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Editors: McMahon, Patrick J.
Title: Current Diagnosis & Treatment in Sports Medicine, 1st Edition

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Dedication
To my father, William J. McMahon, MD
In memory of Kevin L. Armstrong, MD, for his outstanding patient care, easy smile, and laughter
Editors: McMahon, Patrick J.
Title: Current Diagnosis & Treatment in Sports Medicine, 1st Edition
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Preface
Current Diagnosis & Treatment in Sports Medicine is an easy-to-read reference for all clinicians involved in the care of athletesâ the weekend warrior as well as the elite athlete. Physicians, physical therapists, athletic trainers, and nurses will find information on, not only musculoskeletal injuries, but also important medical conditions. Knowledge in all of these areas is valuable in keeping the athlete healthy and performing best.
Each chapter is organized in a straightforward and consistent style to promote quick comprehension. A guide for pre-participation and on-the-field evaluation of the athlete is presented first. Medical and musculoskeletal conditions, which are the topics of the subsequent chapters, are organized by body part from the lower extremity to the upper extremity and spine. Specific information affecting female athletes and the latest data in treating concussions follow. The final chapter presents rehabilitation principles for preventing injury and returning the athlete to play after injury.
Outstanding Features

- An easy-to-read, complete text that can be carried to the office, training room, and field of play
- A straightforward and consistent organizational style throughout the book
- Evidence-based recommendations
- Numerous figures and tables for easy comprehension
- Complete evaluation and management of musculoskeletal injuries of the extremities and spine
- Nonoperative and operative therapies
- Specifics for the female athlete
- The latest data in treating and preventing concussions
- Rehabilitation principles to prevent injury and return the athlete to play after injury

Intended Audience
All clinicians caring for athletes, including physicians, physical therapists, athletic trainers, and nurses will find this text a useful resource. Written for those in training, it is an excellent resource for practicing clinicians as well. Numerous figures and detailed information in both tabular and text forms provide a ready reference for evaluation, selecting diagnostic procedures, and management.
Unlike larger manuals that sit on a shelf and are used only as a reference, this book is compact enough to be carried to the office, training room, and the field of play. In addition, its easy readability makes it possible to read in its entirety.
Acknowledgments
I would like to acknowledge all those, including patients, who have taught me that while medicine is a science, its practice is an art. I am also grateful to the staff at McGraw-Hill, especially Harriet Lebowitz and

Hilarie Surrena, for their professionalism and dedication. Most importantly, I thank my family for educating and encouraging me and God, who is my strength.
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Medical Aspects of Sports Medicine
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Medical Benefits of Exercise & Participation in Sports
A continuously growing body of evidence indicates that regular physical activity is associated with dramatic reductions in cardiac events and all-cause mortality. Despite this, there are an estimated 200,000 deaths annually in the United States related to sedentary life-style. In addition to the well-known cardiovascular benefits, improvements in social, mental, and other aspects of physical wellness with sports participation and exercise have been documented. Some of these benefits are listed in Table 1-1.
As more and more research reveals the tremendous benefits of exercise, physicians are expected to encourage and even prescribe physical activity to their patients. With this, they must be aware not only of the potential injuries associated with sports and exercise involvement, but also of the potential medical issues pertinent to each patient and specific to particular sports. It is important to note that all medical issues are of potential concern in an active population. Therefore, the goals of this chapter are to provide an introduction to some medical issues that (1) can be associated with significant morbidity and/or mortality in an active population (eg, arrhythmias related to acute chest trauma or congenital cardiac disease), (2) are very common in general (eg, diabetes mellitus), or (3) may be unique to physically active individuals (eg, exercise-associated asthma).
Preparticipation Evaluation
In the 2002â 2003 school year in the United States there were 6.9 million high school athletes and over 375,000 National Collegiate Athletic Association (NCAA) athletes participating in school-sponsored sports programs, and these numbers are steadily increasing. The American Heart Association, the American Medical Society for Sports Medicine, the American Academy of Family Practice, the American Orthopedic Society for Sports Medicine, and other health and sports organizations have made recommendations regarding the usefulness of a preparticipation evaluation (PPE). Despite the large number of athletes, the above recommendations, and decades of history, the structure, the appropriate content, and even the overall utility of the PPE are still under debate.
The goals of the PPE come from many perspectives (athlete, school, preventive health care, safety, legal issues, etc). These goals include (1) screening for life-threatening illness or injury that would preclude participation in sports; (2) identifying medical or musculoskeletal conditions that could predispose the athlete to further problems or could limit the athlete's performance; (3) collecting baseline data such as medical history, allergies, and vital signs, and in some cases neuropsychological testing, body composition measurements, and other components that can be referred to if the need arises; (4) providing what is often the only exposure of young healthy individuals to the healthcare system with education on issues such as smoking, sexually transmitted diseases (STDs), and supplement use; and (5) meeting organizational or state/school requirements for legal and/or insurance reasons.
Although the exact structure of the evaluation varies between organizations, all are based on a good history and physical examination (Figure 1-1). It has been shown that the history is the most important component of the PPE, often providing clues to issues that require further investigation. The history should include not only the basics (past medical history, family history, medications, allergies), but also a detailed review of systems and a questionnaire regarding symptoms that could raise concern for a particular problem, for example, increased risk of concussion, disordered eating, or exercise-induced asthma. A positive screening question should then prompt a more detailed, targeted history and examination in that area of concern. Although few conditions preclude sports participation completely, the examiner must be aware of these. The conditions of most concern are those that increase the risk of sudden cardiac death. Although the cardiovascular benefits of physical activity and participation in sports are well

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known, participation carries a small but real risk of a serious cardiovascular event. The yearly incidence of sudden cardiac death in young athletes (<35 years of age) is quite small (approximately 1/100,000) but is nevertheless devastating. In this group, the majority of deaths are not caused by coronary artery disease as in an older population, but rather by a group of congenital and acquired conditions. Many of the cardiac and noncardiac conditions that should preclude participation in high-intensity sports are listed in Table 1-2.
Figure 1-1. Sample preparticipation history and physical examination. (Source:
Leawood KS. American Academy of Family Physicians, American Academy of Pediatrics, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, American Osteopathic Academy of Sports Medicine, 1992, 1996.
)

Table 1-1. Potential benefits of regular physical activity.

Decreased all-cause mortality
Decreased risk of coronary disease, cardiac events, and death
Improved control of blood pressure
Slowed progression of early carotid atherosclerosis and reduction in risk of stroke
Improved lipid profile and control of obesity
Improved glycemic control and prevention of type II diabetes mellitus
Improved overall function in patients with certain chronic illnesses (cardiopulmonary, rheumatologic, cancer, etc)
Improved bone mineral density and decreased long-term risk of osteoporosis and fractures
Improved immunity
Modest protection against breast and other cancers
Decreased disability, improved cognitive function, and increased autonomy in elders
Decreased â riskyâ behavior (in adolescent females) including drug use, smoking, and unwanted pregnancy
Improved self-image, self-esteem, and overall mental health
Decreased health-related costs

The physical examination portion of the PPE should then be performed by a clinician trained and experienced in a general medical, cardiovascular, and musculoskeletal examination. This is an opportunity to expand on issues raised by the history and to identify new potential problems. The cardiovascular examination should, at minimum, include auscultation in supine and standing positions. Occasionally, an irregular murmur or other abnormality can be identified. Unfortunately, even a thorough history and a cardiac examination are limited in terms of sensitivity for detecting risk of sudden cardiac death. Despite this, an echocardiogram and/or stress testing are not routinely recommended unless the history or physical examination dictates. Although there is still much debate among â expert panelsâ and health organizations, electrocardiogram (EKG) testing is not uniformly recommended as part of the PPE. Currently, the American Heart Association and the American College of Cardiology do not recommend a routine EKG for athletes less than 35 years of age. The International Olympic Committee Medical Commission recommends EKGs every other year, and the recent 36th Bethesda Conference report states the following: the EKG â may be of use in the diagnosis of cardiovascular disease in young athletes and it has been promoted as a practical and cost-effective strategic alternative to routine echo.â

If there is no previous history of injury, a general musculoskeletal screening examination is usually adequate. In the case of previous injury, an expanded examination of the affected area can identify risk factors for further injury or need for further rehabilitation. A targeted examination may also be performed on sport- or position-specific areas (eg, the dominant shoulder of a baseball pitcher) to increase sensitivity. In the majority

of cases the musculoskeletal examination does not result in disqualification but clearly adds to the goals of safe participation and optimization of performance.

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The Athlete â Downâ

Acute Medical Issues & Injuries

Sideline physicians must have a â disaster planâ in place for the possibility of a catastrophic injury or event. Knowledge of the potential hazards of the competition can be very helpful. Ideally, the sideline physician is familiar with the players and their individual risks, the athletic trainers and coaches, as well as any emergency medical service (EMS) personnel covering the event. A disaster plan should include who to call, which hospital to use, and who would be in charge. Disaster protocol begins with an immediate on-field evaluation to determine the extent of injury and the urgency of the event. In the case of a persistently unconscious or otherwise unstable athlete, initiation of basic life support should begin and EMS should be activated early.

Table 1-2. Contraindications to participation in sports.

Symptomatic hypertrophic cardiomyopathy
Modest to severe aortic stenosis (and other significant valvular disease)
Modest to severe coarctation of the aorta
Symptomatic mitral valve prolapse
Long-QT syndrome, Wolffâ Parkinsonâ White syndrome
Ventricular dysrhythmias
Symptomatic atrioventricular block
Infective carditis
Uncontrolled hypertension
Marfan's disease (with cardiac and valvular involvement)
Sickle cell disease
Uncontrolled asthma
Active tuberculosis
Pulmonary insufficiency with exercise-induced deoxygenation
Recurrent pneumothorax
Uncontrolled seizure disorder
Continued symptoms and/or cognitive deficits postconcussion

Adapted from the 36th Bethesda Conference, 2005, American Heart Association and American Academy of Pediatrics guidelines.

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Collapsed or Unconscious Athlete

Loss of consciousness in the athlete is most commonly the result of trauma, but heat illness, neurologic conditions, metabolic disorders, and hypoxia can also cause a severe change in mental status. Initial evaluation begins with the â ABCsâ familiar to all clinicians: Airway, Breathing, and Circulation. The possibility of a cervical spine injury should be assumed and appropriate precautions must be taken in every case. In a helmeted and padded athlete (eg, football player), the helmet should be left in place to avoid neck hyperextension. All medical personnel should know the on-field location of screwdrivers or clippers used to remove the facemask.

Injuries that pose an immediate threat to life require emergent treatment followed by transport to a hospital. These include respiratory and cardiac arrest. Other injuries that require urgent care include seizures, severe head, neck, and back injuries, uncontrolled hemorrhage, facial injuries, burns, heat stroke, hypothermia, near drowning, and severe musculoskeletal trauma. Recognition and initial treatment for some of these conditions are discussed below. Also discussed are some injuries that although not particularly â life threatening,â require relatively urgent treatment for optimal outcome (eg, dental injury).

Acute Pulmonary Issues

A. Respiratory Arrest

Airway obstruction can occur with aspiration of a foreign body (tooth, mouthpiece), direct neck trauma and deformation (eg, laryngeal fracture), or, more commonly, secondary to swelling and edema; or it may simply be due to relaxed oropharyngeal muscles in the supine, unconscious athlete. Maintenance of the airway is the primary concern for the physician and must be addressed immediately, preferably with a jaw thrust maneuver to avoid exacerbation of possible associated cervical spine injury. If this maneuver is unsuccessful, an emergency tracheostomy is indicated with emergent transfer to the nearest hospital. Respiratory arrest can also be the result of an acute asthma attack or anaphylaxis. Athletes with asthma and known severe allergies should be identified during the preparticipation physical. Treatment of both includes inhaled albuterol, 0.3â 0.5 mL of 1:1000 epinephrine injected subcutaneously (Epi-pen), support with 100% oxygen (and intubation depending on the patient's condition), and immediate transport. Intravenous fluids should be initiated in the person with anaphylaxis because of the risk of cardiovascular collapse. Acute pulmonary edema in an athlete at high altitude can rapidly progress to respiratory arrest and is discussed later in this chapter.

B. Thoracic Trauma

Pulmonary contusions occur from compression of the air-filled lung, producing increasing pressure and tearing of the parenchyma. Often examination is unrevealing (although occasionally rales will be present), and because of this, a minor injury can go undetected. An athlete who presents with hemoptysis and pain, however, must be evaluated further and closely monitored, as rapid progression to acute respiratory distress syndrome (ARDS) and respiratory collapse may occur. Chest radiographs may reveal consolidation or nodular densities, but can take hours to develop and often underestimate the severity of the lesion. Computed tomography (CT) of the chest is more sensitive and should be used for diagnosis.

Pneumothorax may occur spontaneously or secondary to chest trauma. The athlete typically presents with unilateral chest pain, tachypnea, and dyspnea. Physical examination reveals hyperresonance and diminished or absent breath sounds on the affected side. Often symptoms can initially be mild and physical examination not be obvious. Because of this, all athletes with chest trauma must be monitored closely for increasing problems. Tension pneumothorax, presenting with cyanosis and tracheal

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deviation, is a potential complication and may result in vascular compromise and hypotension. If this occurs, insertion of a large-bore needle into the second intercostal space of the anterior thorax may be life saving.

Acute Cardiovascular Issues

A. Cardiac Injury

Myocardial contusion is a serious complication of severe blunt trauma to the chest, presenting most often with nonspecific chest pain and sinus tachycardia. Other arrhythmias, an S3 gallop, a pericardial friction rub, and pulmonary rales may be present. An echocardiogram is the diagnostic test of choice to evaluate for wall motion abnormality and pericardial effusion. An EKG may reveal conduction abnormalities, ST changes, and/or T wave inversions. Serial enzymes (CK-MB and troponin I) can also help with the diagnosis. Most athletes with myocardial contusion have complete recovery, but occasionally, ventricular dysfunction, thrombus, or other complications can result. A normal EKG and enzymes have a reliable negative predictive value for further complications. The athlete should not return to activity for several months until the echocardiogram has normalized; chest protection is then recommended.

B. Sudden Cardiac Death

Commotio cordis is a very rare, but dramatic event defined as sudden collapse and cardiac arrest following blunt chest trauma. Up to two-thirds of the cases are seen in baseball and are the result of precordial ball impact. Adolescents and children are believed to be at higher risk because of increased chest wall compliance. Prevention is aimed at improved protection, particularly in high-risk positions such as a baseball catcher and a hockey goalie. The etiology of sudden cardiac death in this setting is likely ventricular fibrillation or acute bradycardia; thus, although no intervention to date has shown benefit, prompt recognition, cardiopulmonary resuscitation (CPR), and electroshock with an on-field automatic cardiac defibrillator could prove valuable. Causes of sudden cardiac death are listed in Table 1-3.

Sudden death in the athlete is in general a very rare yet devastating occurrence. Cardiovascular causes predominate (85% of 158 athlete deaths in the United States from 1985 to 1995). The most common cause is

hypertrophic cardiomyopathy (HCM), which accounts for approximately 36% of the total sudden cardiac deaths in athletes. HCM is an inherited disease of the sarcomere that causes a hypertrophied, nondilated left ventricle. The clinical course is highly variable, with some patients remaining asymptomatic throughout their lives and others developing severe symptoms of heart failure or premature death. The clinical presentation may include dyspnea, angina, arrhythmia, or syncope, but sudden death during vigorous exercise, without antecedent symptoms, is often seen in children and young adults. Routine cardiac screening is unreliable in detecting HCM and risk of sudden cardiac death. On physical examination, a harsh, mid-systolic crescendoâ decrescendo murmur that increases with decreased preload (eg, valsalva) may indicate HCM, but this is not present in many cases. An EKG may indicate left ventricular hypertrophy (LVH), left atrial enlargement (LAE), and/or conduction abnormalities, but is often normal. Diagnosis is made by echocardiogram, usually revealing asymmetric LVH >15 mm. It is important to note that persons under 15 years of age with HCM may not yet manifest significant hypertrophy and therefore the diagnosis can be missed. Also, echocardiogram alone does not reliably predict risk of sudden death. Eligibility for participation in sports may be judged on a case-by-case basis keeping in mind the risk factors listed in Table 1-4. In most athletes with HCM, competitive sports should be prohibited.

Table 1-3. Causes of sudden cardiac death in athletes.

Cardiomyopathies: hypertrophic cardiomyopathy, dilated cardiomyopathy, myocarditis, arrhythmogenic right ventricular dysplasia
Congenital malformation of coronary arteries
Coronary artery disease
Aortic rupture: Marfan's disease, coarctation of the aorta
Valvular heart disease: aortic stenosis, mitral valve prolapse
Arrhythmias: Wolffâ Parkinsonâ White syndrome, long-QT syndrome, idiopathic ventricular tachycardia
Commotio cordis
Drugs and â supplementsâ : anabolic steroids, amphetamines, cocaine, Ma Huang, Ephedra

Congenital coronary artery anomalies are the second most common cause of sudden cardiac death in young athletes (<30 years). Athletes with congenital coronary artery anomalies are often asymptomatic, but may experience syncope or chest discomfort. Evaluation for exercise-induced myocardial ischemia is indicated for athletes suspected of having such an anomaly. If ischemia is found, athletic participation must be restricted and surgery should be considered. Return to competition can be considered in athletes who have had successful surgical repair and a documented absence of exercise-induced ischemia.

Myocardial ischemia secondary to atherosclerotic coronary artery disease is the most common cause of

exercise-related sudden death in persons over 30 years of age. Most of these athletes have abnormal risk profiles (hypercholesterolemia, diabetes mellitus, a family history of cardiac disease, tobacco use) and often have prodromal chest pain. In any collapsed â mastersâ athlete, myocardial infarct should be suspected. Symptoms are similar to those presenting in nonathletes and include chest pain or tightness, diaphoresis, nausea, dyspnea, and a feeling of impending doom. Acute management includes O_2, Aspirin, nitroglycerin, Emergency Medical Services (EMS) activation, and transport to decrease morbidity and mortality. Immediate EKG monitoring and treatment of arrhythmia in the field with CPR and an automatic cardiac defibrillator can be life saving. Clinicians should be prepared for this scenario when possible. It is important to note that absence of symptoms in highly fit individuals does not guarantee that they are coronary artery disease free. Because of this, testing is recommended in patients with risk factors. Participation in high-intensity competitive sports is not recommended for athletes with documented ischemic disease, regardless of whether the patient has symptoms, has a history of myocardial infarction, or has undergone complete revascularization. Lower intensity activities may be permitted, but each athlete requires individual evaluation and assessment of risk.

Table 1-4. Risk factors for sudden death in patients with hypertrophic cardiomyopathy.

Ventricular tachycardia
Family history of sudden cardiac death due to hypertrophic cardiomyopathy
Syncope
Severe hemodynamic abnormalities (dynamic left ventricular outflow tract gradient >50 mm Hg, exercise-induced hypotension, moderate-to-severe mitral regurgitation)

Enlarged left atrium (>50 mm)
Paroxysmal atrial fibrillation
Abnormal myocardial perfusion
Seizure
Seizure in the athlete is often the result of a closed head injury (ie, concussion), but can be primary or related to other illness. Secondary causes include heat illness, dehydration, and hyponatremia. Metabolic disorders, structural disease, and previous trauma with development of a subacute bleed should also be considered. Airway and cervical spine management is of utmost importance. Transport to stabilize the patient-athlete and to ensure that no significant brain injury has occurred should follow. There will be further discussion on seizure in the athlete later in this chapter.
Head & Neck Injuries
Concussion, discussed in Chapter 8, is a common cause of change in mental status in the athlete. The most serious and most common complications include intracranial hemorrhage and associated spinal injury. Any concern for acute bleed as suggested by persistent altered mental status, focal neurologic findings, or severe headache and other signs of increased intracranial pressure should prompt immediate transport for further evaluation and imaging. A CT scan is a more rapid test in the potentially unstable patient, but magnetic resonance imaging (MRI) has been shown to have better sensitivity and specificity and should be considered after negative CT in the athlete who continues to have findings that are of concern. MRI is also appropriate when an athlete has subacute complaints or physical findings. Once severe associated injury is ruled out, the traumatic brain injury itself must be monitored closely.
Spinal injury should always be assumed in the â down athlete.â The annual incidence of traumatic spinal cord injury is estimated to be between 30 and 45 cases/1,000,000. The majority result from motor vehicle accidents, but 5â 14% occur during sports and recreational activities. Unsupervised diving accounts for 75% of these injuries, but in the United States the risk is highest in supervised sports, such as football, gymnastics, and hockey, in descending order. Once the adequate ABCs have been established, and the cervical spine is immobilized in neutral (eg, with the helmet and pads in place in football players), a thorough history and a neck and neurologic examination are necessary. Radiologic imaging (anteroposterior, lateral, and odontoid views or CT scan) is necessary when the history and physical examination are either inconclusive or of concern. Indications are listed in Table 1-5.

Table 1-5. Indications for radiologic evaluation in the athlete with possible neck injury.

High-risk mechanism of injury
Multiple trauma and/or distracting injures that do not allow for appropriate evaluation of the spine
Altered mental status and/or poor cooperation with the examination
Pain on the top of the head
Neck pain, tenderness, or deformity
Limitation of neck movement
Acute neurologic deficit
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After acute musculoskeletal neck injury, contraindications to return to play include permanent dysfunction, permanent and significant peripheral nerve root dysfunction, and spinal fusion above the C5 level. In addition, stability, as assessed with dynamic radiographs, helps to dictate whether return to play is appropriate in cases of fracture and ligamentous injury. It is generally believed that athletes with cervical burners (transient brachial plexus injury) may return to play when completely asymptomatic. Return to play for athletes who have cervical cord neuropraxia (transient quadriplegia) remains controversial.
Facial Injuries
A. Orbital Trauma
The most common sports-related eye injuries are â black eyeâ (edema/ecchymosis), corneal abrasions, foreign bodies, and lacerations in and around the eyelid. More severe injuries that require urgent specialized evaluation and treatment include lacerations to the globe, commotio retinae (edema of the retina) and retinal hemorrhage, hyphema, and orbital blow-out fractures. Danger signs are listed in Table 1-6. Sports with a very high risk for eye injury include boxing, wrestling, and full-contact martial arts, but hockey, basketball, baseball, softball, racquet sports, and others also carry a relatively high risk. The functionally one-eyed athlete should not participate in very high-risk sports, but may participate in most other sports with the appropriate

(3-mm thick polycarbonate lenses), well-fitted, protective eyewear.

B. Dental Trauma

Dental injuries such as tooth avulsions, fractures, and impactions are not uncommon from sports-related trauma. Tooth avulsion is considered a dental emergency as time is of the essence to preserve function. The contaminated tooth should be gently rinsed and reinserted (assuming the athlete is conscious), with immediate referral to a dentist for splinting and antibiotic prophylaxis. Chances of retaining the tooth after avulsion diminish rapidly with delays in reinserting the tooth. If immediate on-scene reimplantation is not possible, the tooth should be transported in the patient's buccal sulcus, in milk, or in a specialized tooth solution. Fractures limited to the enamel may not require immediate treatment, but dental follow-up is necessary and complete diagnosis for a dental injury should include radiographs at some point. In many cases of facial injury, the airway can be rapidly compromised and needs to be constantly reassessed. Additionally, concussion frequently accompanies significant facial and dental injury and should be considered in all such cases.

Table 1-6. Signs and symptoms of potential serious eye injury.

Acutely decreased vision or loss of field of vision (complete or partial)
Pain with eye movement
Photophobia
Diplopia
â Lightning flashesâ
Halos around lights
Eye protrusion or â sunkenâ eye
Irregularly shaped pupil
Blood in the anterior chamber or a â red eyeâ

Acute Gastrointestinal & Genitourinary Problems

A. Abdominal & Pelvic Trauma

Abdominal and pelvic injuries, although not extremely common in sports participation, can be serious, can cause severe blood loss, and can lead to hypovolemic shock. The liver and spleen are the most commonly injured organs, followed by the pancreas, bowel, kidney, bladder, and blood vessels. Signs of significant injury include abdominal tenderness, rigidity and rebound, hematuria, and hypotension. When any of these signs are present, urgent transport for further imaging and patient-athlete stabilization and treatment are warranted.

B. Hematuria

Major renal trauma will often cause acute pain, but may present with delayed bleeding. Evaluation by ultrasound, CT scan, and/or intravenous pyelogram (IVP) is necessary as this often requires surgical intervention. Minor renal trauma usually presents with hematuria alone. If bleeding is mild, history, physical examination, and urinalysis are usually adequate, with return to play after 2â 3 weeks of relative rest. Renal calculi cause painful hematuria and occur in 12% of men and 5% of women, making kidney stones a relatively common issue in both athletes and the general population. Dehydration can increase the risk of calculi, but in general, athletes are not considered to be at increased risk overall.

A common cause of painless hematuria in athletes who run is believed to be secondary to mild bladder wall trauma. The incidence of this â runners hematuriaâ is between 17% and 69%, with the highest incidence in ultramarathon runners. Hematuria may also arise from the perineal trauma experienced in bicycle, motocross, and recreational cyclists. Athletic pseudonephritis is a combination of hematuria, proteinuria, and casts secondary to nephron ischemia and hypoxia. It can be seen

in high-intensity runners and swimmers. These sports-related hematurias tend to clear after 48 hours. In cases of persistent bleeding, medical conditions such as carcinoma, von Willebrand's disease, and sickle cell disease should be considered.

C. Rhabdomyolysis

Rhabdomyolysis is a condition of significant muscle breakdown leading to renal impairment. The typical cause is heat illness and dehydration, but underlying metabolic issues and supplement or alcohol use can be contributing factors. The athlete may present with â on field collapseâ from severe muscle pain or the causative heat illness, but usually the presentation is subacute and will need to be evaluated with blood work that includes creatine phosphokinase (CPK), blood urea nitrogen (BUN), and creatinine as well as a urinalysis

that includes myoglobin.

D. Testicular Torsion

Testicular torsion is a medical urgency that should not be missed. It presents with unilateral pain and swelling that is exacerbated by lifting the testes above the pubic symphasis. (This is in contrast to epididymitis, in which pain is relieved by this maneuver.) If diagnosis by examination is uncertain, an ultrasound or testicular scan (90% accurate) should be performed. Derotation can be attempted by turning the testes anteriorly and away from midline. If unsuccessful, surgical treatment within 4 hours provides better outcomes.

Musculoskeletal Injuries

â Collapseâ of an athlete on the field is often the result of a musculoskeletal injury. The most common injuries and their evaluation and management are covered in other chapters in this book. Musculoskeletal injuries that can pose an immediate risk to the athlete are discussed here.

A. Open Fractures

Open fractures should be splinted in the position found after a sterile dressing has been placed. Urgent transport is necessary for definitive treatment.

B. Dislocations

Hip or knee dislocations can cause significant vascular injury, as can posterior sternoclavicular dislocations. Because of this, athletes with these injuries should be urgently transported to a hospital emergency department that has the ability to evaluate such problems. In the case of a joint dislocation with neurovascular compromise, a person with proper training should attempt reduction. Neurovascular status must be checked and documented before and after successful (or attempted) reduction. All reductions should have follow-up radiographs to rule out associated fracture in addition to further evaluation (eg, vascular) as necessary.

Environmental Issues

A. Heat Illness

Exertional heat syndromes form a continuum: heat stress â heat cramps â heat exhaustion â heat stroke â death (Table 1-7). Heat dissipation (ie, removal of heat) occurs by four methods: radiation, conduction, convection, and evaporation. If the environmental temperature is greater than 35Â°C (95Â°F), all heat loss must be through evaporation. Humidity of greater than 75% slows evaporation dramatically and sweating becomes inefficient. The body loses no heat when a temperature of greater than 35Â°C is combined with a humidity greater than 90%. Thermoregulation is under the control of the autonomic nervous system via the anterior hypothalamus. Thermoregulation failure, which can occur when there is no heat dissipation, can eventually lead to multiple organ system collapse and death. Factors that increase the risk of heat illness include vigorous physical activity, impermeable or wet clothing, poor muscle conditioning, lack of acclimation, obesity, extremes of age, diuretic beverages and supplements, or medications that affect the autonomic nervous system (eg, stimulants, anticholinergics, and Î±-adrenergics such as decongestants). It is important to note that heat illness and dehydration go hand in hand. Exertional heat syndromes can be prevented with adequate hydration in addition to education regarding dangerous environmental conditions (wet bulb globe temperature greater than 19Â°C), use of proper clothing and equipment, acclimation, and gradual physical conditioning.

B. Cold Injuries

Cold-related injuries are most often associated with winter sports such as skiing, skating, and mountaineering, but can also be seen in other sports such as running, cycling, and swimming. Body heat is produced by four mechanisms: basal heat production is via normal metabolic processes, muscular thermoregulatory heat is produced by shivering and increases body heat three to five times basal level, increased muscular activity during mild to moderate exercise produces five times basal heat production, and high-intensity exercise can produce up to 10 times basal heat but can be sustained for only several minutes. Mechanisms of heat loss have been mentioned previously. To avoid illness and injury, the core temperature must be maintained within a narrow range. Heat conservation occurs by external sources, body insulation, and shunting of blood away from the body's surface area to the core (via peripheral vasoconstriction).

Medical problems that can be stimulated by cold exposure include cold-induced asthma or bronchoconstriction, cold urticaria, and Raynaud's phenomenon. Local cold injury ranges from mild frostnip to the much more severe injury, frostbite. Frostnip is reversible ice crystal

formation on the skin's surface that is treated with gradual rewarming. Frostbite is caused by actual freezing of

the skin and is classified as first to fourth degree based on the depth of soft tissue involvement. Factors that increase risk of frostbite include constricting clothing, smoking, atherosclerosis, diabetes mellitus, immobilization, and use of vasoconstrictive drugs. In severe injury, bullae or dry black eschar will form in previously waxy yellow or mottled blue areas, signifying that the tissue will eventually be lost. At extremes, P.12

mummification and autoamputation can occur. Treatment is aimed at prevention of further tissue damage. Rapid rewarming in a hot bath at 40Â°C is appropriate treatment, but only if there is a mechanism to maintain warmth, as thawing and refreezing result in increased injury. Dry heat and rubbing the affected areas are contraindications. Most cases require hospitalization and analgesic support.

Table 1-7. Continuum of exertional heat syndromes.

	Predominant Pathophysiology	Core Temperature	Symptoms	Treatment
Heat stress	Increased temperature	Normal	Increased blood pressure and heart rate, dizziness, fatigue	Mild cooling, oral hydration
Heat cramps	Total body Na+ deficiency (predominant theory)	Normal	Increased heart rate, muscle cramps/spasm, weakness, fatigue, nausea/vomiting	Mild cooling, oral hydration with electrolyte solution (IV if vomiting), gentle stretching, ice
Heat exhaustion	Hypovolemia Dehydration Electrolyte loss	Normal to 40Â°C (104Â°F)	Orthostasis, syncope, dyspnea, weakness, profuse sweating, flushing and piloerection, headache, and irritability No significant central nervous system dysfunction	Moderate cooling, (move to cool environment, remove excess clothing, water and fans), oral versus IV hydration (depends on ability to take water PO)
Heat stroke	Hyperthermia Thermoregulatory failure	>40Â°C Poor prognosis with temperature >42Â°C	Change in mental status, +/- seizure and coma Hypotension, vomiting, diarrhea, sweating â hot, dry skin Can rapidly progress to rhabdomyolysis, neurologic injury, kidney and liver failure, diffuse intravascular dissemination, acute respiratory distress syndrome, death	Rapid cooling to core temperature 39Â°C (with the above methods + ice packs/bath), IV fluid challenge (monitor for pulmonary/cerebral edema), respiratory assistance and O_2, urgent transport

Hypothermia is a potentially life-threatening systemic injury that occurs when the body core temperature decreases to less than 35Â°C (95Â°F) and is classified as mild, moderate, and severe. Symptoms progress from shivering, chills, and increased respiratory rate, to increased fatigue, loss of shivering, and peripheral numbness, and eventually to changing levels of consciousness. At core temperatures less than 90Â°F, respiratory rate, blood pressure, and pulse are depressed, and there is significant danger of pulmonary edema and fatal cardiac arrhythmia. Treatment is active rewarming with intravenous fluids, warmed peritoneal dialysis, etc under close monitoring. Prevention is through attention to nutrition and hydration needs, appropriate windproof and insulated layered clothing, avoidance of getting wet, and abstinence from alcohol.

C. Altitude Sickness

There is significant physiologic stress placed on the body when adapting to the lower barometric pressures and resultant hypoxia at high altitude (Table 1-8). Syndromes of high-altitude sickness are essentially maladaptations to this physiologic stress and range from mild, acute mountain sickness (AMS) to severe high-altitude pulmonary edema (HAPE) and high-altitude cerebral edema (HACE).

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Nonemergent & Chronic Medical Issues

Neurologic Disorders

A. Headache

Headaches are a common complaint in the general population as well as in the athlete. Specific exercises and sports-related headaches that must be considered in athletes are listed in Table 1-9.

Additionally, typical migraine, sinus, and tension headaches can be aggravated or induced by activity. In an athlete, a headache not only can impair performance, but may signal a more significant underlying medical problem and thus complaints should be taken seriously. Headache management can pose a challenge in this population because of side effects as well as restrictions on drug use by governing bodies. The workup for the athlete with headache starts with a detailed history and physical examination. The initial interview should include a search for a previous history of headaches as well as causative and precipitating factors. The physical examination should rule out neurologic deficits, cervical spine issues, and other contributing factors of concern. If an athlete presents with acute onset of severe headache, or if the headache is brought on by exertion, further workup is warranted. Findings of particular concern are shown in Table 1-10.

Exertional headaches occur in 12% of the general population and in up to 50% of athletes. Although most are benign in origin, studies show a 10â 40% association with underlying illness that includes intracranial mass, bleeds, and other significant pathology. A thorough investigation, often including blood work and imaging, is necessary when an obvious precipitating factor cannot be determined. A diagnosis of â benign exertional headacheâ can be made once organic disease and inciting factors other than exertion have been ruled out. The initial treatment is usually indocin. Ergots and triptans are also effective, but their use may be limited because of untoward side effects in the athletic population. An athlete with headaches may return to full activity once underlying disease is ruled out and the pain is adequately controlled.

B. Epilepsy

Seizures, caused by an abnormal paroxysmal neuronal discharge in the brain, are relatively common in the general population (with a lifetime risk of 10% and 1â 2% having a diagnosis of epilepsy). The prevalence in active individuals and specifically athletes has not been well studied. Trauma (ie, closed head injury) can cause transient seizure activity, but there is no evidence that this increases the overall risk of developing a chronic seizure disorder. Other factors associated with participation in sports can cause seizures and/or aggravate an underlying disorder, but the relative risk is believed to be low. Aerobic exercise has been shown, overall, to decrease seizure frequency, but it can also, at times, exacerbate a condition. Historically, seizure excluded people from participation in sports, but experience has dictated a more moderate approach in recent years. Current recommendations encourage physical activity and, in general, support involvement in athletics provided the seizure disorder is under adequate control. In counseling patients, the type of activity is clearly an important issue.

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Some groups recommend restrictions for certain sports, including sky diving, mountain climbing, and scuba diving, because of the potential for disaster should the athlete seize during the activity. Other cases of seizure and sports participation need to be evaluated on a case-by-case basis.

Table 1-8. High-altitude illness syndromes.

Syndrome[1]	Severity	Course	Altitude (feet)	Signs and Symptoms	Treatment and Prevention
AMS	Mild; self-limited	Very mild brain edema that occurs in	7,000â 10,000	Mild to moderate headache, loss of	Symptoms abate at the same altitude in 1â 2

		the first 2â 36 hours of arrival at moderate altitude		appetite, lethargy, nausea, vomiting	days without treatment Watch for worsening symptoms Prevent by slow ascent and/or acetazolamide (125 mg bid)
APE	With rapid treatment, all patients recover, but can rarely progress to death	Pulmonary edema, with onset at 2â 4 days. Often preceded by AMS	>10,000	Dry cough, SOB, decreased exercise tolerance, hypoxemia leads to increased dyspnea, frothy pink sputum, and death (if untreated)	Treat with low flow O_2 when available and descent by 2000â 3000 feet (if descent is not possible, a hyperbaric bag can temporize) Patient may be able to reascend once symptoms clear Prevent by slow ascent and/or nifedipine XL (30 mg qid)
ACE	Can be rapid in onset and fatal, but with acute treatment, most fully recover	Brain edemaâ severe end of AMS spectrum May occur with HAPE	>12,000	Severe headache, changes in mental status, ataxia, tachycardia, tachypnea	Treat with immediate descent, O_2, dexamethasone (10 mg IM followed by 4 mg PO qid) Patient cannot reascend Prevent with slow ascent

[1] AMS, acute mountain sickness; HAPE, high-altitude pulmonary edema; HACE, high-altitude cerebral edema.

Table 1-9. Differential diagnosis of exercise-related headaches.

Increased intracranial pressure
Traumatic: postconcussive, maxillofacial trauma
Metabolic: overtraining, hypoglycemia, anemia, acute mountain sickness, barotrauma (scuba diving), exercise induced asthma
Muscle tension: temporomandibular joint, C-spine degenerative joint disease/strain, facet syndrome, postural (cycling, wrestling)
Equipment related: goggle headache, occipital neuralgia (overtight headgear)
Depression
Eye strain
Analgesic rebound
Benign exertional headache
Pulmonary Disorders

A. Asthma

Asthma is characterized by airway obstruction (bronchospasm), inflammation, and hyperresponsiveness to stimuli such as allergens, chemicals, viral infections, cold air, or exercise. It affects approximately 10 million people in the United States. Once a contraindication to athletic participation, today, asthma should not prohibit participation in sports, with scuba diving as the exception, if adequate treatments are used.

Table 1-10. Headache â red flags.â

Severe headache reaches maximal intensity within a few seconds or minutes
â First or worstâ
Preceding infection
Rapid onset after trauma or with exercise, cough, or sexual activity
Associated neck/shoulder pain
Change in mental status, personality, or level of consciousness
Focal neurologic signs and symptoms (with or without other signs of increased intracranial pressure, focal or

infectious lesion)
Exercise-induced asthma (EIA) typically occurs 5â 10 minutes following, but may occur during, strenuous exercise and usually resolves spontaneously within 20â 30 minutes. Attacks are rarely life-threatening. Up to 20% of high-school athletes and up to 10% of world class athletes have been diagnosed with EIA. Although some athletes report wheezing, symptoms can vary widely and are often nonspecific, such as cough, shortness of breath, and chest tightness after exertion. Diagnosis is made by history and examination and with pulmonary function testing that reveals a decrease by at least 15% in forced expiratory volume in 1 second (FEV_1) after a free running challenge. Methacholine challenge testing is more sensitive than a running or ergometry challenge, but has a much lower specificity for EIA. Treatment of asthma and EIA should be individualized, but initial therapy is almost always with inhaled Î²-agonists. It should be noted that oral, long-acting Î²-agonists are banned by the NCAA and International Olympic Committee. Other pharmacologic treatments for prevention of attacks may include other bronchodilators (ie, anticholinergics) and antiinflammatories (glucocorticoids, khellin derivatives such as cromolyn, and leukotriene antagonists). Nonpharmacologic treatment can be useful in some cases. Approximately 50% of athletes with EIA are able to induce a â refractory periodâ following either 3â 4 minutes of high-intensity exercise or about 1 hour of low-intensity warm-up. Although aerobic training may have some preventive benefit, there is no way to predict whether a person will be able to induce a refractory period or not.
B. Chronic Obstructive Disease
Exercise in patients with chronic obstructive pulmonary disease, chronic bronchitis, and cystic fibrosis has been shown to decrease dyspnea and fatigue and to increase endurance and overall quality of life. Patients with mild to moderate disease should be allowed to participate in athletics based on severity of symptoms. In patients with chronic obstructive pulmonary disease, care should be taken when environmental conditions can increase airway reactivity, specifically cold and windy or hot and humid conditions. Those with cystic fibrosis lose more sodium and chloride in their sweat and therefore need to be counseled about proper hydration practices in the heat.
Cardiovascular Disorders
A. Hypertension
Hypertension is the most common cardiovascular condition in adults, affecting over 50 million people in the United States. Cardiovascular endurance and resistance
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exercise programs have been shown to have beneficial effects on hypertension of all levels and in general should be encouraged. Despite a lack of evidence for increased risk of sudden death or even of end-organ damage, persons with severe hypertension are believed to require limitation of activity, primarily from sports with high static demands. There are no limitations of activity for athletes with mild and moderate hypertension. In a newly diagnosed athlete, a thorough evaluation, including history, physical examination, EKG, and blood work (for evidence of target organ damage), is appropriate. Exercise stress testing in all patients over the age of 35 years with hypertension (even in the absence of other cardiac risk factors) has been recommended, but the utility of this is debatable. Further evaluation may be necessary if a secondary cause is suspected.
Antihypertensive treatment should be implemented in athletes with the goal of minimizing organ damage just as in a nonathletic population, but many of the more commonly used medications can affect performance and may not be well tolerated. Diuretics may lead to dehydration and hypokalemia, especially in endurance athletes. Î²-Blockers may produce fatigue and decreased exercise tolerance. Both diuretics and Î²-blockers are banned by the International Olympic Committee. Angiotensin-converting enzyme (ACE) inhibitors and calcium channel blockers tend to be well tolerated and efficacious and should be considered first-line therapy in most athletes unless accompanying medical issues dictate otherwise.
B. Valvular and Congenital Disease
In an athlete with valvular or congenital disease, recommendations for participation in competitive sports are based on several factors including type and severity of lesion, ventricular function, presence of arrhythmias or altered hemodynamics, and presence of other cardiac abnormalities. Recommendations that have been set by the 36th Bethesda Conference are not based on precise information, but nonetheless are believed to follow prudent judgment. In general, full participation should be allowed in athletes with mild, asymptomatic disease. In the setting of mitral valve prolapse, mild stenosis (aortic, mitral, pulmonic), or mild to moderate

regurgitation, it should be demonstrated that the athlete has normal exercise tolerance and no signs of ventricular enlargement, abnormal ventricular function, or arrhythmias before full clearance. Persons with small septal defects (atrial or ventricular), or those with small patent ductus arteriosus, can participate without restriction when there is no accompanying pulmonary hypertension, arrhythmias, or evidence of myocardial dysfunction. Athletes who have more significant disorders must be evaluated on an individual basis.

C. Arrhythmias

Arrhythmias that can potentially lead to sudden cardiac death, such as Wolffâ Parkinsonâ White (WPW) and long-QT syndrome, have already been discussed briefly, but it is important to note that there are many cardiac arrhythmias that pose little if any threat to the athlete and therefore should not limit activity. Marked sinus bradycardia, first-degree and type I second-degree (Wenckebach type) atrioventricular (AV) block, and uniform premature ventricular contractions occur frequently in healthy athletes, often directly related to their conditioning. These individuals nonetheless require evaluation and periodic follow-up. The type and complexity of an arrhythmia, the presence of structural heart disease, associated ventricular dysfunction or ischemia, and the response of the arrhythmia to exercise determine its significance. A full evaluation of and recommendations for specific arrhythmias, which are beyond the scope of this chapter, can be referenced in the 36th Bethesda Conference report.

Endocrine Disorders

A. Diabetes Mellitus

Diabetes mellitus, characterized by relative or absolute insulin deficiency, is extremely common in the general population, affecting approximately 17 million people in the United States (1 million with type I and 16 million with type II). Most patients with diabetes mellitus can safely exercise and even participate in elite level competitive sports. Adequate glucose control is extremely important to minimize risk and optimize performance. Although it is recommended that athletes with diabetes with complications such as nephropathy, neuropathy, and retinopathy refrain from certain high-intensity sports, in general, regular physical activity should be encouraged. In addition to the desirable affects seen in nondiabetics, this population often sees even more profound improvements in overall well being, weight control, lipid profile, and other cardiac risks. Improved glycemic control leads to a reduction in microvascular complications, diabetes-related deaths, and all-cause mortality (35%, 25%, and 7% reduction, respectively, for each percentage point reduction in hemoglobin (Hgb)A_{1C}. This is usually the result of moderate caloric restriction and regular exercise, and is enhanced by exercise-induced weight loss and resultant improved insulin sensitivity.

The benefits of regular exercise can be dramatic, but there are serious risks as well. The major risks for most athletes with diabetes involve complications in metabolic control, specifically hypoglycemia. Hypoglycemia can occur during or after exercise if caloric intake and/or medications have not been properly adjusted. Patients on insulin or sulfonylureas tend to be at higher risk for this complication. Symptoms of hypoglycemia are variable, but often include dizziness, weakness, blurred vision, confusion, diaphoresis, nausea, cool skin, and/or parasthesias of the tongue or hands. Recommendations for minimizing the risk of hypoglycemia in the active individual are listed in Table 1-11.

Table 1-11. Minimizing hypoglycemic risk.[1]

Closely monitor glucose levels before, during, and after activity

Daily morning exercise (as opposed to sporadic exercise) facilitates medication and caloric adjustments

Ensure immediate access to glucose (oral carbohydrates or SQ/IM 1 mg glucagon injection) if necessary

Adjust medications/food intake

Insulin adjustments before exercise

Avoid insulin injection into exercising extremityâ abdomen is preferred site

Decrease short-acting insulin based on planned minutes of exercise as follows: decrease dose by 30% for <60 minutes, by 40% for 60â 90 minutes, and by 50% for >90 minutes of planned exercise; intense exercise may require even further reductions

Decrease intermediate insulin (neutral protamine Hagedorn; NPH) by one-third (33%)

Consider using Lispro (faster onset, shorter duration)

For insulin pumps, decrease basal rate by 50% 1â 3 hours before and during exercise

If exercise is planned immediately after a meal, reduce premeal bolus by 50%

Food intake adjustment

Eat a well-balanced meal 2â 3 hours prior to exercise

Take a carbohydrate snack just before exercise if glucose is <100 (15 g of carbohydrates raises glucose

approximately 50 mg/dL)
Eat 30â 60 g carbohydrate/hour of activity (when >1 hour)
Maintain adequate hydration
[1] Note that these are general recommendations. Each patient-athlete needs individualized assessment and adjustments.
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Delayed-onset hypoglycemia often occurs at night, 6â 15 hours after exercise and therefore can potentially be even more dangerous. It is usually brought on by inadequate replenishment of glycogen stores immediately after exercise and in the ensuing hours. Delayed-onset hypoglycemia may develop as long as 30 hours after exercise, reflecting continued exercise-heightened insulin sensitivity with increases in glucose uptake and glycogen synthesis in skeletal muscle. Glycogen is repleated more slowly in liver than in muscle, so carbohydrate requirements may be increased for up to 24 hours after prolonged exercise.
Hyperglycemia is also a potential danger secondary to increased hepatic glucose production. This is seen with a rise in counterregulatory hormones: epinephrine, norepinephrine, glucagon, cortisol, and growth hormone. Diabetic ketoacidosis can result in patients with insulin-dependent diabetes and hyperosmolar coma can result in those with non-insulin-dependent diabetes mellitus.
Because of these risks, exercise should be avoided if glucose levels are greater than 250 mg/dL and ketosis is present. In the absence of ketosis, exercise may be allowed with glucose levels greater than 300 mg/dL, but extreme caution is recommended. Because of the significant cardiovascular risk associated with diabetes mellitus, physicians must have a heightened awareness of issues in this population and potentially a lower threshold for cardiac screening. The American Diabetes Association recommends exercise stress testing if moderate to high-intensity activity is planned in patients with any of the conditions listed in Table 1-12.
Finally, foot problems can be a major issue in active patients with diabetes. Although a full discussion of these foot problems will not be included here, it is important to mention them as a great source of morbidity. It is imperative that physicians working with patients with diabetes and promoting active life-styles must also educate patients regarding proper shoes that fit well, have a wide enough toe box, and cushioned mid-sole, moisture-wicking socks, and appropriate foot hygiene to avoid problems.
B. Thyroid Disorders
Although they rarely limit athletic participation, thyroid disorders are quite common, affecting approximately 5% of the general population. Hypothyroidism results from insufficient thyroid hormone secretion and presents with
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decreased exercise tolerance, lethargy, muscle ache, constipation, and intolerance to cold. It may also be part of a syndrome of proximal muscle weakness and fatigue with elevated CPK levels that may initially be confused with rhabdomyolysis. Hyperthyroidism causes hypermetabolism due to excessive hormone secretion. Patient-athletes complain of tremors, nervousness, palpitations, fatigue, proximal muscle weakness, and intolerance to heat. Women may present with oligo/amenorrhea. Diagnosis of a thyroid disorder is made by laboratory history, physical examination, and laboratory testing for thyroid-stimulating hormone (TSH) and thyroxine (T_4). Hypothyroidism is treated with hormone replacement. Hyperthyroidism is treated with antithyroid medications, radioactive iodine, or surgery. The effects of exercise on thyroid function are not clear, therefore, guidelines for return to play after treatment have not been established. In general, there are no absolute restrictions, but the athlete should be medically stable and able to tolerate the intensity of exercise demanded by the sport. It may be prudent to require several weeks of a persistent euthyroid state, especially in patients who have suffered cardiac manifestations, before allowing progression to intense activity. The athlete should then be followed clinically and through laboratory testing on a regular basis.

Table 1-12. American Diabetes Association recommendations for exercise stress testing.

Age >35 years
Diabetes mellitus I >15 years duration
Diabetes mellitus II >10 years duration
Known coronary artery disease
Additional coronary artery disease risk factors (hypertension, tobacco use, family history, cholesterol)
Presence of microvascular disease

Peripheral vascular disease
Autonomic neuropathy
Gastrointestinal Problems
Gastrointestinal problems are common in the general population and up to 60% of competitive athletes complain of symptoms. The problems vary widely according to the specific sport, condition of the athlete, level of intensity, and other factors.
A. Nausea and Vomiting
Nausea and vomiting are frequent occurrences in athletes. They are very common in athletes who simply exceed their exertional capacity, but can also be seen with anxiety, heat illness, hypoglycemia, head injury, and other significant issues. In females, pregnancy should be considered. In the absence of other etiology, treatment consists of rest and rehydration (occasionally with intravenous fluids if the athlete is unable to take fluids orally). In some extreme cases antiemetics (compazine, tigan, thorazine) are useful adjuncts.
B. Gastroesophageal Reflux Disease
Studies have shown that vigorous exercise can induce gastroesophageal reflux disease (GERD) even in normal subjects. Running and swimming seem to cause the majority of the problems related to esophageal sphincter relaxation. Although there are no good studies on the treatment of exercise-related GERD, it is accepted that most young people with symptoms such as belching, heartburn, and regurgitation can be treated without further diagnostic workup. The initial treatment consists of limiting food intake in the several hours preceding exercise, avoiding foods that delay gastric emptying (fatty foods), and using non-magnesium-containing antacids. If this is unsuccessful, H2 blockers should be used; proton pump inhibitors may be necessary in refractory cases. Individuals who have persistent problems, or those who experience abnormal symptoms such as dysphagia or weight loss, must be evaluated with further diagnostic studies.
C. Abdominal â Stitchâ
Transient, sharp, subcostal pain, referred to as a â stitch,â is well known by athletes. It is an entity of unclear etiology, possibly attributed to gas, ischemia, or muscle spasm. It is most often experienced by runners, is exacerbated by deep breathing, and is decreased by rest. Frequency tends to decline with endurance training and does not typically require further investigation unless pain persists.
D. â Runner's Diarrheaâ
Among endurance athletes, cramps, urgency, diarrhea, and incontinence are some of the most common and bothersome of symptoms experienced. It is speculated that the repetitive jarring of foot-strike during running may stimulate mass movements in the colon. This â runner's diarrheaâ often occurs during or immediately following high-intensity exertion. Initial management is dietary adjustment (eg, limiting high-lactose and high-fructose foods). If non-exercise-related causes (infection, irritable bowel disease, malabsorption, cancer) are ruled out and dietary changes are ineffective, use of a prophylactic antidiarrheal 1 hour prior to activity can be considered.
E. Gastrointestinal Bleeding
Although it has been shown that up to 20% of marathon runners have occult blood in their stools following competition, gastrointestinal (GI) bleeding is in general relatively uncommon in otherwise healthy athletes. Positive guiac testing in endurance athletes has been thought, in many cases, to be related to use of nonsteroidal antiinflammatory drugs, but there are no studies to date showing a correlation. Other theories include GI ischemia secondary to decreased splanchnic blood flow and simple biomechanical trauma from repetitive jarring during running. Most cases are self-limited, but athletes with GI bleeding should, nonetheless, be considered for further medical evaluation to rule out pathologic causes.
Genitourinary Issues
A. Chronic Renal Failure
Research regarding chronic renal disease and exercise has not been at the forefront (in contradiction to cardiac, pulmonary, and neurologic issues). Despite the lack of aggressive prospective trials, there have been many studies
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suggesting improvement in gait speed, strength, muscle mass, hematocrit, and overall function in patients with chronic renal failure who participate in regular physical activity. This is believed to be particularly important in the preservation of muscle mass for those on a low-protein diet. Although there are significant obstacles in

many patients including anemia, severe muscle fatigue (with vitamin D deficiency and secondary hypoparathyroidism, androgen abnormalities), and steroid myopathies, dramatic benefits from strength and aerobic training have been obtained by patients who are predialysis, on chronic dialysis, and even posttransplant. It is therefore routinely recommended that these patients engage in a low-to-moderate intensity regular workout program keeping individual limitations in mind.

B. Single-Organ Athlete

The overall risk of losing a kidney due to contact sports is very low, but nonetheless, a major consideration in competitive athletes with one functional kidney. Current recommendations are that these athletes avoid all collision sports. Limited-contact sports are felt to be safe if the solitary kidney is normal in anatomy and function. Protective equipment (eg, a flak jacket) may improve this safety even further and, when appropriate, should be used. The athlete with one abnormal kidney (pelvic, multicystic), however, should likely be precluded from contact sports. In the athlete with a single testicle, most contact and collision sports are felt to be safe if a protective cup is worn. In all cases of single organs, proper education regarding risks is imperative.

Infectious Diseases

A. Upper Respiratory Infection

Acute infections are associated with a variety of immune system responses that are triggered by cytokines and are correlated with fever, muscle pain, fatigue, and anorexia, along with other signs and symptoms. Acute viral and bacterial illness can potentially hinder exercise ability by affecting multiple body systems, including cardiopulmonary function, fluid status, and temperature regulation. Current recommendations regarding exercise and participation in sports follow a â neck checkâ approach. Because of potentially detrimental effects, patient-athletes with symptoms â below the neckâ (ie, fever, chills, chest congestion, ongoing diarrhea, or nausea/vomiting) should refrain from intense exercise. However, in patient-athletes with has symptoms only â above the neckâ (ie, nasal congestion, sore throat), continued participation in sports as tolerated is reasonable. Because no research offers clear evidence-based guidelines regarding exercise during viral infections, degree and manifestation of illness, as well as type of sport, intensity of training, potential risk of spreading disease, and other factors, should be considered in each case.

B. Myocarditis

Myocarditis is an inflammatory condition of the myocardial wall most commonly caused by coxsackievirus B infection. It is a rare cause of sudden cardiac death in athletes. The typical clinical picture consists of fatigue, chest pain, dyspnea, and, occasionally, palpitations. There are no accurate predictors of risk of sudden death in patients with myocarditis, but because of the potential, the 26th Bethesda Conference guidelines take a conservative stance, recommending withdrawal from all competitive sports for about 6 months. Before returning to competition, the athlete should demonstrate normal ventricular function and dimensions on echocardiography and no signs of arrhythmia with ambulatory monitoring.

C. Mononucleosis

Infectious mononucleosis is caused by the Epsteinâ Barr virus and is typically characterized by fatigue, sore throat, tonsillar enlargement, lymphadenopathy, and splenomegaly. Activities are often self-restricted because of severe malaise and inability to perform hard physical exertion. The literature suggests that athletes may begin a noncontact exercise program as soon as they become afebrile without detrimental effects. Splenic involvement with mononucleosis and potential rupture are the primary concerns for most clinicians. Rupture occurs in 0.1â 0.5% of cases. The majority are spontaneous and occur within the first 3 weeks from onset of illness when there is profuse lymphocytic infiltration putting the spleen in an enlarged and â fragileâ state. There are no clear guidelines on whether to use palpation or an imaging technique (eg, ultrasound) to determine splenic size and therefore presumptive risk of rupture. Although it is well documented that palpation alone has a low sensitivity for splenic enlargement, return to play decisions are based on the ability to palpate the organ, implying that the rib cage can adequately protect even an enlarged spleen. Again, there is no evidence for or against this assumption. Although there have been only a few cases of splenic rupture associated with participation in sports reported in the literature, a prudent course is still recommended, particularly within the first few weeks of illness. The American Academy of Pediatricians recommends that a patient with an acutely enlarged spleen should avoid all sports and that a patient-athlete with a chronically enlarged spleen needs individual assessment before participation.

D. Hepatitis

Viral hepatitis can present as a broad spectrum of clinical syndromes ranging from asymptomatic to fulminant and fatal. Common symptoms of acute infection include fatigue, myalgia, arthralgias, anorexia, and nausea.