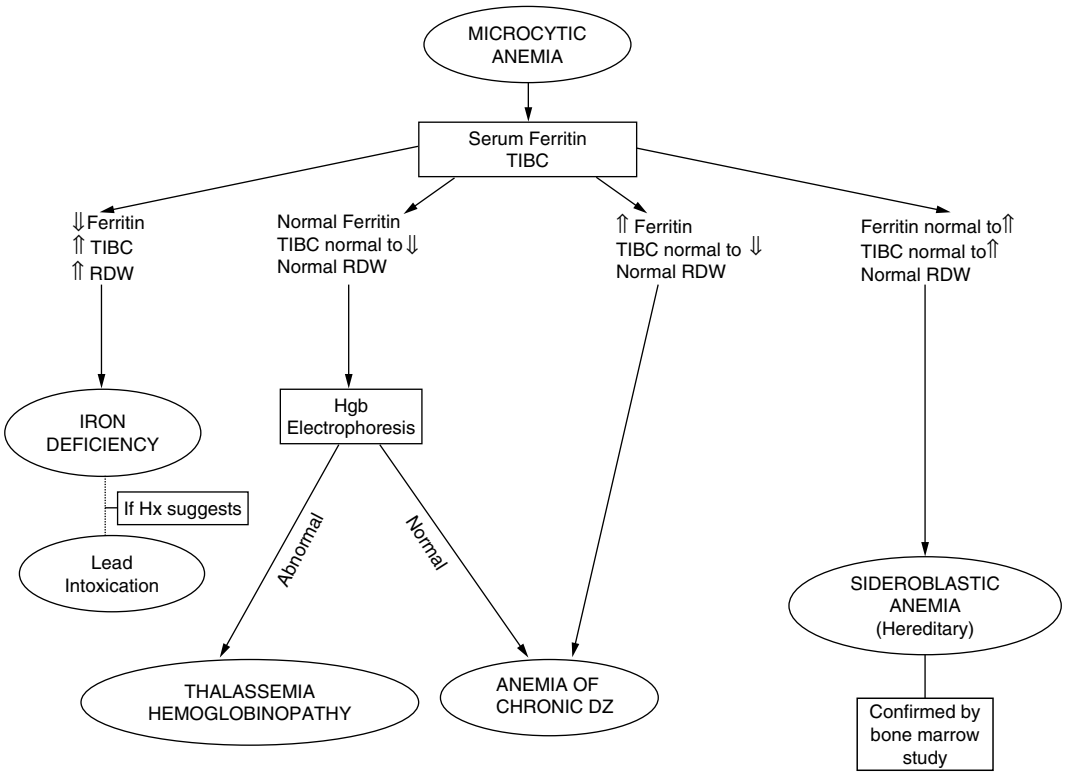

Figure 59.3

TABLE 59.1
Causes of Microcytic Anemia

Iron-deficiency anemia
Anemia of chronic disease
Disorders of iron metabolism
Disorders of globulin synthesis (thalassemias)
Disorders of porphyrin and heme synthesis
Sideroblastic anemia
Lead intoxication

work indicates a pathologic alteration of the WBC population, examination should include a thorough assessment of lymphatic and hematologic systems with investigation for infectious, toxic, or oncologic causes. Readily treatable etiologies such as infection are addressed as indicated; otherwise, early referral to a hematologist for bone marrow assessment may be necessary, particularly if the etiology is unclear or profound leukopenia, leukocytosis, or disturbances of other cell lines are present suggestive of hematologic malignancy.[20]

ABNORMALITIES OF PLATELETS AND COAGULATION

Exercise, particularly endurance activities, seems to have a net neutral effect on platelets and coagulation. Certain drugs, toxins, autoimmune disorders, infections, malignancies, and other conditions that trigger disseminated intravascular coagulation (DIC) may produce thrombocytopenia ranging from mild to severe[20] Acute development of petechiae, bruising, and bleeding problems should prompt investigation for etiologies in these areas. Long-standing history of mild bleeding or bruising problems may indicate von Willebrand's disease or mild factor VIII or IX deficiency. Also, diets deficient in green vegetables may manifest coagulopathy due to impairment of vitamin-K-dependent factors.[20] Evaluation of platelet and coagulation disorders focuses on identification of causative conditions as listed above. Laboratory assessment should start with a CBC with peripheral smear looking for abnormalities in all hematologic cell lines. Coagulation studies (prothrombin time, PT; partial thromboplastin time, PTT; and international normalized ratio, INR) should be conducted as well. If the

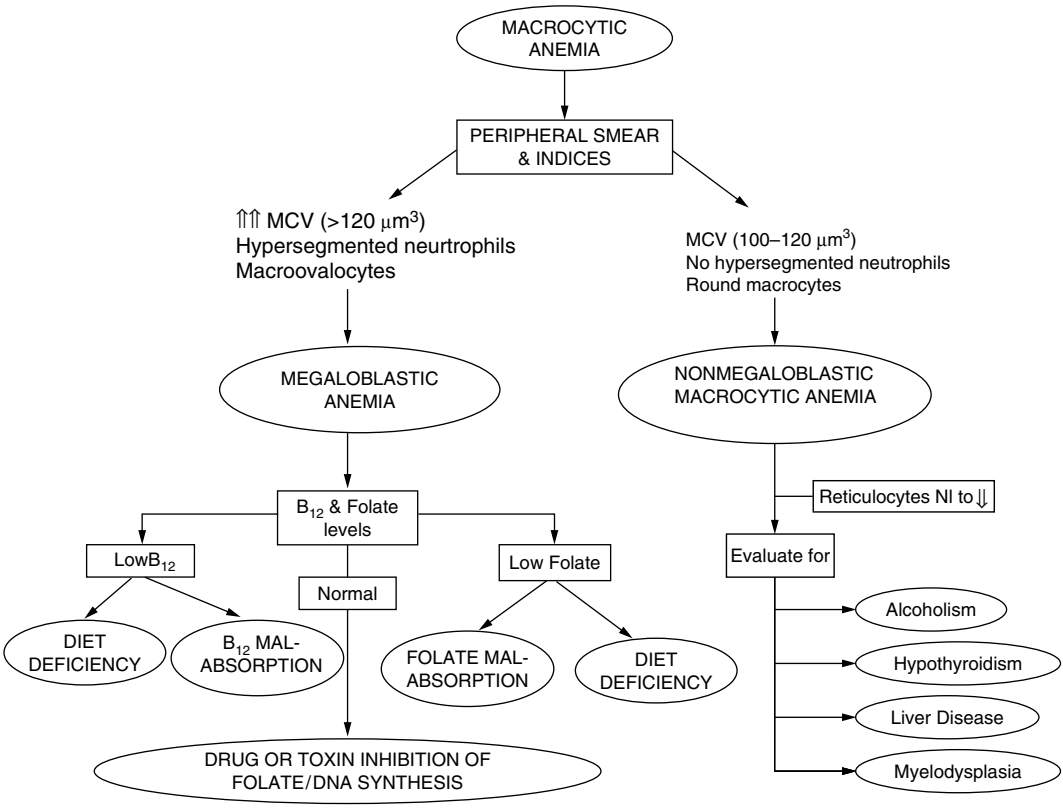

Figure 59.4

clinical picture suggests DIC (low platelets, fragmented RBCs, prolonged coagulation times), confirmatory testing to include fibrinogen, fibrin split products, and D-dimer should be added.[20] Thrombocytosis is often a transient condition, typically a manifestation of an acute response to physiologic stress. Transient isolated thrombocytosis is rarely of significance. Persistent thrombocytosis should prompt investigation for infection, inflammatory disorders, malignancies, or other hyperproliferative disorders (e.g., polycythemia vera, myeloproliferative diseases).[19]

SPECIAL CONSIDERATIONS

Exertional Rhabdomyolysis

Rhabdomyolysis is a condition of skeletal muscle breakdown with release of myocyte contents into the circulation. Biochemically, muscle injury causes a release of myoglobin and muscle enzymes (creatine phosphokinase, LDH, transaminases). Severe states with a large volume of muscle damage typically cause electrolytes disturbances (potassium, phosphate, and calcium) plus extracellular fluid shifts into injured tissues.[21–23]

TABLE 59.2
Causes of Macrocytic Anemia

Vitamin B_{12} deficiency
Folate deficiency
Combined B_{12} and folate deficiency
Disorders of DNA synthesis (inherited)
Alcoholism
Drug or toxin inhibition of DNA synthesis
Erythroleukemia
Blood loss (hemolysis or hemorrhage)
Liver disease
Hypothyroidism
Chronic obstructive pulmonary disease (COPD)
Myelodysplastic anemia
Myelophthisic anemia
Acquired sideroblastic anemia

Rhabdomyolysis may arise from a variety of insults (e.g., drug or toxin exposure, infection, ischemia, direct trauma such as crush injury or

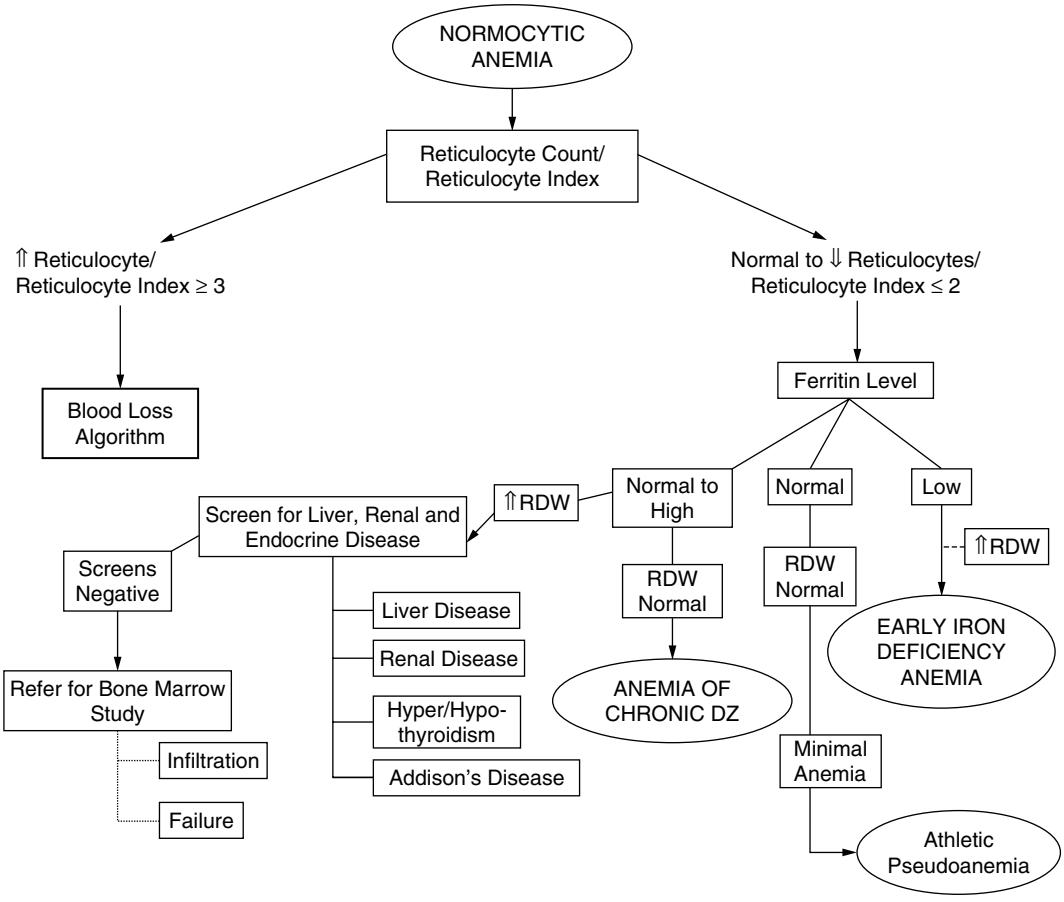

Figure 59.5

TABLE 59.3
Causes of Normocytic Anemia

Blood loss (early)
Hemolysis (early)
Anemia with impaired marrow response
Aplastic/hypoplastic anemia
Leukemia
Multiple myeloma
Myelodysplastic anemia
Early iron deficiency
Renal disease
Mixed anemia (iron deficiency/thalassemia and megaloblastic anemia)
Anemia of chronic disease
Malnutrition

electrical shock, and heat stroke).[21–23] Excessive overload as in weightlifting can produce rhabdomyolysis of an isolated muscle or muscle group. Typically, this is self-limited, rarely manifesting systemic effects beyond the involved muscle. *Exertional rhabdomyolysis* is the term applied to rhabdomyolysis associated with vigorous exercise; it is most frequently seen in running or prolonged exertion activity and often associated with exertional heat illness.[14,22–24] Certain individuals have higher susceptibility to exertional rhabdomyolysis, particularly those with underlying muscle enzyme deficiencies or metabolic diseases such as diabetes or thyroid disease.[22,23,25] An increased risk of severe rhabdomyolysis is associated with sickle cell trait as well. Often this occurs in conjunction with exertional heat illness (particularly heat stroke) but occasionally manifests in settings of rapidly accelerated physical

training and events involving sustained maximal exertion (see Sickle Cell Trait section, above).[13,14,24] Alcohol consumption, infection, dehydration, preexisting electrolyte disturbances, and chronic acidosis also enhance susceptibility to exertional rhabdomyolysis.[21–23]

The spectrum of exertional rhabdomyolysis ranges from mild muscle injury with negligible symptoms and systemic effects to fulminate cases with large muscle mass injury, severe metabolic derangements, disseminated intravascular coagulation, and death.[14,21–24] Many cases fall between these extremes, with symptoms and laboratory studies indicative of mild to moderate injury. While not at imminent risk, these individuals may incur renal injury from myoglobin release and be susceptible to severe rhabdomyolysis if injured muscles are overtaxed prior to completion of healing. The renal toxicity from myoglobin may correlate with both total myoglobin load and duration of renal tubule exposure;[21] therefore, patients with myoglobinuria or myoglobinemia must be treated with aggressive hydration to maintain high urine output until the myoglobin has cleared (Level of Evidence C, consensus opinion).[22,23]

Management of rhabdomyolysis focuses on recognizing the occurrence of significant myocyte injury, determining the magnitude of injury, and initiating interventions appropriate to the degree of injury. Clinical indicators of concern are severe muscle pain or weakness much greater than expected. Symptoms may start off being relatively mild but progress in intensity in subsequent hours. Concomitant occurrence of dark urine indicates myoglobinuria. Initial laboratory studies should include basic electrolyte panel ("Chem 7"), creatine phosphokinase (CPK), transaminases, LDH, uric acid, CBC, and urinalysis with microscopy. In more severe cases, calcium, phosphate, prothrombin time, partial thromboplastin time, fibrinogen, and fibrin split products should be added. It is important to note that muscle enzyme abnormalities often peak 1 to 2 days after the injury. Urinalysis findings of positive hemoglobin with no RBCs are used as indicators of myoglobinuria, as myoglobin studies are not quickly available in most settings.[22,26] Muddy casts indicate heavy myoglobin load and likely renal toxicity.[21]

Severity of rhabdomyolysis is gauged initially by magnitude of symptoms and perturbations of blood chemistries. Extreme pain, collapse during exertion, and early electrolyte shifts with acidosis are ominous indicators. In the presence of heat stroke, mental status alterations are typical, with multisystem toxicity manifesting early.[21,24,26] Assessment for the presence and resolution of myoglobinuria is important, as myoglobin-associated renal failure may occur even with mild symptoms.[21–23] Initial treatment in all cases of rhabdomyolysis is hydration (Level of Evidence C, consensus opinion).[22,23]

Mild cases manifest minimal symptoms that quickly resolve. CPK levels remain low (typically, ≤3000 IU/L) with no other laboratory abnormality. If these individuals remain asymptomatic, they may be treated with oral rehydration and rest with return to activity the next day. With more prominent symptoms, rapid IV hydration with 2 L isotonic fluids is indicated. If heat illness is present, rapid cooling measures must be implemented. The patient should be reassessed as fluid bolus is completed and laboratory studies become available. The patient with near or complete resolution of symptoms, modest muscle enzyme elevations (e.g., CPK, 3000–10,000; transaminases less than twice normal), and otherwise normal studies may be released with continued oral hydration and restricted activity but must be reevaluated within 12 to 24 hours to assess for persistent or worsening symptoms or significant rise in muscle enzymes (e.g., CPK rise, >1000 mg/dL; transaminase values greater than thrice normal). Serial evaluations should continue until all parameters return to normal. Any case with severe or inadequately improving symptoms, continually rising muscle enzymes, early metabolic derangement, or persistent myoglobinuria requires more aggressive fluid treatment that is optimally done in the hospital (Level of Evidence C, consensus opinion).[26] Hospitalization is also warranted if the clinical picture is unclear, other features of concern are present, or compliance with rest is suspect.

Occasionally, patients present with fulminate rhabdomyolysis with massive muscle necrosis. These individuals manifest early severe metabolic derangements with acidosis often accompanied by shock. Many cases of noncardiac exertional sudden death are believed to arise from this condition due to electrolyte-induced dysrhythmias.[14,22] These cases require treatment according to advanced life support protocols for the dysrhythmias and transfer to an intensive-care facility for management of the metabolic derangements. Muscle necrosis in these cases is often perpetuated by increased compartment pressures, even with low elevations, and improves with early fasciotomy of involved muscle areas (Level of Evidence C, expert opinion).[27,28]

Experience with Marine recruits demonstrates that healthy individuals with uncomplicated mild to moderate rhabdomyolysis may return to activity immediately after all enzymes have returned to normal. It may be prudent, though, to resume exercise in a graduated manner, particularly if restricted from activity for more than a few days. Recurrent bouts of rhabdomyolysis or any severe episodes warrant investigation for an underlying disease process (Level of Evidence C, consensus opinion).[14]

CONCLUSION

With the exception of athletic pseudoanemia, it is uncommon to encounter significant persistent hematologic alterations from running. While high-intensity and prolonged endurance training may result in alterations of several hematologic parameters and occasionally lysis of RBCs, rarely are these of pathologic significance; however, signs and symptoms of hematologic disease may manifest at an earlier state in athletes due to physiologic demands that require maximal hematologic system performance. The condition of exertional rhabdomyolysis may occasionally manifest in athletes advancing training too rapidly but may also appear in a conditioned athlete in association with underlying disease states or as a consequence of severe over-exertion or exertional heat illness. Identification and early treatment of those with myoglobin release or severe myocyte injury is crucial to preclude serious complications.

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60 ALLERGIC DISEASES IN ATHLETES

David L. Brown and Linda L. Brown

INTRODUCTION

While allergic symptoms may at first appear to be a minor nuisance affecting athletes, physicians should not underestimate their prevalence and impact. Allergic rhinitis alone affects over 50 million Americans.[1] It is the fifth most common chronic disease and the most prevalent in patients under 18 years of age.[2] Each year it is directly related to 3.8 million lost work and school days as well as healthcare costs of approximately $8 billion.[3] Other allergic conditions can beset athletes as well. Urticaria and angioedema are quite common, affecting 20 to 30% of the population sometime during their lifetime.[4] Approximately 20,000 to 50,000 patients with anaphylaxis present for medical care in the United States each year, resulting in 400 to 1000 deaths.[5] More important to the individual athlete is that these conditions can affect not only their physical capacity but also their psychological motivation to train and compete. Allergic rhinitis, in particular, may also be a harbinger of other atopic conditions such as eczema and asthma.

RHINITIS

Pathophysiology

Allergic rhinitis occurs when an individual develops immunoglobulin E (IgE) sensitization to aeroallergens. Subsequent inhalation of these aeroallergens cross-links IgE receptors on mast cells in the respiratory epithelium, leading to the release of histamine and other chemical mediators of inflammation. The etiology of non-allergic rhinitis, which can be difficult to distinguish clinically from allergic rhinitis, is unknown.

Evaluation

A detailed history is of the utmost importance in differentiating allergic and non-allergic rhinitis. Allergic rhinitis is typically associated with rhinorrhea, post-nasal drip, and congestion. Other common symptoms include sneezing and cough as well as nasal and soft palate pruritus. Eye pruritus, injection, irritation, and watery discharge may indicate coexisting allergic conjunctivitis. Patients may also complain of generalized irritability and fatigue. Symptoms will occur upon re-exposure to any aeroallergen to which a patient

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is sensitized; therefore, asking about seasonal exacerbations as well as indoor vs. outdoor predominance of symptoms is crucial. Seasonal symptoms will often be due to pollen exposure. Spring and early summer exacerbations will occur with tree and grass pollination, while late summer and fall symptoms are usually due to weeds and mold. Perennial symptoms may be a sequential combination of these allergens. Alternatively, especially if symptom flares are mainly indoors, the athlete may be sensitive to cockroaches, dust mites, pet dander, or molds.

Allergic and non-allergic rhinitis presentations can be nearly identical, but non-allergic rhinitis patients complain about prominent nasal congestion while nasal, eye, and soft palate pruritus are usually absent. Non-allergic rhinitis symptoms are often perennial and triggered by strong odors or smoke. However, fluctuations in air temperature, humidity, and barometric pressure occurring with the change in seasons may lead to exacerbations, making it difficult to differentiate from allergic rhinitis. After questioning the athlete regarding the symptoms and precipitants listed, the physician should also conduct a thorough review of the athlete's past medical and family history looking for asthma, allergies, and eczema that would lead to a higher index of suspicion for allergic rhinitis.

The utility of physical examination in distinguishing allergic and non-allergic rhinitis is poor; however, some pertinent clues can be helpful. The nasal mucosa in allergic rhinitis is classically described as pale or bluish; however, as in non-allergic rhinitis, the mucosa can be red and edematous or may appear normal. Posterior pharyngeal cobblestoning is associated with post-nasal drip of any etiology. Likewise, so-called "allergic shiners" from infraorbital venous congestion are also nonspecific. Findings that are more suggestive of allergic rhinitis include an accentuated transverse nasal crease seen in children who repeatedly rub their nose due to pruritus, atopic stigmata such as eczema, and wheezing on ausculation.

Management

Treatment for allergic rhinitis is multifaceted but typically starts with avoidance measures. If these are ineffective, medical therapy can be initiated in a stepwise fashion (Figure 60.1). Ultimately, immunotherapy may be necessary if symptoms remain uncontrolled.

Allergen Avoidance

Allergen avoidance is a very important treatment modality in managing allergic rhinitis (Level of Evidence C, consensus opinion).[6] For patients allergic to animal dander, avoidance is always

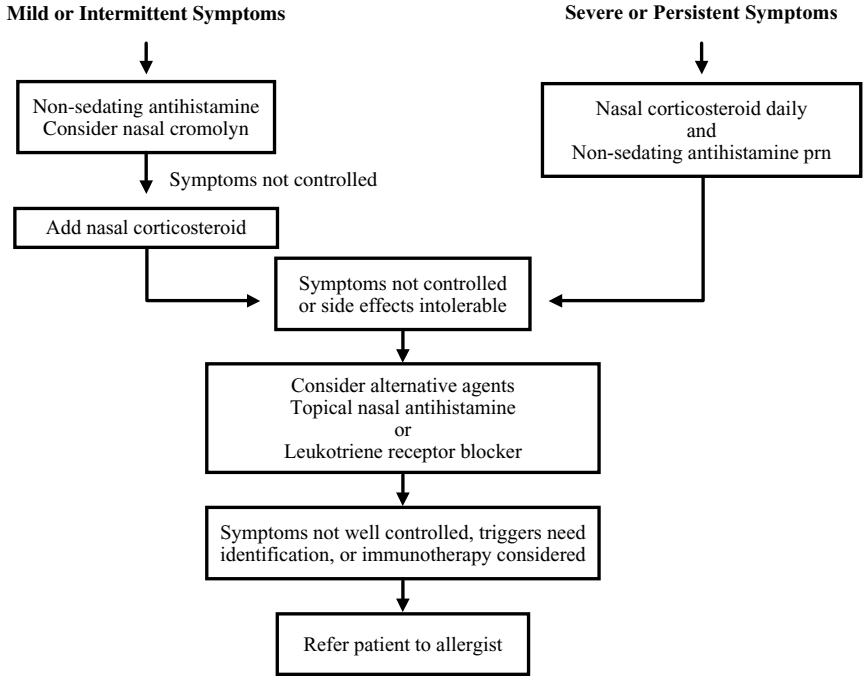

Figure 60.1 Stepwise therapy for allergic rhinitis.

TABLE 60.1
Second-Generation Oral Antihistamines

	Dose	Sedation
Fexofenadine (Allegra®)	Age ≥ 12: 180 mg qd or 60 mg bid Age 6–11: 30 mg bid	No different than placebo
Cetirizine (Zyrtec®)	Age ≥ 6: 5–10 mg qd Age 2–5: 2.5–5 mg qd (syrup)	Slightly higher than placebo but less than first generation
Loratadine (Claritin®)	Age > 6: 10 mg qd	No different than placebo at 10 mg; sedating at higher doses
Desloratadine (Clarinex®)	Age ≥ 12: 5 mg qd	No different than placebo[a]

[a] Note that 7% of population may have sedation due to decreased metabolism of the drug.

best, but consistent exclusion of the pet from the bedroom and use of a HEPA filter may provide some benefit. For dust mite allergy, occlusive covers on the pillows, mattress, and box springs are essential. Frequent washing of bed linens and blankets in hot water is helpful. Dehumidifiers and removing carpet may also be required for highly sensitive patients. HEPA filters are ineffective because dust mite products are not airborne for an extended period of time. Mold allergen can be difficult to control, but dehumidifiers and scrupulous cleaning can be beneficial.

Medical Therapy

Several medication classes are available for the control of rhinitis, including decongestants, oral and topical antihistamines, cromolyn, corticosteroids, and leukotriene receptor blockers. Oral decongestants such as pseudoephedrine can provide effective symptom relief for congestion due to allergic and non-allergic rhinitis. They have been found to be most effective when used in combination with an oral antihistamine for allergic rhinitis (Level of Evidence B, randomized controlled trials).[7] Side effects of oral decongestants include insomnia, irritability, tachycardia, and palpations. Because of peripheral vasoconstriction, they also interfere with heat dissipation and should be avoided during training or competition in the heat. Topical nasal decongestants such as oxymetazoline should be reserved for short-term control of severe congestion. Their use should be limited to no more than 3 days. If used longer than 5 to 7 days, they can cause severe rebound congestion and rhinorrhea known as rhinitis medicamentosa.

Antihistamines act as competitive binders to the H1 receptor and have proven effective in relieving sneezing, itching, and watery rhinorrhea in allergic rhinitis. Their efficacy is roughly equivalent to nasal cromolyn but less than nasal steroids. They provide little relief of nasal obstruction and are generally ineffective in the treatment of non-allergic rhinitis. First-generation antihistamines can cause significant sedation, decreased alertness, and performance impairment, making them undesirable for most competitive athletes. These effects can exist even without an individual being aware of them. Dosing sedating antihistamines only at night may still cause daytime impairment. Second-generation antihistamines are at least as effective as first-generation antihistamines and possess much lower rates of sedation (see Table 60.1). The other caveat to antihistamine use is that they have the potential to decrease heat dissipation by their anticholinergic effects on sweat glands. Thus, as is true for sympathomimetics, athletes should use them with caution during high-intensity training or competition and when ambient temperatures are high.

Nasal corticosteroids are the most effective therapy for persistent or severe symptoms (Level of Evidence Level A, randomized controlled trials).[8] They work by inhibiting gene expression of inflammatory mediators. Several days of treatment are usually necessary for maximal effectiveness. Nasal steroids can be used periodically during the year, depending on the athlete's allergy season. However, when such treatment is initiated, the steroid must be administered regularly for consistent efficacy (see Table 60.2). Topical steroids have a low rate of side effects, which include irritation, burning, sneezing, and bloody nasal discharge.

Some providers may be concerned about starting nasal steroids for fear of side effects from chronic use or growth disturbance in a skeletally immature athlete; however, when taken appropriately, even chronic nasal steroid use is not associated with any significant adrenal suppression, nasal

TABLE 60.2
Topical Nasal Corticosteroids

	Dose (Sprays per Nostril)
Flonase® (fluticasone)	Age ≥ 12: 1 bid or 2 qd; ages 4–11: same, but start at 1 qd
Nasonex® (mometasone furoate)	Age ≥ 12: 2 qd; ages 3–11: 1 qd
Rhinocort Aqua® (budesonide)	Age ≥ 12: 1–4 qd; ages 6–11, 1–2 qd
Nasarel® (flunisolide)	Age > 14: 2 bid to qid; ages 6–11: 1–2 qd
Nasacort AQ® (triamcinolone)	Age ≥ 12: 2 qd; ages 6–11: 1–2 qd
Beconase AQ® (beclomethasone)	Age ≥ 12: 1–2 bid; ages 6–11: 1 bid
Vancenase AQ DS® (beclomethasone)	Age ≥ 6: 1–2 qd

or pharyngeal candidiasis, cataracts, or glaucoma.[9,10] Furthermore, although growth studies on beclomethasone (BDP) dosed at 168 μ g twice daily for 1 year in children ages 6 to 9 years showed a mean decrease in height compared to placebo of 0.9 cm ($p < 0.01$), growth studies for mometasone furoate (Nasonex®) dosed at 100 μ g daily for 1 year in children ages 3 to 9 years and for fluticasone (Flonase®) dosed at 2 sprays daily in ages 4 to 11 years showed no difference in growth compared to placebo.[11–13]

Cromolyn, a topical mast-cell stabilizer, has a low potential for toxicity and provides modest improvement in the sneezing, itching, and rhinorrhea associated with allergic rhinitis. It is useful when administered prophylactically prior to allergen exposure; however, it often requires dosing up to 4 to 6 times daily to be effective, which may affect compliance and therefore symptom control.

Leukotriene receptor antagonists such as montelukast (Singulair®) provide mild improvement in symptoms of seasonal allergic rhinitis with efficacy similar to second-generation antihistamines (Level of Evidence B, systematic review).[14] These agents are well tolerated with a side-effect profile no different than placebo. Leukotriene receptor antagonist therapy should be considered when nasal steroids and/or antihistamines fail or side effects are intolerable. They can also be considered for use in an athlete who may benefit from this therapy for treatment of concomitant asthma. Dosing is 4 mg for ages 2 to 5, 5 mg for ages 6 to 11, and 10 mg for ages 12 and above.

Topical nasal antihistamines such as azelastine (Astelin®) are effective in providing short-term relief of symptoms due to both allergic and nonallergic rhinitis. Side effects include drowsiness and an unpleasant aftertaste. While intranasal steroids have greater relief of nasal symptoms, azelastine can be considered as an alternative when the response to an oral antihistamine and a nasal corticosteroid is inadequate (Level of Evidence A, meta-analysis).[15] Dosing for patients ages 5 to 11 years is 1 spray twice daily; for those 12 years or older, the dose is 2 sprays twice daily.

Ipratropium bromide (Atrovent®) 0.03% nasal spray is effective for treating rhinorrhea, particularly vasomotor-induced rhinorrhea triggered by cold air or exercise. It has no effect on pruritus or congestion. Side effects include occasional epistaxis and nasal dryness but no systemic anticholinergic or rebound effects. The dose is 2 sprays 30 minutes prior to exercise or exposure, and it may be used two or three times daily for patients over 6 years of age.

When these treatments fail, it is important to consider not only medication inadequacy or noncompliance, but also the possibility of other diagnosis such as anatomical or physical obstruction and chronic sinusitis.

Athlete-Specific Medication Issues

Athletes, especially at the collegiate or elite levels, have to be especially careful as medication use may affect their eligibility for competition. Because restrictions on over-the-counter and prescription medications can change, athletes should discuss the status of medications with the governing body for their particular sport or level of competition prior to its use. This would include the National Collegiate Athletic Association (NCAA) and U.S. Olympic Committee (USOC). In general, the NCAA has no restrictions on any allergy-related products with the exception that any products containing ephedrine are banned. The USOC, however, is much more stringent. All sympathomimetic-containing medications are banned. Antihistamines are allowed in all but the shooting sports, in which they are completely banned. While the USOC does not restrict cromolyn or leukotriene receptor blockers, written

TABLE 60.3
Allergic Conjunctivitis Topical Medications

Topical Agent	Mechanism of Action	Dose
Patanol® (olopatadine)	Mast cell blocker/antihistamine	1 drop bid; age >3
Zaditor® (ketotifen)	Mast cell blocker/antihistamine	1 drop bid to tid
Alomide® (lodoxamide)	Mast cell blocker	1–2 drops qid
Alamast® (pemirolast)	Mast cell blocker	2 drops qid
Alocril® (nedocromil)	Inhibits activation and mediator release from inflammatory cells	1–2 drops bid
Livostin® (levocabastine)	Antihistamine	1–2 drops qid
Emadine® (emadastine)	Antihistamine	1 drop qid
Optivar® (azelastine)	Antihistamine	1 drop bid
Crolom® (cromolyn)	Mast cell blocker	1–2 drops, 4–6 times/day
Naphcon A®, Opcon A®, Vasocon A®, Visine A®	Antihistamine/decongestants	Up to qid

permission should be obtained for nasal or inhaled steroid use.[16]

Allergy Testing

Typically, referral to an allergy specialist for skin testing should be made when a patient's history is suggestive of allergic rhinitis and institution of immunotherapy is being considered for poor response to medical therapy. Allergy consultation is also recommended when drastic environmental interventions are being considered such as pet elimination, taking up carpets, or purchasing new mattresses, bedding, dust mite covers, etc. It is important to stop antihistamines for one week prior to testing so as not to blunt the cutaneous response to skin testing. Allergy testing is contraindicated in the setting of severe lung disease or poorly controlled asthma with a forced expiratory volume in 1 second (FEV1) of less than 70%. Skin testing is preferred over *in vitro* radioallergosorbent testing (RAST) because it is more sensitive. *In vitro* RAST is helpful for validating the diagnosis and supporting environmental controls. It is less sensitive than skin testing, but is a reasonable alternative when skin testing cannot be performed.

Allergen Immunotherapy

Allergen immunotherapy (AIT) is effective treatment for allergic rhinitis and allergic asthma and has the advantages of providing long-lasting symptom remission as well as reducing the risk of developing new allergies and asthma in children (Level of Evidence A, meta-analyses).[17] Patients should be advised that notable symptom relief usually takes several months of treatment. Athletes also need to commit to 3 to 5 years of treatment in order to sustain remission of their symptoms. Any individual who is unable to fully commit to treatment, has poorly controlled asthma, or is on a beta-blocker should not receive AIT. It is important that AIT be administered by a board-certified allergist who can engage in a thorough discussion with the athlete of the benefits and potential risks of therapy. Ongoing follow-up with the allergist is warranted to ensure appropriate dosing to achieve optimal benefit while avoiding systemic reactions.

ALLERGIC CONJUNCTIVITIS

The etiology for acute allergic conjunctivitis is the same as for allergic rhinitis. In this particular case, symptoms occur upon inoculation of the allergen onto the mucosa of the eyes. Individuals with animal dander sensitivity are especially prone to ocular symptoms. Seasonal allergic conjunctivitis is most likely to be severe in pollen-sensitive patients during tree pollen season. The treatment of allergic conjunctivitis can be achieved with the same measures as discussed with allergic rhinitis; however, if ocular symptoms persist despite other therapies or occur in isolation, targeted use of medications specifically for the eye is warranted (see Table 60.3). Combination mast cell blocker and antihistamine topical therapy is very effective. Other options include topical mast cell blockers or antihistamine alone, topical decongestants, and topical mast cell stabilizers. Topical corticosteroids are associated with significant complications and should only be used after consultation with an ophthalmologist.

URTICARIA AND ANGIOEDEMA

Pathophysiology

Urticaria is a condition occurring secondary to mast cell degranulation in the superficial dermis. It is characterized by pruritic, erythematous, cutaneous elevations that blanch with pressure. These evanescent hives may occur anywhere on the body but more often occur on the trunk and extremities. Immunologically, a variety of potential mediators are involved in urticaria, all leading to blood vessel dilation and edema. These mediators include histamine, prostaglandins, leukotrienes, platelet-activating factor, anaphylatoxins, bradykinin, and Hageman factor. Acute urticaria is defined as new-onset symptoms of less than 6 weeks in duration. If symptoms persist longer than 6 weeks, it is considered chronic urticaria. In patients who go on to have chronic urticaria, 75% have symptoms over 1 year, 50% have symptoms over 5 years, and 20% have symptoms for decades. Angioedema is similar pathologically to urticaria but occurs in the deeper dermis and subcutaneous tissues. Unlike urticaria, angioedema is more painful and burning than it is pruritic and often involves the face. Urticaria can occur at any age but is most common in children and young adults. Approximately 50% of patients at presentation have both urticaria and angioedema, 40% have urticaria only, and 10% have angioedema only.[18]

In most cases, the cause of urticaria is idiopathic; however, the many potential triggers for urticaria include medications, insect stings, foods and food additives, and infections. It is beyond the scope of this text to review all of these, but Table 60.4 lists the most common known triggers. In addition to these triggers, various physical exposures can lead to urticaria as well (see Table 60.5). These physical urticarias are especially important to consider in athletes because they can be triggered by conditions that occur during practice and competition.[19]

Cholinergic urticaria is caused by an elevation in core body temperature and is classically precipitated by exercise or use of hot tubs. Initially small, papular, pruritic papules will occur followed by wheals with flare. Symptom onset is usually within 2 to 30 minutes and lasts up to 90 minutes.

Cold urticaria is precipitated by rewarming following contact with a cold object. Within 2 to 5 minutes, the area exposed to the cold develops swelling and pruritus. Symptoms generally worsen after the area is warmed and can last up to 2 hours. Placing an ice cube on the skin for 15 minutes and observing for presence of hive formation can confirm the diagnosis. A risk of anaphylaxis exists with this condition due to massive histamine release if patients have a significant drop in core body temperature, such as with swimming or diving; therefore, patients with cold urticaria should avoid these activities.

Aquagenic urticaria is extremely rare and is caused by contact with water. In athletes who participate in water sports, this condition could

TABLE 60.4
Common Triggers for Urticaria

Medications
Antibiotics (beta-lactams, sulfa compounds)
Nonsteroidal antiinflammatories (NSAIDs)
Progesterone
Local anesthetics
Opioid analgesics
Physical contacts
Latex
Nickel
Plants and plant resins
Fruits/vegetables
Raw fish
Animal saliva
Insect stings
Foods and food additives
Milk
Egg
Peanut
Nuts
Soy
Wheat
Fish/shellfish
Sulfites
Infections
Coxsackie A and B
Hepatitis A, B, C
Human immunodeficiency virus (HIV)
Epstein–Barr virus
Herpes simplex
Intestinal parasites
Dermatophyte infections

be confused with both cholinergic and cold urticaria. Unlike cholinergic urticaria, with aquagenic urticaria symptoms still occur even when the water temperature is cool and even if the patient is not exercising in the water. It can be distinguished from cold urticaria because it will not be precipitated by application of a cold object that is not water based.

Solar urticaria is rare and is precipitated by exposure to ultraviolet light. Anaphylaxis could occur if large body areas are exposed. Avoidance of ultraviolet light exposure is the best therapy in addition to liberal sunscreen use.

Pressure urticaria (angioedema) accounts for less than 1% of all urticarias. It is precipitated by direct pressure on the skin. In this type of urticaria, skin pressure is followed 3 to 12 hours later by localized hives as well as fever, malaise, and leukocytosis. Symptoms can last up to 24 hours. It can be precipitated by running, clapping, sitting, or using hand equipment. The diagnosis can be confirmed by applying a 15-pound weight to an individual's skin for 20 minutes and then examining the patient 4 to 8 hours later for characteristic signs and symptoms.

Symptomatic dermatographism is another type of physical urticaria. It occurs in 2 to 5% of the general population and is characterized by the development of linear, pruritic wheals 2 to 5 minutes after stroking or rubbing the skin.

Evaluation

The history is most important in helping to define the onset and time course of these conditions. In the acute setting, providers should also assess the existence of coexisting symptoms that could indicate anaphylaxis rather than isolated urticaria or angioedema (see Anaphylaxis and Anaphylactoid Reactions section, below). While in most cases the precipitant remains unknown, a detailed history may isolate a trigger. Searching for a trigger is more beneficial in acute urticaria as compared to chronic urticaria, where an etiology is found in less than 10% of cases.

When the cause is found, drug hypersensitivity is most common; thus, individuals should be asked about any recent prescription or over-the-counter medications. For athletes, in particular, it is important to inquire about supplement use. Food and food additives rarely cause isolated urticaria, but the relationship to food inhalation, contact, and consumption should be documented. It is also important to document the relationship to physical triggers (e.g., heat, cold, sunlight, water, and skin pressure), occupational exposures, recent insect envenomations, and any recent illnesses. A thorough review of systems is important to help rule out any disease associations such as an acute bacterial or viral illness, parasitic infection, autoimmune/collagen vascular disease, serum sickness, endocrine disease, and malignancy.[20]

The physical examination is especially helpful in the acute setting when skin manifestations are present. This can help document whether urticaria and angioedema are occurring in isolation or together as well as any signs of an anaphylactic reaction. A thorough exam should also look for evidence of the other diseases that are more rarely associated with urticaria and angioedema.

The use of laboratory and imaging studies should be targeted by the history and physical. A monospot or Epstein–Barr virus antibody titers can be ordered if acute mononucleosis is suspected. Testing for hepatitis A, B, and C as well as acute human immunodeficiency virus (HIV) infection may be warranted. The association of urticaria with other viral infections remains unclear and routine testing for other viral pathogens is not recommended. If a significant travel history is discovered and the complete blood count shows eosinophilia, stool studies should be obtained looking for intestinal parasites. Progressive weight loss and/or the presence of lympadenopathy or hepatosplenomegaly on exam would warrant an evaluation for an underlying lymphoreticular malignancy. If enlargement or nodularity of the thyroid is present, a thyroid function panel, thyroid autoantibodies, thyroid ultrasound, and nuclear medicine thyroid studies should be considered. Testing for hereditary or acquired C1 esterase inhibitor deficiency should be considered in any athlete presenting with recurrent isolated angioedema. A skin biopsy looking for vasculitis is indicated when individual urticarial lesions last longer than 24 hours or are associated with purpura, pain, hyperpigmentation, or systemic symptoms (Level of Evidence C, consensus opinion).[21]

If the history and physical exam are unrevealing, conducting a limited laboratory evaluation consisting of a complete blood count with differential, urinalysis, erythrocyte sedimentation rate, and liver panel is reasonable to screen for occult conditions. See Table 60.5 for evaluation of the physical urticarias.

Management

After the initial evaluation, the management of urticaria and angioedema becomes primarily symptomatic. Any known trigger, of course, should be avoided. Mild symptoms can be controlled with

TABLE 60.5
Physical Urticarias

Type	Precipitant	Evaluation	Treatment
Cholinergic urticaria	Elevation in core temperature; exercise	History	Premedicate with non-sedating antihistamine.
Cold urticaria	Rewarming after contact with cold object	Place cold object on skin for 15 minutes and look for urticaria upon rewarming.	Use non-sedating antihistamines as needed; avoid swimming and diving sports due to risk of anaphylaxis.
Aquagenic urticaria	Water contact	History; expose skin to water and look for changes.	Use non-sedating antihistamines.
Solar urticaria	Ultraviolet light exposure	Expose small, unprotected patch of skin to sunlight.	Limit sun exposure; wear protective clothing; use sunscreen.
Pressure urticaria/ angioedema	Direct pressure on skin; running, prolonged sitting, clapping, etc.	Place 15-lb weight on patient for 20 minutes and look for skin changes; test for fever and leukocytosis 3 to 12 hours later.	Avoid precipitants; use non-sedating antihistamines and NSAIDs; consider steroid burst/taper if symptoms are severe.
Symptomatic dermatographism	Stroking or rubbing skin; areas where clothing or equipment abrades skin	Look for linear, pruritic wheal 2 to 5 minutes after rubbing the skin.	Wear loose-fitting clothing; treatment usually not necessary; non-sedating antihistamines can be used but only for severe symptoms.

a low sedating antihistamine (see Table 60.1). If symptoms are more moderate or poorly controlled, the antihistamine dose should be maximized prior to considering add-on therapy. Additive therapies include leukotriene antagonists, H-2 blockers, and night-time doxepin. For periods of moderate to severe symptoms, prednisone therapy tapered over 6 to 12 days can be helpful. Because food and food additives are a rare cause of chronic urticaria and angioedema, elimination diets are usually unnecessary unless the patient gives a history pinpointing a specific food. The management of the physical urticarias is detailed in Table 60.5. Referral to an allergist is recommended for suspected allergic component precipitating symptoms, when symptoms are not well controlled with the therapies listed above, when the patient has a history of respiratory distress or hypotension suggesting anaphylaxis, or in cases of severe angioedema. The athlete should be referred to dermatology for skin biopsy if urticarial vasculitis is suspected.

ANAPHYLACTIC AND ANAPHYLACTOID REACTIONS

Pathophysiology

Anaphylaxis is an acute, life-threatening, systemic reaction mediated through IgE antibodies and their receptors. It requires previous sensitization and subsequent re-exposure to an allergen. Anaphylactoid reactions are clinically indistinguishable from true anaphylaxis because both are caused by massive release of potent chemical mediators from mast cells and basophils. As such, both are managed with the same treatment measures discussed here. The difference is that anaphylactoid reactions are not mediated by IgE antibodies, they do not require prior sensitization, and they are less commonly associated with severe hypotension and cardiovascular collapse.

A typical case of anaphylaxis includes cutaneous signs or symptoms accompanied by obstructive respiratory symptoms and/or hemodynamic changes. Additional features include gastrointestinal complaints and experiencing a sense of impending doom. The onset of symptoms typically begins seconds to minutes after the inciting cause. More rarely, symptoms may be delayed for up to 2 hours. Approximately half of cases have a uniphasic course with abrupt, severe onset and death within minutes despite treatment. Up to 20% of cases have a biphasic presentation with immediate symptoms followed by an asymptomatic period for 1 to 8 hours. A late-phase reaction subsequently ensues with recurrence of severe symptoms. These symptoms are protracted, persisting for several hours in 28% of individuals.[22]

Evaluation

Making the diagnosis of anaphylaxis can be affected by variability in the standard definition of a case. Obtaining as much information from the affected athlete and any witnesses is important to define the time course and severity of the reaction as well as the potential cause. Anaphylaxis has many triggers including food, medications, and insect stings (see Table 60.6). Thus, exposure to any of these causes needs to be documented in the history. Hymenoptera sensitivity should be suspected as a cause of anaphylaxis in any athlete with a reaction that occurs outdoors, even if the patient does not recall being stung. Any food exposure prior to the onset of symptoms should be documented. Of special concern would be exposure to the most common food allergens, which include eggs, peanut, cow's milk, nuts, fish, soy, shellfish, and wheat. Several medications have been known to cause anaphylaxis, with the most common being beta-lactam antibiotics. Documenting exposure to prescription medications as well as over-the-counter medications and supplements is also important.

Because this is a sports medicine text, the discussion would be incomplete without mentioning exercise-induced anaphylaxis (EIA). This rare condition is associated with exercising within 2 to 4 hours after food ingestion. It is characterized by the usual manifestations of anaphylaxis beginning within 5 to 30 minutes of exercise and lasting up to 3 hours. The medical history should explore the relationship of symptom onset to physical exercise to assess for this rare trigger.

The physical manifestations of anaphylaxis involve multiple sites including the skin, upper airway, lower airway, and cardiovascular system (see Table 60.7). The physical examination should start by evaluating upper-airway patency by listening for inspiratory stridor and looking for oral or pharyngeal edema. The athlete's respiratory status should then be assessed by observing their work of breathing and accessory muscle use. Auscultation may reveal wheezing, indicating acute bronchospasm. A set of vital signs is critical to patient management, looking for any evidence of cardiovascular or respiratory compromise. Once the ABCs are assessed and secured, the skin can be examined for the presence of generalized erythema, urticaria, and angioedema.

TABLE 60.6
Causes of Anaphylaxis

Idiopathic
Medications
Antibiotics
Intravenous and local anesthetics
Aspirin/NSAIDs
Chemotherapeutic agents
Opiates
Vaccines
Allergy immunotherapy sera
Radiographic contrast media
Blood products
Latex
Hymenoptera envenomation
Foods
Eggs
Peanut
Cow's milk
Nuts
Seafood
Soy
Wheat
Exercise (EIA)

Acute Management

Initial management of anaphylaxis should always include administration of epinephrine (0.2 to 0.5 cc IM) or SQ of 1:1000), even if symptoms are mild. The IM route is preferred, especially in children, as SQ injection may delay absorption. The dose may be repeated every 10 to 15 minutes if symptoms are not resolving. Using intravenous epinephrine at 1 μg/min of 1:10,000 (10 μg/mL) can be considered for ongoing symptoms resistant to repeated SQ or IM administration. This dosage can be increased to 2 to 10 μg/min for severe reactions. Patients on beta-blockers may not respond to epinephrine. In these cases, glucagon (2 to 5 mg IM/SQ) is beneficial. Supportive therapy with oxygen for hypoxemia, recumbent positioning, and intravenous fluids for hypotension, as well as inhaled beta-agonists or racemic epinephrine for bronchospasm, are also important tools. Antihistamines such as diphenhydramine (1 to 2 mg/kg or 25 to 50 mg IV/PO) may provide additional benefit. Corticosteroids, such as prednisone (0.5 to 2.0 mg/kg up 125 mg) should also be considered to prevent late-phase reactions. However, it cannot be over-emphasized that neither antihistamines nor steroids should be used as substitutes for epinephrine. Their onset of

TABLE 60.7
Symptoms and Signs of Anaphylaxis

Psychological
Sense of impending doom
Cutaneous
Tingling/pruritus
Generalized erythema
Urticaria
Angioedema
Upper airway
Nasal congestion
Rhinorrhea
Sneezing
Globus sensation
Throat tightness
Dysphonia
Dysphagia
Lower airway
Dyspnea
Wheezing
Cough
Cardiovascular
Lightheadedness
Syncope
Palpitations
Shock
Gastrointestinal
Abdominal cramps
Bloating
Nausea/vomiting

action is much slower and they are insufficient to prevent or treat more severe anaphylaxis with respiratory or cardiovascular involvement. Athletes presenting with anaphylaxis should be observed a minimum of 3 hours after symptoms have resolved following a mild reaction. An individual should be observed at least 6 hours after a more severe reaction and hospitalization should be strongly considered to monitor for late-phase reactions.

Long-Term Management

It is critical that all patients with anaphylaxis have an action plan. This should include not only identification of their particular allergens but also symptom recognition and appropriate treatment. All of these individuals should carry an epinephrine autoinjector on their person at all times. While remembering to carry the injector is important, it is equally crucial that they have good education on indications and proper technique for its use. A provider knowledgeable in allergic disease should offer education on allergen avoidance, hidden allergens, and cross-reacting substances. These athletes should wear medical alert bracelets at all times, indicating their condition and allergy if known.

Unfortunately, for the athlete with exercise-induced anaphylaxis, no proven preventive therapy exists for this condition. Avoiding eating food or ingesting nonsteroidal anti-inflammatory drugs (NSAIDs) or aspirin 2 to 4 hours prior to exercise may help. Additionally, avoiding outdoor exercise during periods of high humidity and extremes of temperature or when it is the individual's allergy season may limit attacks (Level of Evidence B, epidemiologic studies).[23] Pretreatment with antihistamines may be helpful.[24] Occasionally skin testing can identify a specific food that the patient can avoid, but often the results are inconclusive. The individual must always have access to an epinephrine autoinjector during practice and competition. Athletes such as cross-country runners, cyclists, or skiers should carry the autoinjector on their person when they do not have immediate access to their gear bags. They should discontinue exercising at the first sign of symptoms and self-administer their epinephrine. Their trainers and coaches should also be familiar with the recognition of anaphylaxis and use of epinephrine. Affected athletes should be advised to wear a medical alert bracelet and never to exercise alone.

Indications for allergy specialist referral include when further testing is necessary for an unclear diagnosis or when the inciting agent is unknown, when reactions are recurrent and difficult to control, or when desensitization is required such as for stinging insects or antibiotic administration. Allergists also serve as an important resource for athletes, parents, and coaches who need additional education on allergen avoidance as well as institution or reinforcement of an individual's action plan.

SUMMARY

Allergic diseases can be important causes of morbidity and potentially mortality in athletes. Management begins with an understanding of the pathophysiologic mechanisms involved and hinges on an appropriate history and physical. In the majority of athletes, rhinitis and urticaria can be managed at the primary care level with allergen avoidance and medications. Special attention should be paid to the use of any restricted or banned medication as dictated by the athlete's level of competition. When symptoms are more significant or difficult to control and when advanced testing is required, appropriate subspecialist referral is necessary.

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61 NEUROLOGIC DISORDERS

Jay Erickson and Seth Stankus

INTRODUCTION

Neurological problems in sports medicine can be divided into three main categories: (1) headaches, (2) concussions and post-concussive syndrome, and (3) epileptic type seizure syndromes. This chapter focuses on the first two categories, and seizure-related syndromes are discussed elsewhere. Within each category, the priority for the on-site physician or trainer is to determine the seriousness of the condition and how best to evaluate and provide treatment recommendations for each athlete upon presentation.

HEADACHE

According to Mauskop and Leybel,[1] chronic headaches afflict over 40 million Americans. With the additional head trauma suffered by many athletes in a variety of sports, subsequent acute headaches would only add to this total. But, whether the headache symptoms are acute or chronic, the key to distinguishing significant pathology from a benign headache lies in the history. Combined with a thorough neurological exam, an accurate history will offer the best chance to obtain the most appropriate level of care at the earliest opportunity.

Assessment

Clues to the correct diagnosis and effective treatment of an athlete with headache can be found in the detailed history. Four key aspects of any headache include:

- Precipitating factors
- Character of headache pain
- Location of headache
- Preceding and accompanying symptoms

Additionally, personal and family medical history, current or recent illnesses, and substance intake (e.g., caffeine, medications, and drugs of abuse) can be confounding factors. All aspects of the neurological examination are critical, and a thorough fundoscopic examination to evaluate for possible papilloedema is especially important in the child athlete with headache. It is also vital to remember that even headaches classified as benign based upon all signs, symptoms, and evaluation may still be the initial presentation of patients with serious underlying abnormalities. One small study of benign exertional headache found organic brain lesions as the headache source in nearly 10% of its patients.[2] It is because of these infrequent but possible missed lesions that many neurology experts will recommend a screening head and neck radiographic study of all first-time headache patients, regardless of their severity categorization.

Classifi cation

The International Headache Society (IHS) provides an extensive classification system for headaches (Table 61.1). While this system is academically accurate and specific, the provider on site is best served by a decision analysis method that distinguishes the athlete's headache among one of three categories based on treatment needs:

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TABLE 61.1
International Headache Society Classification of Headache

1. Migraine
2. Tension-type headache
3. Cluster headache and chronic paroxysmal hemicrania
4. Miscellaneous headaches unassociated with structural lesion
5. Headache associated with head trauma
6. Headache associated with vascular disorders
7. Headache associated with nonvascular intracranial disorders
8. Headache associated with substances or their withdrawal
9. Headache associated with noncephalic infection
10. Headache associated with metabolic disorder
11. Headache or facial pain associated with disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures
12. Cranial neuralgias, nerve trunk pain, and deafferentation pain
13. Headache not classifiable

- *Serious* — Requiring immediate evaluation and treatment
- *Concerning* — Requiring evaluation and treatment but not immediate
- *Benign* — No further evaluation or treatment required

Applying this algorithm to the multitude of headache classes allows the on-site provider to quickly classify a patient's headache without the need for advanced technology or testing methods in all headache presentations.

Serious Headache

Headache occurs either with or without trauma and is associated with other neurological symptoms, such as mental status changes, nausea, vomiting, increased neck stiffness, or focal neurological findings on examination. Any of these signs or symptoms may be indicative of underlying hemorrhage, both within and outside of the brain parenchyma. Not every patient with a subarachnoid hemorrhage will declare it as being "the worst headache of my life." Testing must include a thorough neurological examination including a mental status exam to be followed by a magnetic resonance imaging (MRI) study of the head and neck region. The use of computed tomography (CT) scans in the immediate evaluation of post-traumatic headache is limited and should not be used to rule out hemorrhage. A negative CT in the patient with significant signs or symptoms should undergo an MRI whenever possible.

Concerning Headache

The patient meets the criteria for concussion (confusion, amnesia, incoordination, slurred speech, emotional lability, or delayed motor or verbal response) but has none of the serious headache signs or symptoms. While it could be argued that confusion and amnesia indicate mental status changes, the additional factor of symptom duration may be used to distinguish between serious and concerning headaches. Although it would appear that concerning headaches are only post-traumatic in origin, additional causes of both concerning and serious headaches include environmental factors such as altitude- and diving-related barotrauma. The athlete with high-altitude sickness and headache must receive prompt treatment to prevent permanent neurological damage secondary to cerebral edema. Descent to altitudes below 2500 meters is the quickest and most obvious form of treatment.

Benign Headache

While best categorized using a diagnosis of exclusion, the benign headache in athletes can be defined as a tension-type headache that occurs as the result of exertion or effort. Frequently labeled as Weightlifter's headache or acute effort migraine, depending on initiating factors, these benign headaches can be either tension type or vascular type in origin. The general lack of additional signs and symptoms seen in either a concerning or serious headache is critical in making any diagnosis of a benign headache. While immediate treatment is not required, the impact of a benign headache on an athlete's performance can be significant. Of note, many athletes who develop benign exertional headaches have associated nausea and vomiting. This combination of symptoms can lead to the use of neuroimaging studies to rule out more serious pathology, but once completed such tests can be used to reassure the athlete in the event of future recurrent episodes of similar symptoms. These advanced studies can be beneficial in that subarachnoid hemorrhage, cerebral aneurysm, Arnold–Chiari malformation, and intracranial neoplasm are all associated with exertional headache. Despite the infrequent occurrence of these syndromes, the

use of CT or MRI to evaluate an athlete's first sudden exertional headache is strongly recommended.

Specifi c Headaches in Athletes

Benign Exertional Headache

Typically precipitated by even minimal physical activity, this headache is best described as dull and throbbing in nature; can be located unilaterally, bilaterally, or occipitally; and may last for several hours. Treatment includes the use of nonsteroidal anti-inflammatory drugs (NSAIDs), either acutely or prophylactically (Table 61.2). In athletes competing at higher altitudes or for more poorly conditioned athletes, a vascular type headache can occur after prolonged exercise. Similar in presentation to the benign exertional headache, additional precipitating factors include poor physical conditioning, exercising unacclimatized at high altitudes, or exercising soon after alcohol consumption. Treatment includes NSAIDs in addition to increased conditioning and improved hydration efforts prior to commencing exercise or athletic participation.

Effort Migraine

Usually brought on by extremely intense exercise, it is more commonly unilateral and throbbing in nature and is generally preceded by scotomata or visual aura as seen in other forms of migraine headaches. Additional symptoms may include nausea, vomiting, and photophobia, and the symptoms may last anywhere from minutes to hours. In those athletes with abrupt resolution of symptoms, treatment is not indicated. For those athletes with prolonged debilitating symptoms, the use of classic migraine medications may allow for a faster return to activities (Table 61.2). Although the use of prophylactic migraine medications is not indicated for patients with effort migraine, the symptoms can be prevented by the use of longer warm-up periods and a gradual increase in workout intensity.

Weightlifter's Headache

This self-explanatory headache occurs in athletes straining to lift heavy weights. Usually a posterior headache in either the occipital or the upper cervical region, the symptoms may last for days to weeks and resolves relatively slowly when compared to other benign headaches. Of note, several forms of underlying pathology can present as a strain-type headache such as weightlifter's headache including Arnold Chiari malformations. While treatment includes rest, ice, and NSAIDs, recurrent strain-type headaches warrant further neurological evaluation to include CT and/or MRI to rule out pathology.

Trauma-Induced Migraine

Seen in athletes competing in contact sports, this headache is typified by preceding minor head trauma without loss of consciousness followed within minutes by visual, motor, and sensory aura, including scotomata, paresthesias, and even hemiplegic symptoms. The headache is usually unilateral, throbbing, and retro-orbital. It may be accompanied by photophobia, nausea, and vomiting and may last for hours to days in duration. Because of the confounding presentation, the first episode should be treated as a concerning headache with complete neurological evaluation, including neuroimaging studies. Once a pathological source is ruled out, recurrent attacks may be treated as classic migraines by using acute migraine medications (Table 61.2). Prophylactic migraine medications are generally not recommended because this syndrome is too unpredictable in its timing.

CONCUSSION

The increasing frequency of concussion in athletes in all age groups has made concussion an important public health issue. Additionally, research into the long-term effects of repetitive minor head trauma has led to even greater restrictions on returning these athletes to competition. In many ways, the athlete who sustains a severe head injury evidenced by skull fracture or severe post-traumatic symptoms is more likely to receive appropriate and immediate treatment. Attempting to diagnose the athlete at greatest risk for second-impact syndrome and potentially fatal progressive cerebral edema has become more difficult and medically critical to the provider on site. With an estimated 20% of high school football players sustaining brain injuries annually[3] and with additional pressure from non-medical personnel to return star athletes to competition as soon as possible, the athletic trainer or team physician faces a critical decision when returning the post-concussive athlete to the field of play.

Mild traumatic brain injury (MTBI), or concussion, is a common consequence of collisions, falls, and other forms of contact in sports. The rapid yet accurate determination of which athletes require immediate emergency room evaluation may be critical to an athlete's survival. Similar to the serious headache, the serious concussion must be diagnosed as early as possible to ensure

TABLE 61.2
Headache Medications for Athletes

Category/Agent	Trade Name	Route	Dosing
Analgesics			
Acetaminophen	Tylenol®	Oral	325–1000 mg every 4 to 6 hours as needed
Nonsteroidal Anti-inflammatories			
Aspirin	Ecotrin®	Oral	325–650 mg every 4 to 6 hours as needed
Ibuprofen	Motrin®, Advil®	Oral	400–800 mg every 8 hours as needed
Naproxen	Naprosyn®, Anaprox®, Alleve®	Oral	220–500 mg every 12 hours as needed
Muscle Relaxants (Potential Sedating Effect)			
Cyclobenzaprine	Flexeril®	Oral	10 mg every 12 hours as needed
Methocarbamol®	Robaxin®	Oral	500–1500 mg every 6 hours as needed
Migraine Sedatives (Potential Sedating Effect)			
Butalbital, acetaminophen, caffeine	Fioricet®	Oral	One every 6 hours as needed
Butalbital, aspirin, caffeine	Fiorinal®	Oral	One every 6 hours as needed
Isometheptine, dichloralphenazone, acetaminophen	Midrin®	Oral	Two at onset, then one per hour until headache is relieved
Ergotamines			
Ergotamine tartrate, caffeine	Cafergot®	Oral	Two at onset, then one per hour until headache is relieved
Dihydroergotamine mesylate, caffeine	Migranol® Nasal	Nasal	One spray in each nostril, repeat in 15 minutes if needed
Triptans			
Sumatriptan	Imitrex®	Oral	One at onset, repeat every 2 hours as needed to maximum of 200 mg
	Imitrex®	Nasal	One spray in each nostril; may repeat once in 2 hours
	Imitrex®	Subcutaneous	One injection at onset, may repeat once after 1 hour

that necessary intervention is commenced early enough to preclude permanent neurological sequelae when possible. Multiple concussion grading scales have been developed and are readily available for use by coaches and athletic trainers (Tables 61.3 through 61.5).

Whether a concussion is classified as mild, moderate, or severe, the key factors in any evaluation are loss of consciousness and its duration along with the presence and duration of post-traumatic amnesia. Once classified by significance, the athlete with a concussion must be closely monitored and evaluated prior to being considered for return to competition. Post-event monitoring is important because many symptoms not initially present may develop over minutes or even hours. Severe delayed symptoms occurring despite no loss of consciousness may be indicative of severe traumatic brain injury and may require immediate treatment to prevent prolonged neurological deficits. This is even more critical in the athlete suffering a second or subsequent post-trauma head injury. In fact,

TABLE 61.3
Guidelines for Return to Sports after Cerebral Concussion

Grade	Features	Management	Return to Play
1 (Mild)	No loss of consciousness Posttraumatic amnesia less than 30 minutes	Remove from contest. Observe on the sidelines.	May return if asymptomatic at rest and with exertion (no headache, dizziness, or impaired concentration plus full recall of the events occurring before injury) Second grade 1 concussion: May return in 2 weeks if asymptomatic at that time for 1 week Third grade 1 concussion: Terminate season; may return next year if asymptomatic
2 (Moderate)	Loss of consciousness less than 5 minutes or posttraumatic amnesia longer than 30 minutes	Remove from contest and disallow return that day. Athlete should be evaluated by a neurologist at a medical facility. Take cervical spine precautions as indicated.	Return after being asymptomatic for 1 week Second grade 2 concussion: Wait at least 1 month; may return then if asymptomatic for 1 week; consider terminating season Third grade 2 concussion: Terminate season; may return next year if asymptomatic
3 (Severe)	Loss of consciousness longer than 5 minutes or posttraumatic amnesia longer than 24 hours	Transport athlete to nearest hospital with neurosurgical facilities with head and neck immobilization. Admit to hospital to check for intracranial bleeding.	Wait at least 1 month; may return then if asymptomatic for 1 week Second grade 3 concussion: Terminate season; may return next season if asymptomatic

Source: Adapted from Cantu, R., *Phys. Sportsmed.*, 14, 1986. With permission.

several authors have commented that loss of consciousness does not necessarily correlate with the severity of the head injury (Level of Evidence B, systematic review).[4]

Although multiple evaluation criteria have been developed over the years, the Standardized Assessment of Concussion (SAC) was developed as a simple, reproducible, and fast method of providing a gross measurement of mental status. The SAC evaluates orientation, immediate memory, concentration, and delayed memory recall.[5] Despite the advancement of neuroimaging techniques, it is important to remember that patients with cerebral concussion can have normal findings on both CT and MRI. The use of neuroimaging to rule out serious pathology must be done with caution and may require follow-up studies to ensure that delayed hemorrhage is not present. The introduction of single-photon emission computed tomography (SPECT) scans has yet to be proven useful as a diagnostic tool in traumatic head injury.

The occurrence of a fatal brain injury after minor head trauma has been documented in contact sports. This second-impact syndrome has historically been seen in football players, hockey players, and boxers. The typical scenario is a player who suffers a relatively minor blow to the head with mild concussion symptoms and then returns to competition within a week. The athlete then receives a second blow to the head and experiences rapid neurological demise and death within hours of this second head injury. While there are various theories on the cause of death in these cases, it appears that the initial trauma causes a loss of vasomotor tone, allowing an increase in intracranial volume. If the patient receives a second minor head impact during this brief period of decreased intracranial compliance, the diffuse and uncontrollable cerebral edema that subsequently occurs can be fatal.

Return-to-play decisions for the team physician are most difficult. Recently, the Department of Health and Human Services distributed an all-encompassing handout with both educational

TABLE 61.4
Guidelines for Management of Concussion in Sports

Grade	Features	Management	Return to Play
1	Confusion without amnesia	Remove from contest.	May return if asymptomatic at rest and with exertion for at least 20 minutes
	No loss of consciousness	Examine immediately and at 5-minute intervals for development of mental status abnormalities or post-concussive symptoms at rest with exertion.	Second grade 1 concussion in same contest: Disqualify athlete for that day Third grade 1 concussion: Terminate season
2	Confusion with amnesia	Remove from contest and disallow return that day.	May return after 1 full asymptomatic week at rest and with exertion
	No loss of consciousness	Examine on site frequently for signs of evolving intracranial pathology.	Second grade 2 concussion: Return to play after 1 month symptom free at rest and with exertion; consider termination of season
		CT scan or MR imaging if symptoms worsen or persist for longer than 1 week.	Third grade 2 concussion: Terminate season
3	Any loss of consciousness	Transport athlete to nearest emergency department by ambulance with cervical spine precautions, if necessary.	May return after 1 month if asymptomatic at rest and with exertion for at least 2 weeks Second grade-3 concussion: Terminate season; return to any contact sport seriously discouraged

Source: Adapted from Colorado Medical Society Sports Medicine Committee, *Guidelines for the Management of Concussion in Sports*, Denver, Colorado, Medical Society, 1991. With permission.

TABLE 61.5
Guidelines for the Management of Concussion in Sports

Grade	Features	Management	Return to Play
1	Transient confusion No loss of consciousness Concussion symptoms resolve in less than 15 minutes	Remove from contest. Examine immediately and at 5-minute intervals for development of mental status abnormalities or post-concussive symptoms at rest and with exertion.	Return if clear within 15 minutes Second grade 1 concussion in same contest: Disqualify athlete; allow return in 1 week if asymptomatic at rest and with exercise
2	Transient confusion No loss of consciousness	Remove from contest and disallow return that day. Examine on site frequently for signs of evolving intracranial pathology.	May return after 1 full asymptomatic week with exertion
	Concussion symptoms last more than 15 minutes	CT scan or MR imaging if symptoms worsen or persist for longer than 1 week	Second grade 2 concussion: Return to play after 2 weeks symptom free at rest and with exertion
3	Any loss of consciousness, either brief (seconds) or prolonged (minutes)	Transport athlete to nearest emergency department by ambulance with cervical spine precautions, if necessary.	Brief (seconds) grade 3 concussion: Withhold from play until asymptomatic for 2 weeks at rest and with exertion Second grade 3 concussion: Withhold from play for a minimum of 1 asymptomatic month

Source: Adapted from Kelly, J.P. and Rosenburg, J.H., Practice parameter: the management of concussion in sports, *Neurology*, 48, 575–580, 1997. With permission.

TABLE 61.6
Management of Concussion in Sports

Grades of Concussion

Grade 1:

1. Transient confusion (inattention, inability to maintain a coherent stream of thought and carry out goal-directed movements)
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities on examination resolve in **less than 15 minutes**

Grade 2:

1. Transient confusion
2. No loss of consciousness
3. Concussion symptoms or mental status abnormalities (including amnesia) on examination last **more than 15 minutes**

Grade 3:

1. Any loss of consciousness
   a. Brief (seconds)
   b. Prolonged (minutes)

Management Recommendations

Grade 1:

1. Remove from contest
2. Examine immediately and at 5-minute intervals for the development of mental status abnormalities or post-concussive symptoms clear within 15 minutes

Grade 2:

1. Remove from contest and disallow return that day
2. Examine on-site frequently for signs of evolving intracranial pathology
3. A trained person should reexamine the athlete the following day
4. A physician should perform a neurologic examination to clear the athlete for return to play after 1 full asymptomatic week at rest and with exertion

Grade 3:

1. Transport the athlete from the field to the nearest emergency department by ambulance if still unconscious or if worrisome signs are detected (with cervical spine immobilization, if indicated)
2. A thorough neurologic evaluation should be performed emergently, including appropriate neuroimaging procedures when indicated
3. Hospital admission is indicated if any signs of pathology are detected, or if the mental status of the athlete remains abnormal

When to Return to Play

Grade of Concussion	Return to Play Only After Being Asymptomatic with Normal Neurologic Assessment at Rest and with Exercise
Grade 1 Concussion	15 minutes or less
Multiple Grade 1 Concussions	1 week
Grade 2 Concussion	1 week
Multiple Grade 2 Concussions	2 weeks
Grade 3EMDASHBrief Loss of Consciousness (seconds)	1 week
Grade 3EMDASHProlonged Loss of Consciousness (minutes)	2 weeks
Multiple Grade 3 Concussions	1 month or longer, based on decision of evaluating physician

materials and guidelines for providers encountering patients suffering from MTBI.[6] A useful handheld card with concussion grading, management recommendations, and return-to-play criteria is shown in Table 61.6. This palm card combines criteria from the multitude of guidelines published to date. While each has specific variations of definitions, etc., most guidelines still classify concussions into one of three grades for definition and for post-concussion medical management

(Tables 61.3 to 61.5) (Level of Evidence C, consensus/expert opinion). In each guideline, an athlete must be asymptomatic for a specific period of time prior to returning to competition. The commonly used definition of asymptomatic is an athlete without any somatic, behavioral, or cognitive symptoms either at rest or with exertion. The recent Concussion in Sport Group guideline emphasizes the importance of a step-be-step progression to an asymptomatic state, with any recurrence of symptoms necessitating a return to a lower level of activity.[7] The use of baseline and post-concussive neuropsychological testing is also currently being researched as a clinical tool in assisting providers in making return-to-play decisions.

POST-CONCUSSIVE SYNDROME

Post-concussive syndrome generally follows a mild injury and may have one or more symptoms. The most common symptoms are headaches, dizziness, tinnitus, diplopia, blurred vision, irritability, anxiety, depression, fatigue, sleep disturbance, poor appetite, poor memory, impaired concentration, and slowed reaction times. These symptoms are likely secondary to diffuse neuronal injury and possibly even vestibular damage. The frequency of post-concussive syndrome is difficult to determine and is affected by many factors. At the same time, controversy exists regarding the relationship between the duration of posttraumatic amnesia and the persistence of post-concussive symptoms. Because the symptoms are so wide ranging and neurologic examination is usually normal, neuropsychological testing and treatment are indicated in any post-traumatic head injury athlete who is suspected of having neuropsychological symptoms.

Testing and treatment of post-concussive athletes includes standard neuroimaging along with more specific evaluations to include audiologic testing, electronystagmography, posturography, and a battery of tests to evaluate everything from orientation and attention to visual scanning and motor coordination. At the collegiate and professional level, many of these tests are used to set baseline parameters for each athlete, thereby ensuring that any deficits due to post-traumatic head injuries are well documented and appropriately treated prior to return to play.

Treatment is individualized to the specific complaint and can include analgesics, NSAIDs, antidepressants, and muscle relaxants (Table 61.2). More importantly, general education and reassurance that most patients with post-concussive syndrome have complete resolution of symptoms within 3 to 6 months is a vital component to any therapeutic regimen.

SUMMARY

Parents, trainers, and team physicians should understand the importance of timely and accurate assessment of athletes with headaches and those suffering from head trauma can be vital to their successful recovery. Applying this simple assessment algorithm should ensure that each patient receives the appropriate level of evaluation and treatment.

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62 CARDIOVASCULAR CONSIDERATIONS IN THE ATHLETE

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INTRODUCTION

Enhanced cardiovascular health is one of the key benefits of most forms of consistent athletic endeavors throughout life. Regular physical activity promotes cardiovascular fitness and lowers risk of disease. While generally a net cardiovascular benefit can be gained from athletic activity, an increased cardiovascular risk can exist for certain susceptible individuals. These individuals may be known and have identified cardiovascular disorders or they may be unrecognized until the adverse event occurs.

CARDIOVASCULAR BENEFITS OF EXERCISE

Numerous studies[1–5] have clearly identified physical inactivity and sedentary lifestyle as significant risk factors for the development and progression of coronary heart disease and for the incidence of adverse cardiovascular events, including death. Moreover, other studies have consistently confirmed the cardiovascular benefit of aerobic exercise, with a reduction in the number of adverse events and a reduction in mortality (Level of Evidence A, randomized controlled trials).[6–9] While a definite increased risk exists for certain susceptible individuals, particularly middle-aged persons with coronary artery disease and a sedentary lifestyle who begin a sudden intensive exercise program or young athletes with congenital or genetic cardiovascular disorders, abundant evidence[10] has been found of net cardiovascular benefits gained from consistent vigorous exercise as a primary prevention recommendation for coronary disease in asymptomatic middle-aged and older persons. The individuals at increased risk need to be identified, stratified, counseled, and guided to an appropriate activity level. Most others need to be encouraged, motivated, and supported in their efforts to achieve a vigorous lifestyle that will reduce cardiovascular risk and promote optimal physical functioning throughout life.

THE ATHLETIC HEART SYNDROME

Vigorous athletic training is associated with some specific physiologic and structural cardiovascular changes,[11] which comprise what has been termed the *athletic heart syndrome*. These changes are non-pathologic and represent appropriate adaptations to physical training. Studies[12] have suggested the existence of a continuum of athletic adaptations depending on the training stress of the athlete — generally, endurance-trained changes, strength-trained changes, and combined endurance- and strength-trained changes. For endurance-trained athletes, the heart has to adapt to principally a chronic volume overload, which results in an increase in both left ventricular end-diastolic diameter and left ventricular wall thickness. This results in eccentric hypertrophy with a more pronounced increase in wall thickness than expected. The strength-trained athletes adapt by

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developing a concentric hypertrophy with an increase in absolute and relative wall thickness without significant changes in end-diastolic diameter. This is secondary to a large increase in pressure load without a large increase in volume load.

The heart rate of well-conditioned athletes is usually between 40 to 60 beats per minute, secondary to enhanced vagal tone and decreased sympathetic tone. Normal sinus arrhythmia may be more noticeable. The physiologic splitting of S2 may be slightly delayed during inspiration. An S3 may be noted in endurance-trained athletes secondary to the increased rate of left ventricular filling associated with the relative left ventricular dilatation.[13] While an S4 may be noted in strength-trained athletes secondary to concentric hypertrophy, its presence always warrants clinical evaluation. Functional murmurs may be noted in 30 to 50% of athletes on careful exam.[14]

Several minor electrocardiographic variations have been commonly noted in highly trained athletes and are considered to be consistent with the athlete's heart syndrome.[14,15] These minor alterations include increased PR interval duration, mild increase in R or S voltage (25 to 29 mm), early repolarization (ST elevation > 2 mm in >2 leads), incomplete RBBB (RSR1 pattern in V1 and V2 of <0.12 seconds in duration), and sinus bradycardia. More significantly, abnormal-appearing electrocardiogram (EKG) patterns have also been identified in otherwise normal athletic hearts. This is especially frequent in younger, male participants in endurance-trained activities. In a recent Italian study,[16] 1005 athletes were consecutively assessed with EKGs and echocardiograms. The study found that 40% of the athletes had abnormal EKGs, not including the minor alterations associated with the athlete's heart syndrome mentioned above. Of these athletes, 36% had distinctly abnormal patterns. Of those with the distinctly abnormal patterns, only 10% actually had evidence of structural cardiac disease, suggesting that the EKG manifestation of the normal athletic heart is much more variable than previously believed.

It is important to remember that the adaptive structural and physiologic responses of the normal athletic heart do not rule out the presence of an underlying pathologic condition. In fact, it makes the task of diagnosing that condition more challenging for the primary care physician and cardiologist. Of note, detraining for 2 to 3 months can result in a reversal of athletic heart syndrome changes which is not seen in pathologic conditions. Each athlete must be assessed individually with an acknowledgment that EKGs and echocardiograms must be interpreted with full attention to symptoms, family history, and physical examination.

SUDDEN DEATH WITH EXERCISE

As mentioned earlier, while considerable net cardiovascular benefit can be gained from exercise, a clear risk does exist for susceptible individuals. Indeed, as Maron[10] has clearly shown, a "paradox of exercise" requires a clinical assessment of risk prior to the initiation of a vigorous program. Overall risk of sudden death during exercise is low. Estimates from various studies[17–22] range from 1:15,000 joggers per year[17,19] to 1:50,000 marathon participants.[20] For high school and college age athletes, the range is estimated at 1:200,000 to 1:300,000 per academic year.21–22 Of course, the risk of sudden death with exercise increases with age. As the demographics of the population continue to shift and more middle-aged and elderly heed the advice to exercise, it should certainly be expected to see an increase in the prevalence of sudden death. In the older athlete, coronary artery disease is the most common etiology. In the younger athlete, multiple etiologies, including congenital abnormalities, can also lead to malignant arrhythmias during intense activity. Generally, it is the combination of underlying cardiac disease (recognized or unrecognized) with intense exercise that leads to the fatal arrhythmia in both age groups.[23] For young athletes, basketball, football, track, and soccer have the highest incidence of sudden deaths. Cardiovascular etiologies outnumber traumatic etiologies nearly 2 to 1 for athletic-related fatalities in high school and college.[24]

The specific etiologies contributing to sudden cardiac death are most closely related to age. Generally, the dividing age is 35.[25] This primarily stems from the observation that, for sudden deaths over age 35, more than 75% are associated with coronary artery disease. The high prevalence of atherosclerosis in this age group clearly predominates as an etiology. In younger athletes, hypertrophic cardiomyopathy (HCM) is the most common etiology. Coronary artery anomalies, premature atherosclerotic disease, myocarditis, and dilated cardiomyopathy are the next most common, at least in the United States. In European studies,[26–28] arrhythmogenic right ventricular dysplasia is more commonly recognized as an etiology than it is in the United States. Other less common etiologies include aortic rupture from

Marfan's syndrome, genetic conduction system abnormalities, idiopathic left ventricular hypertrophy, substance abuse (cocaine and/or steroids), aortic stenosis, mitral valve prolapse, sickle cell trait, and blunt chest trauma (commotio cordis).

Screening for Sudden Death

As alluded to above, screening for the risk factors associated with sudden death requires attention to the subtle details of personal and family history, a careful cardiovascular exam, and directed ancillary studies. The American Heart Association (AHA) Science and Advisory Committee published consensus guidelines for preparticipation cardiovascular screening for high school and college athletes in 1996 (Level of Evidence C, expert/consensus opinion).[29] It is recommended that a complete personal and family history and physical examination be done for all athletes. It should focus on identifying those cardiovascular conditions known to cause sudden death and should be done every 2 years with an interim history between exams. The 26th Bethesda Conference specifies participation guidelines for different conditions.[30] These are summarized in Table 62.1.

It is the primary care physician's responsibility to attempt to identify and assess those conditions through a thorough history and physical and to promptly refer suspected cases to the cardiology consultant for definitive diagnosis and specific exercise recommendations and limitations when required. Family history should include a specific inquiry for a family history of premature coronary artery disease, diabetes mellitus, hypertension, sudden death, syncope, significant disability from cardiovascular disease in relatives younger than age 50, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy (ARVC), Marfan's syndrome, prolonged QT syndrome, or significant arrhythmias. Personal past history should include specific inquiries regarding the detection of heart murmur, diabetes mellitus, hypertension, hyperlipidemia, smoking, or the presence of HCM, ARVC, Marfan's syndrome, prolonged QT syndrome, or significant arrhythmias. Recent history inquiries must include a history of syncope, near syncope, profound exercise intolerance, exertional chest discomfort, dyspnea, or excessive fatigue.

Physical exam should specifically address hypertension, heart rhythm, cardiac murmur, and the findings of unusual facies or body habitus associated with a congenital cardiovascular defect, especially Marfan's syndrome.[31] Cardiac auscultation should be performed in the supine and standing positions and murmurs should be assessed with Valsava and position maneuvers when indicated. Femoral pulses should be assessed and blood pressure measured with the appropriately sized cuff in the sitting position.

TABLE 62.1
Guidelines on Restriction of Exercise for Cardiovascular Disease

Contraindications to vigorous exercise
Hypertrophic cardiomyopathy
Idiopathic concentric left ventricular hypertrophy
Marfan's syndrome
Coronary heart disease
Uncontrolled ventricular arrhythmias
Severe valvular heart disease (especially aortic stenosis and pulmonic stenosis)
Coarctation of the aorta
Acute myocarditis
Dilated cardiomyopathy
Congestive heart failure
Congenital anomalies of the coronary arteries
Cyanotic congenital heart disease
Pulmonary hypertension
Right ventricular cardiomyopathy
Ebstein's anomaly of the tricuspid valve
Idiopathic long Q-T syndrome
Require close monitoring and possible restriction
Uncontrolled hypertension
Uncontrolled atrial arrhythmias
Hemodynamic significant valvular heart disease (aortic insufficiency, mitral stenosis, mitral regurgitation)

Source: Adapted from 26th Bethesda Conference. J. Am. Coll. Cardiol. 24, 845-899, 1994.30,33[GR1]

Ancillary testing should be directed by the patient's history, physical, and age. Lipid profiles should be checked in the older athlete and should be considered in athletes of any age. Exercise stress testing is not recommended as a routine screening device for the detection of early coronary artery disease because of low predictive value and high rates of false positive and false negative results. However, a recent AHA Science Advisory[32] recommends stress testing on those older athletes with a moderate to high cardiovascular risk profile for coronary artery disease. Specifically, this includes men >40 to 45 years old or

women >50 to 55 years old (or postmenopausal) with one or more independent coronary risk factors. The risk factors are as follows: (1) total cholesterol > 200, (2) LDL > 130, (3) HDL < 35 for men and < 45 for women, (4) systolic BP >140 or diastolic BP > 90, (5) current or recent smoker, (6) diabetes mellitus, and (7) history of myocardial infarction or sudden death in first-degree relative < 60 years old. In addition, stress testing is recommended for athletes of any age with suggestive symptoms of coronary artery disease or for those older than 64 years, regardless of risk factors or symptoms.

Electrocardiograms and echocardiograms are not currently recommended as screening tools (Level of Evidence B, systematic reviews).[25,33,34] As mentioned earlier, the normal adaptations of the athletic heart make interpretation of the routine EKG and echocardiogram problematic.[16] Determining the role of these modalities lies in the hands of the cardiology consultant, who must interpret the findings in the setting of the individual patient who has been referred for questionable symptoms or physical findings. At this point, the state of technology does not allow for significant sensitivity to cost effectively find true positives and reliably exclude true negatives. The primary care physician should not rely on these tools to "find" patients. High rates of false positivity, high relative costs, limited availability, and low prevalence of disease make these modalities impractical as screening devices at this point in time.

SYNCOPE AND EXERCISE-ASSOCIATED COLLAPSE

Syncope is most often defined as a sudden loss of consciousness for a brief duration, not secondary to head trauma but usually secondary to a sudden drop in cerebral blood flow or metabolic change (e.g., hypoglycemia, hypoxia). Exercise-associated collapse (EAC) refers to athletes who are unable to stand or walk unaided because of lightheadedness, faintness, dizziness, or outright syncope. The potential differential diagnosis is extensive and includes multiple cardiovascular and neurologic etiologies.[35–36] Athletes who present with a history of passing out with exercise require a careful history and physical to differentiate benign from life threatening etiologies.[34]

The first step involves determining if the event was a brief, true syncopal episode vs. the more common and generally benign EAC event that involves a longer time period of being out of it, even in the supine position with normal vital signs. The second step is to differentiate between syncope that occurs during the event (suggesting a more ominous arrhythmic etiology) and syncope that occurs following the event, usually associated with orthostatic hypotension upon exercise cessation (suggesting a less ominous etiology). It is also critical to identify prodromal symptoms that may have occurred during exercise such as palpitations (arrhythmia), chest pain (ischemia or aortic dissection), nausea (ischemia or vagal activity), wheezing, or pruritus (anaphylaxis).

The physical exam should include a careful assessment of orthostatic vital signs, precordial auscultation especially focusing on ruling out the murmurs of aortic stenosis and HCM, and a careful search for the morphologic features of Marfan's syndrome. An EKG should be ordered in most cases and should be evaluated closely for rate, rhythm, QT interval, repolarization abnormalities, left or right hypertrophy, preexcitation evidence, and complications of ischemic heart disease. Further testing, including blood work, echocardiogram, and stress testing, may be done depending on whether a diagnosis has been made, suggested, or remains unexplained. See Figure 62.1 for a suggested algorithm for the primary care evaluation of exertional syncope in the young athlete (Level of Evidence C, expert opinion).[34] If the etiology is diagnosed or strongly suggested, then the athlete may be reassured, restricted, or referred for further testing depending on the etiology. If the event remains unexplained, then the athlete must remain restricted and undergo further testing with echocardiography, stress testing, and cardiac consultation.

HYPERTENSION IN THE ATHLETE

Systemic hypertension remains one of the most common life-threatening cardiovascular disorders in the United States and affects athletes of all ages and sports. The primary care physician will frequently encounter opportunities to newly diagnose the condition in young athletes during the preparticipation examination process and to manage established hypertensives of all ages engaged in various sporting activities. The diagnosis, work-up, and initial non-pharmacologic approach to treatment does not differ between athletes and non-athletes. This approach is well described in the JNC-VI recommendations (Level of Evidence C, expert consensus opinion).[37] Care must be taken not to overdiagnose the condition in young athletes and to utilize proper-fitting cuffs with three different measures on three different days,

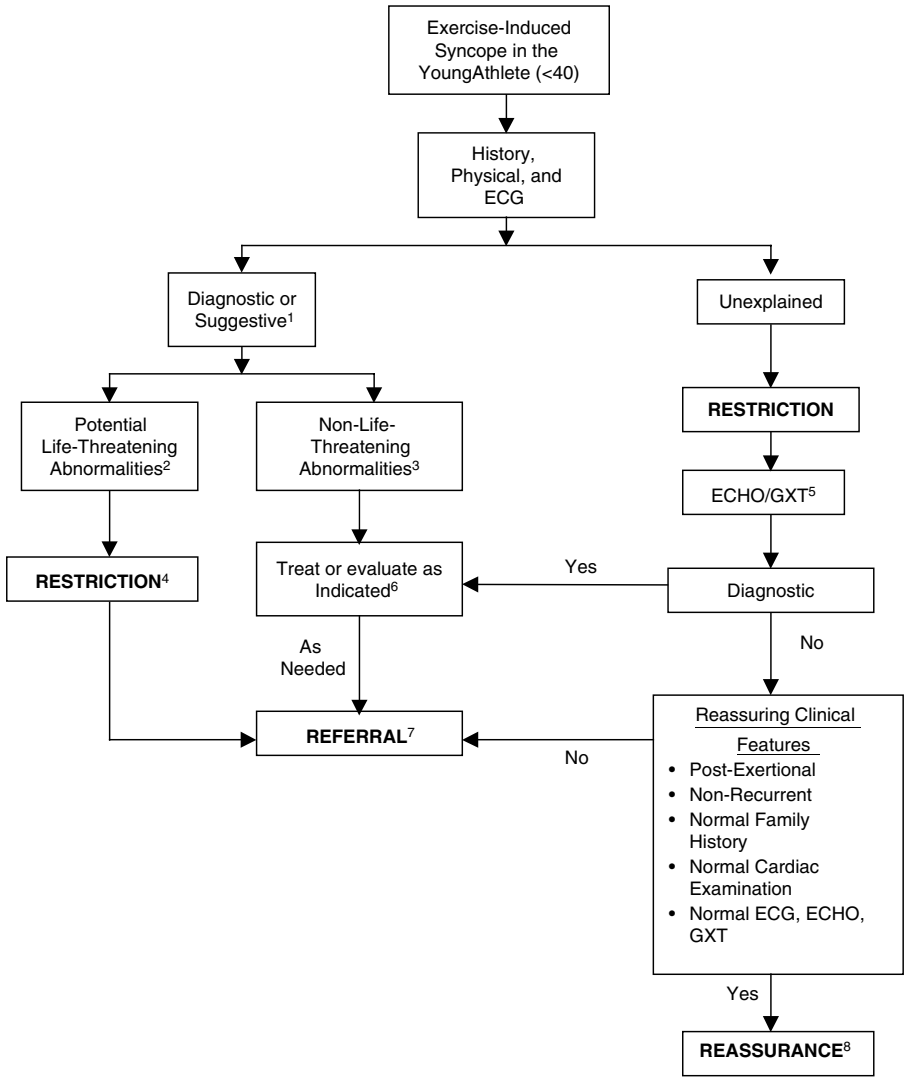

Figure 62.1 Algorithm for the primary care evaluation of exertional syncope in the athlete under 40 years of age. *Diagnostic or Suggestive:* The history, physical examination, and electrocardiographic analysis result in a definitive or presumptive diagnosis (e.g., hypertrophic cardiomyopathy, exertional hyponatremia). Potential life-threatening diagnoses may include hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and heat stroke. Non-life-threatening diagnoses may include hypoglycemia, mild hyponatremia, and mild heat exhaustion. *Restriction:* This individual should be restricted from strenuous/vigorous exercise pending completion of the syncope evaluation. An echocardiogram and exercise stress test are warranted in all cases of unexplained exertional syncope to include post-exertional syncope. Echocardiography should precede exercise stress testing. The diagnostic evaluation should be ordered as indicated according to the diagnosis being entertained; this may be in consultation with a cardiologist, neurologist, and/or psychiatrist. Temporary restriction from vigorous activity should be considered on an individual basis. *Referral:* Consultation is warranted and may include Holter or event monitoring, tilt-table testing, electrophysiologic studies, coronary angiography, electrophysiologic studies, cardiac and/or brain MRI, electroencephalography, and/or psychiatric testing. *Reassurance:* The athlete may return to vigorous activity with an appropriate follow-up plan.

TABLE 62.2
Classification of Hypertension (boys & girls combined) (mm Hg)

Age (years)		High Normal BP (90th-94th percentile)	Significant HTN (95th-98th percentile)	Severe HTN (99th percentile)
6-9	Systolic	111–121	122–129	>129 (129)[a]
	Diastolic	70–77	70–85	>85 (84)
10-12	Systolic	117–125	126–133	>133 (134)
	Diastolic	75–81	82–89	>89 (89)
13-15	Systolic	124–135	136–143	>143 (149)
	Diastolic	77–85	86–91	>91 (94)
16-18	Systolic	127–141	142–149	>149 (159)
	Diastolic	80–91	92–97	>97 (99)
>18	Systolic not given		[140–179][b]	>(179)
	Diastolic not given		[90–109]	>(109)

[a] The values in parentheses are those used for the classification of severe hypertension by the 26th Bethesda Conference on cardiovascular disease and atheletic participation[30]

[b] The values in brackets are those for mild and moderate hypertension given by the 26th Bethesda Conference[30]

Source: Adapted from Committee on Sports Medicine and Fitness, *Pediatrics,* 99(4), 1997.

adjusting for norms for age, weight, and height.[38] An appropriate search for secondary etiologies and target-organ damage assessment should guide the history, physical, and laboratory evaluation. The history should include an inquiry about performance enhancing substances (e.g., anabolic steroids) and labs should include EKG, urinalysis, complete blood count, electrolytes, fasting glucose, lipid profile, blood urea nitrogen (BUN), creatinine, and uric acid. It often includes a chest x-ray and echocardiogram to assess for left ventricular hypertrophy as well as a stress test to assist in determining the intensity level of activity participation.[30,39]

Non-pharmacologic treatment should be properly initiated with enthusiastic physician endorsement.[40–41] It includes engagement in moderate physical activity, maintenance of ideal body weight, limitation of alcohol, reduction in sodium intake, maintenance of adequate potassium intake, and consumption of a diet high in fruit and vegetables and low in total and saturated fat. When indicated, pharmacologic treatment should be initiated. Generally, angiotensin-converting enzyme (ACE) inhibitors, calcium channel blockers, and angiotensin-II receptor blockers are excellent choices for athletes with hypertension. Their low side-effect profiles and favorable physiologic hemodynamics make them generally safe and effective. It is preferable to avoid diuretics and beta-blockers in young athletes. Volume and potassium balance issues limit diuretic use, and beta-blockers adversely impact the cardiovascular training effect of exercise.[33–34] Both substances, as well as a number of other antihypertensives, are banned by the National Collegiate Athletic Association and the U.S. Olympic Committee.[42]

Restriction of activity for athletes with hypertension depends on the degree of target organ damage and on the overall control of the blood pressure.[30,43] Most patients who have controlled BP (<140/90 at rest for adults) and are mild to moderate hypertensives with no target organ damage can have unrestricted participation.[32] Adult patients with target organ damage, uncontrolled blood pressure, or severe but controlled hypertension should be restricted to lower intensity sports. In children and adolescents, the presence of severe hypertension or target organ disease warrants restriction until hypertension is under adequate control. The presence of significant hypertension should not limit a person's eligibility for competitive athletics (Table 62.2) (Level of Evidence C, expert consensus opinion).

CARDIAC ARRHYTHMIAS IN THE ATHLETE

As mentioned earlier, lethal cardiac arrhythmias represent the most serious risk for sudden death in athletes. It is absolutely essential that the symptoms of a potential arrhythmia be taken seriously and thoroughly evaluated. These include the symptoms of syncope, near syncope, palpitations, exertional chest discomfort, severe dyspnea, or

TABLE 62.3
Activity Recommendations for the Common Dysrhythmias

Disturbances of sinus node function (includes sinus bradycardia, tachycardia, arrhythmia, arrest, exit block; wandering pacemaker; sick sinus syndrome)
No symptoms, no treatment; if symptoms require pacemaker, then no collision sports

Premature atrial complexes
No restrictions

Atrial flutter and atrial fibrillation
If no structural heart disease and rate is controlled by drugs, then low-intensity sports; if no flutter or fibrillation for 6 months, then full activity

Supraventricular tachycardia
If episodes are prevented by drugs, then full participation; if structural disease and if syncope or presyncope, no competitive sports; reconsider after 6 months without recurrence

Ventricular pre-excitation (WPW)
If no structural heart disease and no symptoms, then no limit; if structural heart disease and PVCs worsen with exercise, restrict; PVCs plus prolonged Q–T interval should be restricted (high risk for sudden death)

Heart blocks (first-degree or Mobitz I second-degree)
If no symptoms and no structural disease, then no restrictions

Heart blocks (Mobitz II second-degree or third-degree)
If no symptoms and no structural disease, then no restrictions if rate 40–80; if symptoms, then pacer and avoidance of collision sports

Congenital long Q-T syndrome
At risk for sudden death; restricted from all competitive sports

Source: Maron, B.J., Mitchell, J.H., Eds., 26th Bethesda Conference recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities, *Am. J. Cardiol.,* 24, 845-899, 1994, and Kugler, J.P. and O'Connor, F.G., Cardiovascular problems, in *Handbook of Sports Medicine,* 2nd ed., Lillegard, W.A., Butcher, J.D., and Rucker, K.S., Eds., Butterworth-Heinemann, Boston, MA, 1999, p. 339. With permission.

TABLE 62.4
Stratification Categories for CAD Patients by 26th Bethesda Conference

Mildly Increased Risk
- Normal or near normal resting left ventricular systolic function (i.e., ejection fraction >50%);
- Normal exercise tolerance for age (>10 METs if aged <50; >9 METs for ages 50 to 59; >8 METs for ages 60 to 69; and >7 METs if aged >70);
- Absence of exercise-induced ischemia by exercise testing;
- Absence of exercise-induced complex ventricular arrhythmias; absence of hemodynamically significant stenosis in all major coronary arteries if coronary angiography is performed; or successful myocardial revascularization by surgical or percutaneous techniques.

Substantially Increased Risk
- Impaired left ventricular systolic function at rest (i.e., ejection fraction <50%);
- Evidence of exercise-induced myocardial ischemia;
- Evidence of exercise-induced complex ventricular arrhythmias;
- Hemodynamically significant stenosis of a major coronary artery (>50%) if angiography was performed.

Source: Maron, B.J., Mitchell, J.H., Eds., 26th Bethesda Conference recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities, *Am. J. Cardiol.,* 24, 845-899, 1994, and Kugler, J.P., O'Connor, F.G., and Oriscello, R.G., in *Textbook of Running Medicine,* O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, p. 341. With permission.

uncommon exertional fatigue. Structural heart disease must be ruled out before the athlete is allowed to return to sports.[30,33,38] This will include a meticulous history, physical examination, and EKG and may be followed by chest x-ray, echocardiogram, stress test, Holter monitoring, electrolytes, and other laboratory testing. It may very well include early referral to a cardiologist for electrophysiologic study and/or ongoing management.

Various arrhythmias are compatible with competitive sports when they have been diagnosed and controlled. Other conditions may be clearly incompatible with vigorous activity. Symptomatic dysrhythmias require evaluation and consultation with a cardiologist (Level of Evidence C, expert consensus opinion).[44] See Table 62.3 for a summary of the common dysrhythmias and the recommendations from the 26th Bethesda Conference.[30] The Committee on Sports Medicine and Fitness for the American Academy of Pediatrics specifically recommends that the presence of a symptomatic dysrhythmia requires exclusion from physical activity until the problem can be adequately evaluated by a cardiologist and controlled.[44]

CORONARY ARTERY DISEASE IN THE ATHLETE

As discussed in some detail earlier in the chapter, vigorous exercise represents a dangerous paradox for cardiovascular disease.[10] While it may be a potent preventive tool, it can also represent substantial risk for the susceptible individual. This is particularly poignant for the athlete with an established diagnosis of coronary artery disease (CAD). These individuals will absolutely require careful risk stratification prior to returning to their active lifestyle.[34] They will require procedures for left

TABLE 62.5
Summary of 26th Bethesda Conference Recommendations for Patients with Coronary Artery Disease

General

1. All athletes should understand that the risk of a cardiac event with exertion is probably increased once coronary artery disease is present.
2. Athletes should be informed of the nature of prodromal symptoms and should be instructed to promptly cease their sports activity and contact their physician if symptoms appear.

Specific

1. Mildly increased risk. May participate in low and moderate static and low dynamic competitive sports (IA and IIA) and avoid intensely competitive situations.
2. Substantially increased risk. May participate in low-intensity competitive sports (IA) after careful assessment and individualization. These patients should be reevaluated every 6 months and should undergo repeat exercise testing at least yearly.

Source: Maron, B.J., Mitchell, J.H., Eds., 26th Bethesda Conference recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities, Am. J. Cardiol., 24, 845-899, 1994, and Kugler, J.P., O'Connor, F.G., and Oriscello, R.G., in Textbook of Running Medicine, O'Connor, F.G. and Wilder, R.P., Eds., McGraw-Hill, New York, 2001, p. 341. With permission.

TABLE 62.6
Signs and Symptoms Below Which an Upper Limit for Exercise Intensity Should Be Set[a]

Onset of angina or other symptoms of cardiovascular insuffciency.

Plateau or decrease in systolic blood pressure, systolic blood pressure of >240 mm Hg, or diastolic blood pressure of >110 mm Hg.

Greater than or equal to 1 mm ST-segment depression, horizontal or downsloping.

Radionuclide evidence of left ventricular dysfunction or onset of moderate-to severe wall motion abnormalities during exertion.

Increased frequency of ventricular arrhythmias.

Other significant ECG disturbances (e.g., second degree or third degree AV block, atrial fibrillation, supraventricular tachycardia, complex ventricular ectopy, etc.).

Other signs/symptoms of intolerance to exercise.

[a] The peak exercise rate should generally be at least 10 beats per minute below the heart rate associated with any of the above-referenced criteria. Other variables (e.g., the corresponding systolic blood pressure response and perceived exertion), however, should also be considered when establishing the exercise intensity.

Source: American College of Sports Medicine, ACSM's Guidelines for Exercise Testing and Prescription, Sixth ed., Lippincott Williams & Wilkins, Philadelphia, PA, 2000, pp. 165-199.

ventricular assessment, maximal treadmill testing to determine functional capacity, and testing for inducible ischemia. Patients should be tested on their medications. The 26th Bethesda Conference[30] defines clear stratification criteria (Table 62.4), accompanied by activity recommendations (Table 62.5) (Level of Evidence C, expert consensus opinion). This provides a general and conservative approach to the individual in regard to competitive sports.

The recommendations, however, are particularly restrictive and do not address the casual participant and the individual jogger. This puts the primary responsibility on the primary care physician. It requires an individualized activity, risk factor modification, and rehabilitation program that is monitored by the primary care physician in close consultation with a cardiologist in order to safely reintegrate these patients back into a reasonable and active lifestyle. The American College of Sports Medicine has recently published guidelines that assist the primary care physician in guiding the level of aerobic intensity (Table 62.6) (Level of Evidence C, consensus expert opinion).[45]

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APPENDICES

APPENDIX 1

SPORTS MEDICINE RESOURCE GUIDES

AMERICAN COLLEGE OF SPORTS MEDICINE

Street address:
401 West Michigan Street
Indianapolis, IN 46202-3233

Mailing address:
P.O. Box 1440
Indianapolis, IN 46206-1440

Telephone:
National Center — 317-637-9200
Regional Chapter Resource Center: 317-637-9200, ext. 138

Fax:
317-634-7817

Website:
www.acsm.org

Position Stands and Joint Position Statements

March 1, 2002
position stand
Automated External Defibrillators in Health/Fitness Facilities

February 2, 2002
position stand
Progression Models in Resistance Training for Healthy Adults

December 1, 2001
position stand
Appropriate Intervention Strategies for Weight Loss and Prevention of Weight Regain for Adults

December 1, 2000
joint position statement
Nutrition and Athletic Performance

July 1, 2000
position stand
Exercise and Type 2 Diabetes

June 1, 1998
position stand
The Recommended Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory and Muscular Fitness, and Flexibility in Healthy Adults

position stand
Exercise and Physical Activity for Older Adults

joint position statement
AHA/ACSM Joint Statement: Recommendations for Cardiovascular Screening, Staffing, and Emergency Policies at Health/Fitness Facilities

December 1, 1997
joint position statement
ADA/ACSM Joint Statement: Diabetes Mellitus and Exercise

May 1, 1997
position stand
The Female Athlete Triad

December 1, 1996
position stand
Heat and Cold Illnesses During Distance Running

January 1, 1996
position stand
Exercise and Fluid Replacement

Consensus Statements

- Sideline Preparedness for the Team Physician: A Consensus Statement
- Team Physician Consensus Statement
- 4th USOC/ACSM Consensus Statement on "Overtraining"
- The Team Physician and Conditioning of Athletes for Sports: A Consensus Statement

AMERICAN MEDICAL SOCIETY FOR SPORTS MEDICINE

Street and mailing address:
11639 Earnshaw
Overland Park, KS 66210

Telephone:
913-327-1415

Fax:
913-327-1491

Website:
www.amssm.org

Position Statements

- HIV and Other Blood-Borne Pathogens in Sports Position Statement
- Team Physician Consensus Statement
- Team Physician and Conditioning of Athletes for Sports: A Consensus Statement
- Sideline Preparedness for the Team Physician: A Consensus Statement

APPENDIX 2

TYPES OF SPORTS

Category I: Collision

Boxing

Football, tackle

Martial arts, full contact

Category II: Contact

Basketball

Baseball/softball

Wrestling

Soccer

Rugby

Lacrosse

Judo

Football, flag

Martial arts

Volleyball

Parachuting

Hockey

Cricket

Category III: Endurance

Trampolining

Track and field events

Jogging/walking

Sledding/toboganning/luge

Racquet sports

Skateboarding

Cycling

Surfing

Swimming

Skating (roller/ice)

Rowing

Diving, scuba

Skiing

Water polo

Gymnastics

Handball
Climbing/hiking/mountaineering/orienteering
Ultimate Frisbee
Kayak
Dance
Cheerleading
Auto racing

Category IV: Nonendurance/Sedentary

Golf
Bowling
Ballooning
Croquet
Gliding
Fishing
Flying
Yoga
Sailing
Diving
Shooting
Weightlifting
Horseshoe pitching
Caving
Camping
Billiards/snooker
Archery
Equestrian activities

Note: Classification represents a summary of average performance levels. Many sports could be placed in alternate categories under special circumstances. For example, the use of light weights numerous times during weightlifting would classify weightlifting as an endurance sport (i.e., circuit weight training), and the aerobic practice of a soft martial arts style such as Pakua would make it an endurance sport.

APPENDIX 3

EDUCATIONAL AND ORGANIZATIONAL RESOURCES

- Clinic Directory
- Fellowship Directory
- Sports Medicine Groups
- Sports Medicine Links

The Physician and Sportsmedicine (March issues)
4530 W. 77th Street
Minneapolis, MN 55435

Telephone: 952-835-3222
Fax: 952-835-3460
Website: www.physsportsmed.com

APPENDIX 4

MEDICAL SUPPLIES AND EQUIPMENT

First Aid Supplies

Examination gloves
Notebook and pencils
First-aid manual
Various sizes of bandages
Gauze pads 2 × 2, 4 × 4 inch
Gauze rolls 2- and 4-inch
Adhesive tape: 1/2 × 2 inch
Scissors (surgical and 3-inch straight)
Elastic underwrap
Cotton
Razor and shaving cream
Finger splints: 1/2- and 2-inch
Tongue depressors
Wound irrigation materials:
    Povidone-iodine solution (Betadine®)/hydrogen peroxide
    Sterile normal saline
    10- to 50-cc syringes
Antibiotic ointment (Bacitracin® or Betadine®)
Alcohol swabs
Nail clippers
Tourniquet
Acetaminophen (Tylenol®)
Aspirin
NSAID of choice
Ammonia smelling salts
Saline in plastic squeeze bottle
Water in plastic squeeze bottle
Ethyl chloride spray
Petroleum jelly
Flashlight/penlight
Benzoin spray (firm grip)
Tape remover

Dental cotton rolls
Assorted felt/foam rubber
Container for sharps
List of contents

First Aid Equipment

Double-action bolt cutter (1)
Screwdrivers: Phillips, regular (2)
Stretcher (1)
Backboard (short aluminum recommended) (1)
Blankets, Army (2)
Blanket, Mylar space (1)
Sandbags, 4- to 5-pound (4)
Cervical collar, semi-rigid (1)
Crutches, adjustable lengths (2 pairs)
Slings, plus safety pins as required for muslin type (3)
Splints, upper and lower extremity (2 each)
Sling psychrometer or a device with humidity/temperature/activity risk chart (1)
Knee immobilizer
Elastic bandages: 3-, 4-, and 6-inch (12 each)
Ice, with small plastic bags to contain it
Styptic pencil
Weight scale
Chart for recording athletes' weights
Water containers, 5-gallon (2)
Towels
Thomas 1/2 ring
Tables for examination, taping, treatments
Refrigerator
Whirlpool bath(s)
Telephone (coins available if pay phone)
Bulletin board for emergency phone numbers
List of phone numbers of each athlete's parents or guardian
Locked cabinet for medication
Sphygmomanometer (appropriate sizes)
Stethoscope
Goniometer
Stop watch
Skin staple application
Tape measure
Snellen chart

Thermometer

Skin calipers

Illness/injury patient care instructions

List of contents

Cardiopulmonary Resuscitation Crash Kit

Equipment

1. Oral airways (small, medium, and large)
2. Mouth-to-mouth mask
3. Ambu bag with face mask (appropriate size) and adapter for endotracheal tubes
4. Endotracheal tubes, cuffed (small, medium, and large)
5. Disposable laryngoscope with light source
6. Cricothyreotomy kit
7. Syringe (50-mL) and large catheter for suction
8. 5- and 10-cc syringes with 22-gauge needles
9. 14-gauge angiocatheter
10. Automated external defibrillator

Drugs

1. Atropine sulfate, 2 mg in injectable prefilled syringe (10 mL)
2. Epinephrine, 1:10,000 in injectable prefilled syringe (10 mL)
3. Lidocaine hydrochloride, 100 mg in injectable prefilled syringe (5 mL)
4. Sodium bicarbonate, 50 mEq in injectable prefilled syringe (50 mL)
5. Lactated Ringer's (500 mL) and 5% dextrose (500 mL) with tubing and catheters
6. Nitroglycerin, 0.4 mg sublingual tablets
7. Morphine sulfate, 15 mg Tubex® with syringe
8. Meperidine, 100 mg in 2-mL ampoule
9. Furosemide, 40 mg IU ampoule
10. Diazepam (Valium®), 10 mg in prefilled syringe
11. 50% dextrose in water in a prefilled syringe
12. Dexamethasone (Decadron®), 24 mg/mL, 5-mL vial
13. Short-acting beta-agonist inhaler

The Sports Medicine Bag

1. Tongue depressors
2. Padded tongue blade
3. Lidocaine 1% plain
4. Metaproterenol (Alupent®) inhaler
5. Ophthalmologic irrigating solution and eye cup
6. Fluorescein dye strips (3) for staining cornea
7. Tetracaine (Pontocaine®), 0.5%, 2-ml ampoule
8. Small eye spud
9. Sodium sulfacetamide (Sulamyd®) eye drops or ointment

10. Sterile suture set (1) with scalpel and #10 and #11 blades
11. Plastic suture material 5-0, 6-0 (2 each) with swaged PRE-2 needles
12. Ster-I-Strip, 1/4-inch and 1/2-inch (2 packages each)
13. Tape adherent, 1 can
14. Sterile gloves (2 pairs)
15. Sterile gauze pads, 2 × 2 inch, 3 × 3 inch, 4 × 4 inch (3 ea)
16. Povidone-iodine (Betadine®) solution
17. ABD pads (2)
18. Medicine cups (2)
19. Cotton-tipped applicators (Q-tips®), 1 package
20. Betadine® ointment
21. 8-inch heavy-duty bandage scissors
22. Swiss army knife with as many gadgets as possible
23. Disposable flashlight (1)
24. Elastic bandages: 3-, 4-, and 6-inch (1 each)
25. Prewrap foam bandage, 3-inch (1)
26. Elastic tape: 1- and 3-inch (2 each)
27. Cotton web roll, 4-inch (2)
28. Tufskin® (Cramer), 1 can
29. Skin lube® (Cramer), 1 tube
30. Plastic foam sheet, 3/4-inch thick
31. Felt padding, 3/8-inch (4 × 4-inch sheet)
32. Cyanoacrylate (Super Glue®) (1)
33. Stethoscope
34. Aneroid manometer
35. Reflex hammer
36. Ophthalmoscope-otoscope
37. Thermometer
38. Orthoplast® sheet (6 × 6 inch)
39. Band-Aids®, all sizes
40. Moleskin® (6 × 6 inch)
41. Rubber suction device for contact lens removal
42. Alcohol swabs
43. Nasostat®/tampons, regular (2)
44. Hemostat or tongue forceps
45. Two-way radio pager or mobile phone
46. Eye patches (2)
47. 3- and 5-cc syringe (1 each) with 22- and 25-gauge needles (2 each)
48. Antibiotics (penicillin, tetracycline, erythromycin, trimethoprim)
49. Topical antifungal

50. NSAID of choice
51. Ear drops
52. Cold medication of choice
53. Insect repellant
54. Sunscreen (SPF 15)
55. Scalpels (#11, 15) 1 each
56. Prescription blanks
57. 30% ferric subsulfate (Monsel's solution) for abrasions and cuts
58. Hank's balanced salt solution for dental avulsions
59. Small mirror
60. List of contents

GENERAL REFERENCE

Ray, R.L. and Feld, F.X., The team physician's medical bag, *Clin. Sports Med.*, 8(1), 139–146, 1989.
AAFP, AAOS, ACSM, AMSSM, AOSSM, and AOASM, *Sideline Preparedness for the Team Physician: A Consensus Statement*, 2000.

APPENDIX 5

INJURY SURVEILLANCE AND PREVENTION ORGANIZATIONS

Aerobics and Fitness Association of America (AFAA)

Contact

Marti Steele West
15250 Ventura Blvd., Suite 310
Sherman Oaks, CA 91403
(818) 905-0040

Joint survey of injuries incurred by 10,000 exercise instructors, including frequency, type, severity, and location of injuries. Safety hot line: (800) BE-FIT-86.

American Aerobics Association

Contact

Jean Rosenbaum, M.D., Director
P.O. Box 401
Durango, CO 81302
(303) 247-4109

Survey of 3000 aerobics instructors and 30,000 participants, on injury severity, anatomic sites of injury, and equipment associated with specific injuries.

American Medical Soccer Association (AMSA)

Contact

Robert M. Cosby, M.D., President
1130 Ford Avenue
Birmingham, AL 35217
(205) 841-2766

Information on soccer-related injuries.

Athletic Health Care System (AHCS)

Contact

Stephen G. Rice, M.D., Ph.D., M.P.H.
Division of Sports Medicine
GB-15, University of Washington
Seattle, WA 98195
(206) 543-1550

Information about athletic injuries from 20 different high schools based on coaches' records on time loss, type of restriction, anatomic region, and type of injury, as well as uninjured athletes.

Big Ten Injury Surveillance Survey

Contact

John Powell, Ph.D., A.T.C., or
John P. Albright, M.D.
1189 Carver Pavilion
University of Iowa Hospitals
Iowa City, IA 52242
(319) 338-0581, Ext. 425

Survey on injuries in selected men's and women's sports from Big Ten Conference schools.

Division of Injury, Epidemiology, and Control

Contact

Stewart Brown, M.D., Director
Division of Injury, Epidemiology, and Control
Center of Environmental Health
Centers for Disease Control
Atlanta, GA 30333
(404) 454-4690

Institute for Aerobics Research (IAR)

Contact

Harold W. Kohn, M.S.P.H.
12330 Preston Rd.
Dallas, TX 75230
(214) 701-8001

Survey of 6900 patients at Cooper Clinic regarding life-time exercise patterns and orthopedic injury rates, plus planned study of relationship to baseline musculoskeletal fitness.

International Dance-Exercise Association, Inc. (IDEA)

Contact

Diana Page Wood
IDEA, The Association for Fitness Professionals
6190 Cornerstone Ct. E, Suite 204
San Diego, CA 92121
(619) 535-8979

Survey of 800 aerobic dance participants regarding injury frequency and type and correlation to income, age, sex, and use of equipment.

Interscholastic Athletic Injury Surveillance System

Contact

Marjorie J. Albohm, A.T.C., Associate Director
International Institute of Sports Science and Medicine
1199 Hadley Rd.
Mooresville, IN 46153

Collects and analyzes data on injuries (categorized by time lost) among high school athletes and adult recreational athletes in Indiana, involving football, girls' and boys' basketball, gymnastics, and volleyball. Also predicts injury trends.

National Academy of Sports Vision

Contact

A.J. Garner, O.D., Executive Director
200 S. Progress Avenue
Harrisburg, PA 17109
(717) 652-8080

Data on sports-related eye problems and injuries.

National Athletic Injury/Illness Reporting System (NAIRS)

Contact

John W. Powell, Ph.D., Research Associate
University of Iowa
Sports Medicine Research Laboratory
1337 Steindler Building
Iowa City, IA 52242
(319) 335-7350

Database of information on sports-related injuries at the high school and professional level reported from more than 100 high schools across the country and every NFL team.

National Collegiate Athletic Association (NCAA) Injury Surveillance System

Contact

Randall W. Dick
Box 1906
Mission, KS 66201
(913) 384-3220

Tracks injuries in football, men's and women's soccer, lacrosse, gymnastics, women's volleyball, field hockey, ice hockey, wrestling, softball, and baseball from 10% of NCAA member schools that participate in a given sport. Includes type of injury, severity of injury, body part injured, cause of injury, field type, field condition, position played, special equipment worn, and time of season.

National Football Head and Neck Injury Registry

Contact

Joseph S. Torg, M.D.
c/o University of Pennsylvania Sports Medicine Center
Weightman Hall E-7
235 S. 33rd St.
Philadelphia, PA 19104
(215) 662-6943

Data on football-related cervical spine and head injuries that cause hospitalization for 72 hours or death or paralysis.

National High School Athletic Injury Registry

Contact

John LeGear
c/o National Athletic Trainers' Association, Inc.
P.O. Box 3548
Oak Park, IL 60303
(312) 386-1610

Retrospective and prospective data on high school football, basketball, wrestling, and soccer injuries from 100 to 150 schools. Includes age, height, weight, experience in the sport, level of education, anatomic site of the injury, damage incurred, mechanism of injury and characteristics of the environment such as the condition of the floor or ground surface.

The National Injury Information Clearinghouse/National Electronic Injury Surveillance System (NEISS)

Contact

Drucilla Besley
U.S. Consumer Product Safety Commission
5401 Westbard Avenue, Room 625
Washington, D.C. 20207
(301) 492-6424

Collects, investigates, analyzes, and disseminates injury data (age, sex, diagnosis, body part injured, type of treatment, and location where accident occurred) and information relating to the causes and prevention of death, injury, and illness associated with consumer products.

National Injury Prevention Foundation

Contact

Peter R. Francis, Ph.D.
WG406
San Diego State University
San Diego, CA 92182
(619) 265-5625

Injury research in conjunction with other organizations.

National Safety Council (NSC)

Contact

Terry Miller or Ursula Zuehlke
National Safety Council
444 N. Michigan Avenue
Chicago, IL 60611
(312) 527-4800

Data on number of participants, number and severity of injuries, and the number of facilities in 30 sports.

National Society to Prevent Blindness

Contact

Tod Turriff, Manager
Eye Safety Programs
500 E. Remington Rd.
Schaumburg, IL 60173
(312) 843-2020

Collects and analyzes data on sports-related eye injuries.

Regional Spinal Cord Injury System

Contact

Samuel L. Stover, M.D.
National Spinal Cord Injury Statistical Center
University of Alabama at Birmingham
University Station
Birmingham, AL 35294
(205) 934-3330

Consists of 13 regional spinal cord injury systems — a network established to create and consolidate a database on spinal cord injuries and disseminate pertinent information.

Sports Safety Board of Quebec

Contact

Guy Regnier, Ph.D., Research Director
100 Laviolette Avenue, Bureau 114
Trois-Rivieres, PQ G9A 589
(819) 373-8433

Water Safety Services

Contact

Ben Harris, Director
Water Safety Services
666 Dundee Rd., Suite 502
Northbrook, IL 60062
(312) 480-9830

Tracks deaths occurring in swimming pools.

APPENDIX 6

SPORTS AND SPORTS MEDICINE ORGANIZATIONS

Academy for Sports Dentistry (ASD)
12200 Preston Road
Dallas, TX 75230

Amateur Athletic Union
3400 West 86th Street
Indianapolis, IN 46268

American Academy of Podiatric Sports Medicine (AAPSM)
1729 Glastonberry Road
Potomac, MD 20854

American Alliance for Health, Physical Education, Recreation, and Dance
1900 Association Drive
Reston, VA 22091

American College of Sports Medicine (ACSM)
P.O. Box 1440
Indianapolis, IN 46206

American Medical Joggers Association (AMJA)
P.O. Box 4704
North Hollywood, CA 91607

American Medical Society for Sports Medicine
7611 Elmwood Avenue, Suite 202
Middleton, WI 53562

American Orthopedic Society for Sports Medicine
70 West Hubbard, Suite 202
Chicago, IL 60610

American Osteopathic Academy of Sports Medicine
4610 University Avenue, Suite 480
Box 55095
Madison, WI 53705

American Physical Therapy Association (APTA)
1111 North Fairfax Street
Alexandria, VA 22314

Association for Intercollegiate Athletics for Woman
1201 16th Street N.W.
Washington, D.C. 20036

Canadian Academy of Sports Medicine (CASM)
c/o Sports Medicine Council of Canada
National Sport and Recreation Centre
333 River Road
Ottawa, Ontario K1L 8H9
Canada

International Federation of Sports Medicine
c/o Allan Ryan, M.D., Secretary General
5800 Jeff Place
Edina, MN 55436

National Academy of Sports Medicine
26632 Agoura Road
Calabasas, CA 91301
800-460-6276; 818-878-9203
Fax: 818-878-9511
www.nasm.org

National Athletic Trainers Association (NATA)
1001 East 4th Street
Greenville, NC 27834

National Collegiate Athletic Association
P.O. Box 1906
Shawnee Mission, KS 66222

National Federation of State High School Athletic Associations
11724 Plaza Circle
P.O. Box 20626
Kansas City, MO 64195

National High School Athletic Coaches Association
3423 East Silver Springs Boulevard, Suite 9
Ocala, FL 32670

National Strength and Conditioning Association
251 Capital Beach Boulevard, Suite 12
P.O. Box 81410
Lincoln, NE 68501

President's Council on Physical Fitness and Sports
400 6th Street S.W., Room 3030
Washington, D.C. 20201

United States Olympic Committee
1750 East Boulder Street
Colorado Springs, CO 80909

APPENDIX 7

THERAPEUTIC EXERCISES FOR THE INJURED ATHLETE

Robert M. Barney Poole

Physical therapy in the rehabilitation of an injured athlete involves a variety of range-of-motion and strengthening exercises to restore the function of each injured body part.

SHOULDER RANGE OF MOTION

Active-assisted or passive-assisted range of motion in flexion for the shoulder, using a T-bar:

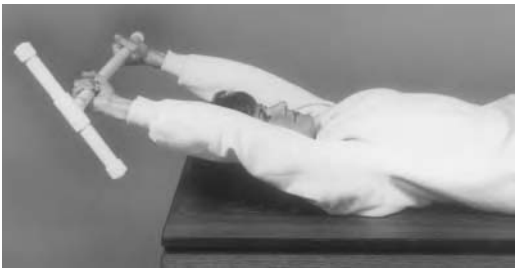

FIGURE 1 *Shoulder Flexion:* The athlete is positioned supine, gripping the stick with both hands, which are a shoulder-width apart. Move the arms over the head as far as possible and hold for 5 counts. Relax. Complete 5 sets of 10 repetitions, 3 times a day.

FIGURE 2 *Shoulder External Rotation:* The athlete is positioned supine with the arm abducted to 90°. Push the arm down toward the floor into external rotation and hold for 5 counts. Relax. Complete 5 sets of 10 repetitions, 3 times a day.

FIGURE 3 *Shoulder Abduction:* The athlete is positioned supine and uses the T-bar to push/pull the arm into abduction. (The arm must remain on the table to get the maximum benefit from this exercise.) Hold for 5 counts. Relax. Repeat 5 sets of 10 repetitions, 3 times a day.

SHOULDER STRENGTHENING

High repetition, low weight, isotonic exercise incorporating eccentric and concentric contractions:

FIGURE 4 *Shoulder Shrugs:* The athlete stands with the arms straight down at the sides and a 1-pound weight in each hand. (Left) Lift the shoulders up toward the ears as far as possible and hold for 5 counts, then (right) pull them back, pinching the shoulder blades together. Again, hold for 5 counts. Relax the shoulders slowly. Repeat 5 sets of 10, gradually increasing the weight to 5 pounds.

FIGURE 5 *Supraspinatus Strengthening:* The athlete stands with the affected arm straight at the side and the thumb joint pointed down. Raise the arm from this point to eye level, holding for 5 counts. Relax. Repeat 5 sets of 10, 3 times a day, gradually progressing from 1 to 5 pounds, as tolerated.

FIGURE 6 *Prone Horizontal Abduction:* The athlete is positioned prone with the involved arm hanging straight toward the floor. Raise the arm to eye level, keeping the thumb pointed up toward the ceiling. Hold for 5 counts. Relax. Repeat 5 sets of 10, progressing from 1 to 5 pounds as tolerated.

FIGURE 7 *Prone External Rotation:* The athlete lies prone with the arm at 90° of abduction, the elbow bent to 90°, and the forearm hanging straight down from the table. With the thumb pointed toward the body, lift the arm into external rotation to eye level. Hold for 5 counts. Relax. Repeat 5 sets of 10, progressing from 1 to 5 pounds as tolerated.

FIGURE 8 A return-to-throwing program gradually progresses the throwing athlete back to competition. A program of short- and long-toss sessions is completed twice per day for two successive days, followed by a day of rest and then two more days of throwing.

ELBOW

Stretching and strengthening using high-repetition, low-weight, isotonic exercise incorporating eccentric and concentric contractions:

FIGURE 9 *Stretching Flexors:* With the elbow straightened, grasp the middle of the hand and thumb and pull the wrist back toward the elbow. Hold for 10 counts. Relax slowly. Repeat 3 sets of 10, 3 times a day.

FIGURE 10 *Stretching Extensors:* With the elbow straightened, grasp the back of the hand and thumb and pull back toward the elbow. Hold for 10 counts. Relax slowly. Repeat 3 sets of 10, 3 times a day.

FIGURE 11 *Wrist Flexion Curls:* The forearm is comfortably supported on a table with the hand over the edge, palm up. Using a 1-pound weight, curl the wrist up as far as possible and hold for 5 counts. Relax slowly. Repeat 5 sets of 10, 3 times a day, gradually progressing from 1 to 5 pounds, as tolerated.

FIGURE 12 *Wrist Extension Curls:* The forearm is supported comfortably on a table with the hand over the edge, palm down. Using a 1-pound weight, curl the wrist up as far as possible and hold for 5 counts. Relax slowly. Repeat 5 sets of 10, 3 times a day, gradually progressing from 1 to 5 pounds, as tolerated.

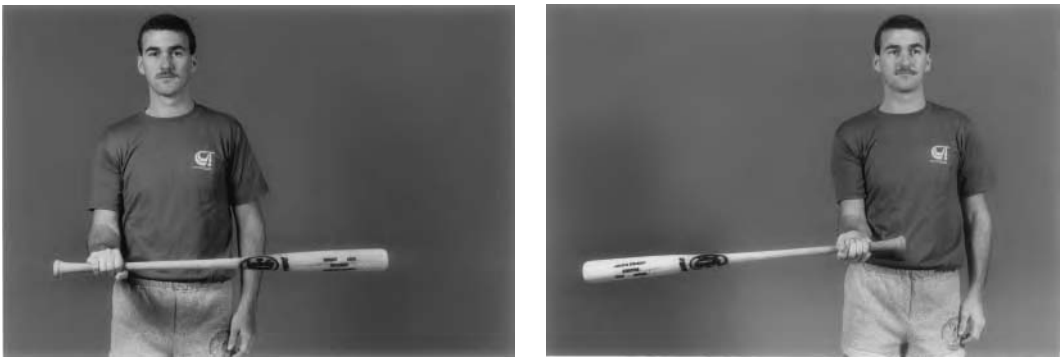

FIGURE 13 *Pronation/Supination Using a Baseball Bat:* With the arm held at the side, bend the elbow to 90°, then grasp the middle of the bat with the arm in the neutral position. (Left) Move the wrist and arm into pronation as far as possible and hold for 5 counts. (Right) Move the arm slowly back to neutral and then into full supination, again holding for 5 counts. Relax briefly in neutral and repeat 3 sets of 10, 3 times a day. The resistance may be increased by moving the hand toward the end of the bat. *Note:* To make exercise more interesting, use objects with which the athlete is familiar.

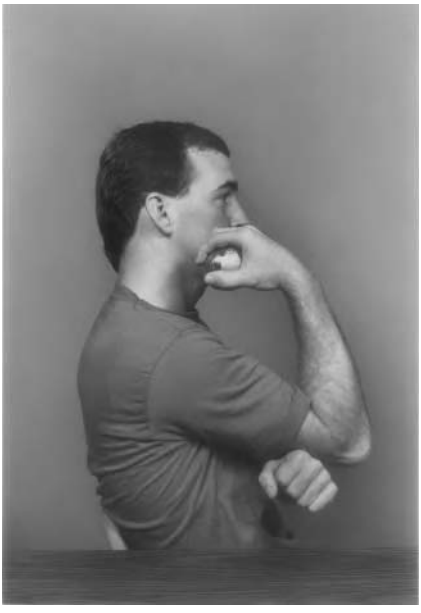

FIGURE 14. *Biceps Curl:* The straightened elbow is supported by the opposite arm, then bent as far as possible and slowly returned to full extension. Begin with a 2-pound weight and work up to 5 sets of 10 repetitions, 5 times a day, progressing to 10 pounds, as tolerated.

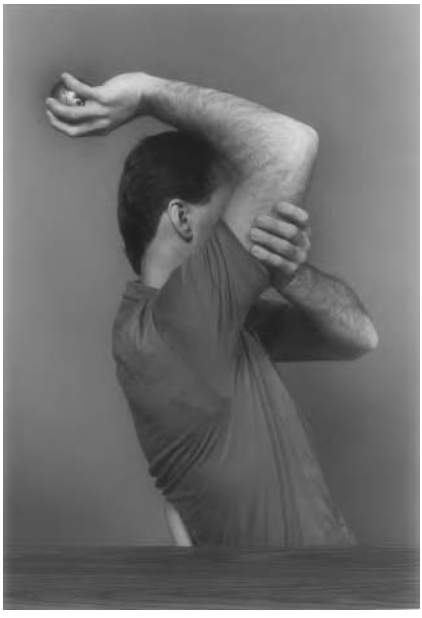

FIGURE 15 *Triceps Curl:* The straightened arm is supported by the opposite hand at the elbow with the arm raised overhead. Bend the elbow as far as possible and then slowly straighten it to full extension. Begin with a 2-pound weight and progress to 5 pounds as tolerated. Repeat 5 sets of 10 repetitions, 3 times a day. *Note:* Therapeutic putty is a good adjunct to an elbow program and is used to maintain strength and function.

KNEE

Strengthening and stretching, by means of isometric and isotonic exercise incorporating high-repetition, low-weight exercises:

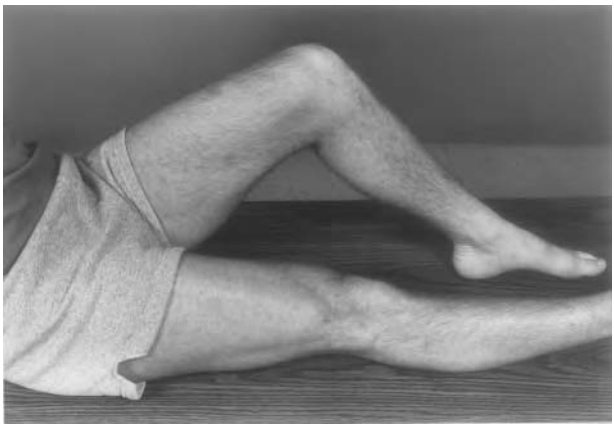

FIGURE 16 *Quadriceps Exercise:* A gradual isometric contraction of the quadriceps is performed by straightening the leg as much as possible. The patella should track proximally. Hold for 5 counts each and perform frequently during the day.

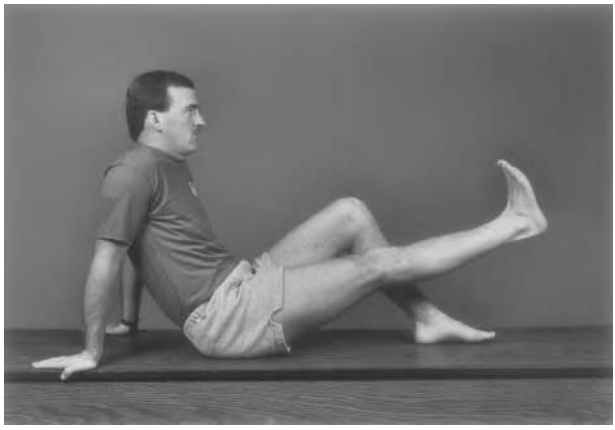

FIGURE 17 *Straight Leg Raise:* The athlete is positioned semi-reclined or supine with the opposite leg flexed to 90° and the foot planted flat next to the involved knee. Contract the quadriceps and lift the left 45°. Hold this position for at least 5 counts, then slowly lower the leg to the floor. Relax and repeat. Lifts are done in sets of 10 with a 30-second rest between each set, 3 times a day. Weight is applied to the ankle for resistance. Gradually progress from 1 to 5 pounds, as tolerated.

FIGURE 18 *Hip Abduction Exercises:* The athlete is positioned side-lying with the unaffected knee flexed to 90° and the hip flexed at 45°. The affected leg is straight, and the body weight is shifted forward. Lift the leg and hold for 5 counts, then slowly lower it back to the starting position. Resistance can be added at the ankle, from 1 to 5 pounds, as tolerated. Repeat 5 sets of 10 repetitions, 3 times a day.

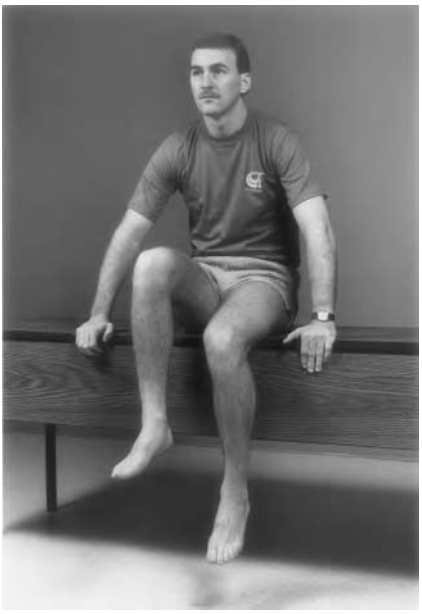

FIGURE 19 *Hip Flexion Exercise:* The athlete is placed in a seated position. Lift the knee toward the chest at a 45° angle and hold for 5 counts. Slowly lower the knee and place the foot on the floor. Repeat 5 sets of 10 repetitions, 3 times a day. Resistance can be added gradually on top of the thigh, from 1 to 5 pounds, as tolerated.

FIGURE 20 *Hamstring Curl:* The athlete stands with the thigh pressed against a wall or table to block hip flexion. Flex the knee as much as possible and hold for 5 counts. Slowly return to the starting position. Repeat 5 sets of 10, 3 times a day. Resistance can be added to the ankle, from 1 to 5 pounds, as tolerated.

FIGURE 21 *Hamstring Stretch:* The athlete is in a sitting position with one leg off the exercise table. The back is straight, and the leg to be stretched is straight. Reach forward slowly and hold for 10 counts. Stretch for at least 5 minutes, 3 times a day. Do not bounce when stretching.

FIGURE 22 *Exercise Bike:* The exercise bicycle is a good way for athletes to increase endurance, strength, and range of motion. The bend in the knee should be 15° when the foot is at the bottom of the pedal stroke. Progress from 10 minutes at minimum resistance to 30 minutes, twice a day.

ANKLE

Stretching and strengthening using high-repetition, low-weight exercises incorporating eccentric and concentric contractions:

FIGURE 23 *Heelcord Stretching Using a Heelcord Box:* The feet should be positioned on the heelcord box for a comfortable, sustained stretch of about 5 minutes, 3 to 5 times daily. The box may be positioned with the higher edge (left) toward or (right) away from the wall. Heelcord stretching is especially important both *before* and *after* sports activities.

FIGURE 24 *Anterior Tibialis Strengthening:* The athlete sits with the foot off the floor. With a 1-pound weight attached to the forefoot, pull the foot up as far as possible and hold for 5 counts. Relax slowly and repeat 5 sets of 10 repetitions, 3 times a day. Progress from 1 to 5 pounds, as tolerated.

FIGURE 25 *Proprioception Exercises:* These may range from simple exercises, such as (top left) balancing while standing on one foot, to more complex exercises. (Top right) The ankle balance board may be weighted to provide strengthening along with proprioception. (Lower left) The new Star Station by Camp Corporation gives a computer-produced color printout of pressure monitored by a pressure-sensitive network beneath the balance board. This is possibly the most complex of all proprioception devices.

FIGURE 26 *Peroneal Strengthening:* The athlete lies on the involved side with the involved foot over the edge of the table. With a 1-pound weight placed on the forefoot, turn the lateral side of the foot up and hold for 5 counts. Relax slowly. Repeat 5 sets of 10 repetitions, progressing from 1 to 5 pounds, as tolerated.

FIGURE 27 *Posterior Tibialis Strengthening:* The athlete lies on the involved side with the involved foot over the edge of the table and a 1-pound weight on the forefoot. Turn the medial side of the foot up and hold for 5 counts. Relax slowly. Repeat 5 sets of 10 repetitions, progressing from 1 to 5 pounds, as tolerated.

FIGURE 28 *Return to Running Program*: The running athlete is gradually progressed from walking to a slow jog to full-speed running and agility drills, as tolerated.

GLOSSARY

GLOSSARY

With the marked growth of the field of sports medicine over the past 20 years, a new vocabulary has emerged; therefore, physicians interested in this subject may be inundated with many new terms with which they have no previous familiarity. It is well known that medical schools are trying to teach students not to use proper names or slang terms, but as the field of sports medicine grows, so does the use of this strange vocabulary. Along with the new terms, sports medicine has also incorporated many of the long-term standards of orthopedics (indicated by an asterisk). The intent of this glossary is to clarify some of the communication problems primary care physicians will face in the care of athletes.

Apprehension shoulder: Feeling of instability in a subluxed or dislocated shoulder than has been reduced.

Athlete's foot:* Tinea pedis (ringworm of the foot); characterized by itching, fissure formation, and inflammation.

Athlete's kidney: Upper calyceal scarring, usually in the right kidney, following multiple traumatic punches.

Athlete's pseudoanemia: Dilutional effect caused by a proportionately greater increase in plasma volume than in red blood cell mass and hemoglobin.

Athletic pseudonephritis: Abnormal urine analysis secondary to exercise-induced renal ischemia.

Backpack palsy: Compression of the upper trunk of the brachial plexus from a heavy backpack.

Baker's cyst:* Popliteal cyst.

Barked shin: Contusive abrasion of the anterior surface of the tibia.

Barton's fracture:* Marginal fracture of distal radial rim due to extreme fixed dorsiflexion of the wrist in association with a pronating force.

Baseball elbow: Worn-out elbow joint seen in veteran pitchers.

Baseball finger: Avulsion of the extensor tendon from its attachment to the dorsal surface of the base of the terminal phalanx; also called mallet finger.

Bends: Arthralgias in scuba diving due to joint air emboli.

Bennett's fracture:* Fracture/dislocation of the carpal metacarpal of the thumb, with fracture of some portion of the medial proximal margin of the base of the metacarpal.

Biker's knee: Occurs when saddle is too high or too low, resulting in excessive patellar pressure, inflammation, and pain.

Black dot heel: Calcaneal petechiae secondary to running.

Black eye: Periorbital hematoma.

Black heel: Calcaneal petechiae secondary to repeated microtrauma (e.g., sudden stops and starts); also called talon noir.

Bladder knock: Microtrauma to bladder wall in long-distance runners, frequently resulting in hematuria.

Blocker's exostosis: See Tackler's exostosis.

Blocker's node: See Tackler's exostosis.

Blowout fracture: Fracture of the orbital floor manifested by limited eye motion and diplopia if the extraocular muscles (i.e., inferior rectus) are entrapped.

Bonking: Seen in endurance athletes when they run out of liver glycogen, presumably due to low blood sugar, causing the patient to get dizzy, shaky, confused, have cold sweats, and experience lack of coordination.

Boot-top fracture: Comminuted fracture at the junction of the middle and lower third of the tibia secondary to high-speed forward fall, angling leg over high, rigid ski boot.

Boutonniére deformity:* See Buttonhole deformity.

Bowler's finger: Perineural fibrosis of the dorsal branch of the radial digital nerve of the ring finger due to repetitive microtrauma.

Bowler's thumb: Digital neuroma of the ulnar digital nerve due to repetitive microtrauma of the thumb base against the hard edge of ball's thumbhole during delivery.

Boxer's elbow: Chip fractures and loose bodies in the elbow joint, produced by repetitive microtrauma in hyperextension (e.g., jabs).

Boxer's fracture:* Fracture of the neck of the fourth or fifth metacarpal.

Boxer's knuckle: Chronically inflamed metacarpal phalangeal bursa due to repetitive trauma.

Breaststroker's knee: Grade 1 medial collateral sprain resulting from the "whip kick."

Buddy system: Alignment of an injured digit to an adjacent digit in order to immobilize it (also, pairing off partners for safety, as in swimming).

Bullrider's thumb: Sprain of the radial collateral ligament and avulsion fracture of the radial portion of proximal phalanx during mechanical bull riding.

Burner: See Stinger.

Button: Chin.

Buttonhole deformity: Disruption of the central slip of the extensor digitorum communis tendon over the PIPJ.

Catcher's hand: Repetitive trauma to the ulnar artery along its course through Guyon's canal, leading to thrombosis and aneurysm.

Cauliflower ear: Ear deformity due to repeated hemorrhage and fibrosis of the perichrondrium and cartilage of the ear. Also called scrum ear and wrestler's ear.

Charley horse:* Contusion of elements of the quadricep muscles.

Chokes: Pulmonary air embolism from rapid diving ascent.

Clarinetist's thumb: Carpometacarpal and metacarpophalangeal joint strain of the dominant thumb in clarinet players.

Clicking hip:* See Snapping hip.

Clipping injury: Valgus force to the knee.

Clothesline injury: Direct blow to the larynx, with pain and difficulty in swallowing and inspiration; acute laryngospasm may occur.

Clutched thumb:* See Trigger finger.

Coaches' finger: Dislocation of the proximal interphalangeal (PIP) joint of the finger, usually middle phalanx dislocated dorsal to the proximal phalanx, generally as a result of hyperextension.

Colles' fracture:* Distal forearm fracture resulting from a fall on the outstretched hand causing dorsal displacement of the distal radius (dinner fork deformity).

Crabs: Pediculosis pubis.

Crazy bone: Contusion of ulnar nerve in the ulnar groove.

Cuber's thumb: Repeated thenar contusions while working Rubik's Cube.

Cyclist's nipple: Cold, painful nipples following exposure to cold during cycling.

Cyclist's palsy: See Handle-bar neuropathy.

Dart thrower's elbow: Olecranon bursitis.

Dead arm syndrome: Sharp paralyzing pain during the cocking phase of throwing due to shoulder subluxation.

Ding: Also known as "getting your bell rung" — a first-degree head concussion with no loss of consciousness, but the athlete may be slightly confused and have dizziness and ringing in the ears.

Drummer boy's palsy: Rupture of the extensor polices longus in drummers from repetitive thumb motion.

Ear squeeze: Pressure differential during diving descent, causing otalgia, decreased hearing, tinnitus, and occasionally blood-tinged sputum due to transudation and blood vessel rupture.

Egyptian foot: Condition in which the first metatarsal ray is the longest.

Exercise addiction: Negative withdrawal symptoms (e.g., feelings of guilt, irritability, anxiety, constipation, sleep disturbance) that occur after stopping long-term, intense, regular exercise program; may be endorphin mediated.

Fartlek: "Speed play" — unstructured program incorporating a combination of techniques such that repetition, interval, and continuous training are used in a single session.

Fighter's fracture: See Boxer's fracture.

Foot drop:* Loss of motor function caused by contusion of the peroneal nerve by a direct blow with significant swelling or hemorrhage.

Football acne: Acne underneath a football tackler's chin strap.

Football finger: See Jersey finger.

Footballer's (English) ankle: Tenderness over the talar bone of the ankle due to talofibial exostoses from repeated explosive push-offs.

Footballer's migraine: Migraine headache secondary to unexpected head blows (e.g., soccer, wrestling) and thought to be due to trauma-induced vasospasm.

Fungo: Three to four weeks rehabilitation for injured pitchers utilizing long, easy throws that progress to short, strong throws.

Gamekeeper's thumb:* Rupture or chronic laxity of the ulnar collateral ligament of the first metacarpal-phalangeal joint (thumb).

Golden period: First 20 to 30 minutes after an injury, when it is easy to identify pathology before swelling sets in.

Golfer's elbow: Medial epicondylitis.

Greek foot: Condition in which the second metatarsal ray is longer than first.

Groin pull: Strain of hip flexors or adductors.

Guitarist's cramp: Spontaneous flexion of the third metacarpophalangeal joint of the right third finger in guitarists.

Gymnast's wrist: Acute or chronic osseous or soft-tissue injury to the wrist in association with compression dorsiflexion forces of gymnastics.

Hammer toe:* Permanent flexion of the midphalangeal joint.

Hamstring pull/tear: Strain of the posterior thigh muscles.

Handle-bar neuropathy: Overuse syndrome associated with cycling, appearing as weakness and loss of coordination in one or both hands due to compression entrapment of the ulnar nerve in Guyon's canal.

Heat cramps: Muscle cramps, twitching, and spasms in the legs, arms, or back, presumably due to electrolyte imbalance.

Heat exhaustion: Weakness, sweating, or dizziness with normal body temperature after prolonged heat exposure; may be associated with dehydration and tachycardia.

Heat fatigue: Inefficient muscle function due to lack of heat acclimatization.

Heatstroke: Extreme hyperthermia with thermoregulatory failure and profound CNS, electrolyte, and metabolic abnormalities.

Heel spur syndrome:* Plantar fasciitis. Inflammation reaction at insertion of the plantar fascia into the calcaneus.

Herpes gladiatorium: Herpes simplex infection; occurs in wrestlers.

Hip pointer: A contusion of the iliac crest.

Hitting the wall: Sudden onset of fatigue and depression usually seen in hot weather among long-distance runners, probably due to depletion of blood glucose and muscle glycogen.

Hooker's elbow: Lateral epicondylitis secondary to "hooking" in ice fishing — repeated jerking on a fishing line attached to a wooden stick.

Hot shot: See Stinger.

Hot spots: Trigger points.

Housemaid's knee:* Prepatellar bursitis.

Hurdler's injury: Avulsion of ischial tuberosity at the attachment of the long end of the biceps femoris and the semitendinosus, due to forcible flexion of hip with knee extended.

Hutchinson's fracture: Push-off fracture of the distal radial styloid.

Impact impotence: Loss of erectile capacity in association with groin paresthesias secondary to long-distance cycling (>100 miles/week).

Jammed finger:* Collateral ligament injury, volar plate injury, articular fracture, or dislocation following a jam injury.

Javelin thrower's elbow: Repetitive valgus stress to medial collateral ligament, causing a sprain or avulsion fracture of the olecranon tip following forceful extension.

Jersey finger: A jam injury; rupture of the flexor digitorum profundus, usually of the second or third finger.

Jock itch: Tinea cruris (ringworm of groin); may also be caused by monilia.

Jogger's foot: See Runner's foot.

Jogger's heel: Nonspecific heel pain possibly due to a heel spur, bursitis, fat pad atrophy, stress fracture, fasciitis, or entrapment of the terminal branches of the posterior tibial nerve.

Jogger's itch: See Judo itch.

Jogger's nipple: Repeated irritation of the nipples from shirt during running.

Jogger's penis: See Penile frostbite.

Joint mice:* Small, opaque, loose bodies, unattached to bone, and interspersed between joint surfaces.

Jones's fracture:* Diaphyseal fatigue fracture of the fifth metatarsal due to repetitive microtrauma.

Judo itch: Intense itching around ankles and wrists, working up extremities to hips and shoulders; appears after judo workouts and sweating.

Jumper's ankle: See Footballer's ankle.

Jumper's knee: Inflammation of the patellar tendon at its attachment to the inferior pole of the patella.

Karate fracture: See Boxer's fracture.

Karate knuckle: See Boxer's knuckle.

Linebacker's arm: See Tackler's exostosis.

Little League shoulder: Injury of the proximal humeral epiphysis.

Little Leaguer's elbow:* Medial, lateral, and posterior elbow pathology secondary to repetitive throwing in youngsters.

Locked knee: See Trick knee.

Maisonneuve fracture:* Fracture of the proximal fibula with sprain of the deltoid and tibiofibular ligaments.

Malicious malalignment syndrome (Patellofemoral pain or stress syndrome): Most common etiology of patellofemoral pain, caused by a broad pelvis, femoral anteversion, genu varum and recurvatum, tibia varum, bilateral medial squinting of the patellae, and compensatory foot pronation.

Mallet finger: See Baseball finger.

Marathon foot: Subungual bleeding due to microtrauma of long-distance running.

March fracture:* Fatigue or stress fracture of the metatarsal shaft (Deutschländer's disease).

Mat burn: See Strawberry.

Miner's elbow: Olecranon bursitis.

Miserable malalignment syndrome: See Malicious malalignment syndrome.

Morton's foot:* Second metatarsal longer than the first, which causes weight and distribution problems and pain.

Morton's neuroma:* Interdigital mechanical neuritis that eventually leads to a fibrous reaction producing a neuroma usually between the third and fourth toes.

Musher's knee:* Iliotibial band irritation casing lateral knee pain while "mushing" dog team, secondary to sharp backward kicking of the leg.

Nun's knee:* Prepatellar bursitis.

Oarsman's wrist: Traumatic tenosynovitis of the wrist radial extensors seen in rowers, canoeists, and weightlifters.

Overreaching: Short-term overtraining.

Overtraining: Chronic imbalance between exercise and recover, resulting in severe and prolonged fatigue.

Overuse syndrome: A wide variety of muscle, tendon, ligament, and bone injury due to repetitive microtrauma.

Pac-Man wrist: See Space Invaders wrist.

Paddle soreness: Ischial bursitis and pruritus ani in novice cyclists.

Patellofemoral pain syndrome: See Malicious or Miserable malalignment syndrome.

Penile frostbite: Superficial penile frostbite seen in joggers training in cold weather (jogger's penis), particularly among those who wear training gear made of polyester.

Pianist's cramp: Spontaneous flexion of the metacarpophalangeal joint of the fourth and/or fifth fingers of the right hand in pianists.

Pitcher's elbow: See Baseball elbow.

Piriformis syndrome: Buttock pain secondary to compression of the sciatic nerve by the piriformis muscle.

Prepatellar bursitis: See Housemaid's knee, Nun's knee, Roofer's knee.

Pudendal neuropathy: Compression of dorsal branch of pudendal nerve between bicycle seat and pubic symphysis.

Pump bump: Inflamed nodule lateral to calcaneal attachment of Achilles tendons.

Punch drunk: Permanent dementia pugilistica — neurologic sequela secondary to multiple blows to the head (e.g., boxing, steeplechase jockeys); slurred monotonous speech, dull facies,

irritability, slowness of mentality, and tremor are characteristic.

Punch fracture: See Boxer's fracture.

Racquet player's pisiform: Subluxation of the pisiform with subsequent chondromalacia of the piso-triquetral joint and possible compression of the ulnar nerve in Guyon's canal; seen in tennis, badminton, and squash.

Raspberry: See Strawberry.

Reverse Colles' fracture: See Smith's fracture.

Reverse ear squeeze: Distension of the tympanic membrane due to overpressurization.

Rider's strain: Strain of adductor longus muscle of the thigh, seen in horseback riders.

Ringman's shoulder: Asymptomatic cortical irregularity of the proximal humerus, seen in all-around gymnasts.

Roofer's knee: Prepatellar bursitis.

Runner's ache: Stitch or catch in the side, thought to be from stretching of the large intestine by a gas pocket.

Runner's bump: Os calcis bone spur from repetitive microtrauma.

Runner's diarrhea: See Runner's trots.

Runner's foot: Tarsal tunnel syndrome or medial plantar nerve entrapment.

Runner's fracture: Stress fracture of the lower end of the fibula or tibia.

Runner's high: Euphoria associated with long-distance running.

Runner's knee: Patellofemoral dysfunction or stress syndrome.

Runner's nipple: See Jogger's nipple.

Runner's toe: See Tennis toe.

Runner's trots: Gastrointestinal cramping and/or diarrhea in long-distance runners.

Saddle soreness: Ischial bursitis and pruritus ani in novice cyclists.

Scrum ear: See Cauliflower ear.

Scrum herpes: See Herpes gladiatorum.

Second wind: Subjective feeling of less fatigue and ventilatory stress after first few minutes of continuous exercise. No definite physiological explanation; may be due to achievement of aerobic threshold.

Shin splints:* Pain of the anteromedial distal 2/3 of the tibial shaft due to muscle–tendon inflammation.

Shoulder pointer: Contusion of the clavicle or acromioclavicular joint.

Skier's douche: See Water skiing douche.

Skier's enema: See Water skiing douche.

Skier's fracture: Comminuted spiral fracture of the tibia in association with a fibular fracture.

Skier's nose: Cold-induced rhinorrhea commonly seen in winter sports (skiing, skating, sledding, etc.)

Skier's thumb: See Gamekeeper's thumb.

Skier's toe: See Tennis toe.

Smith's fracture:* Flexion fracture of distal radius with increased volar angulation (reverse Colles' fracture).

Snapping hip: Medial or lateral audible snap, pop, or click of the hip due to subluxation of the femoral tendon or ligament slipping over a bony prominence; associated pain is due to bursitis, synovitis, or tendinitis.

Snapping neck:* Snapping or popping neck (audible or palpable); may be due to either irregularity at articulation or forced snapping of a tendon over bony prominences.

Snapping shoulder:* Subluxation of the bicipital tendon.

Soccer ankle: Anterior capsule sprain with development of a traction spur on the superior talar neck due to repetitive microtrauma of kicking a heavy soccer ball on a wet pitch with an extremely plantarflexed foot.

Space Invaders wrist: Overuse strain of the wrist secondary to playing video games.

Sports anemia: Mild anemia seen acutely from increased plasma volume during early adaptation to endurance exercise; chronically, from iron deficiency (inadequate dietary intake, gastrointestinal blood loss, and hemolysis).

Squat-jump syndrome: Myoglobinuria and rhabdomyolysis following vigorous squat jumping.

Staleness: See Overtraining.

Stinger: Stretch or impingement of the brachial plexus, cervical plexus, or supraclavicular nerves following improper blocking technique, inexperience, or poor conditioning in football players.

Stitch in side: Upper abdominal pain reported by endurance athletes; cause unknown, but may be due to diaphragm muscle spasm.

Stone bruise: Contusion of the bone (usually foot).

Strawberry: Severe abrasion of skin, usually with weeping, secondary to sliding or rubbing on a floor; also termed raspberry, mat or turf burn.

Student's elbow: Olecranon bursitis.

Surfer's knots: Painless hyperkeratotic skin nodules over the metatarsophalangeal joints and anterior tibial surface.

Swimmer's ear: Acute otitis externa.

Swimmer's knee: See Breaststroker's knee.

Swimmer's shoulder: Impingement of the rotator cuff under the coracoacromial ligament and the acromion (coracoacromial arch).

Tackler's exostosis: Calcified spur on midlateral humerus from repeated tackles/blocks with the arm in football; also called blocker's exostosis, blocker's node, and linebacker's arm.

Talon noir: See Black heel.

Tennis elbow: Lateral epicondylitis.

Tennis leg: Traditionally a tear of the plantaris muscle, actually a strain of gastrocnemius (medial head).

Tennis thumb: Tendinitis with calcification in the flexor pollicis longus, secondary to repeated friction.

Tennis toe: Subungual hematoma produced by pressure, usually of the second toe but also the first and third.

Tennis wrist: Strain/laxity of the radioulnar ligament or fracture of the hamate due to excessive wrist action or direct trauma from racquet butt.

Trick knee:* Medial or lateral cartilage damage causing the knee to pop, click, or lock.

Trigger finger: Stenosing tenosynovitis at metacarpophalangeal joint causing painful snapping, flexion, and extension.

Turf burn: See Strawberry.

Turf toe: Traumatic sprain of first metatarsophalangeal joint capsule secondary to hyperextension.

Unhappy triad:* Simultaneous tears of the anterior cruciate, medial collateral ligament, and medial meniscus, seen in football players (clipping tackle) or skiers.

Urban cowboy rhabdomyolysis: Cramps and tenderness in muscle that may be accompanied by a reddish urine secondary to myoglobinuria; all secondary to strenuous activity (in this particular case, riding a mechanical bull) but also seen with prolonged or excessive exertion (e.g., boxing, karate, and marathons).

Water skiing douche: Water under high pressure may enter body orifice — rectum (skier's enema), vagina (skier's douche), auditory canal, nose, etc. — resulting at times in significant trauma.

Water wart: *Molluscum contagiosum*.

Wind knocked out: Blow to the upper abdomen (solar plexus) causing an inability to catch one's breath.

Wrestler's ear: See Cauliflower ear.

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