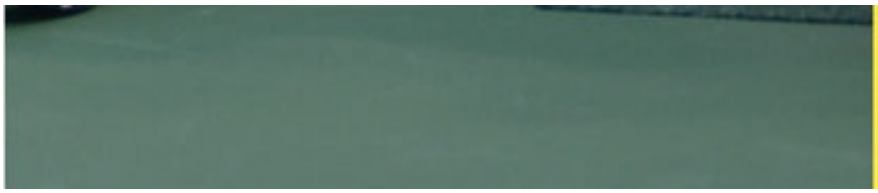
Basic Athletic Training
Course Pack D

Systemic Conditions and Special Considerations

SECTION

VII



Diseases

CHAPTER 32

Dermatology

CHAPTER

24

Cardiovascular Disorders

STUDENT OUTCOMES

1. Describe the signs and symptoms of anemia and explain the potential impact of this blood disorder on sport and physical activity performance.
2. Explain the methods for minimizing the effects of anemia on sport and physical activity performance.
3. Identify the early signs of hemophilia and describe the treatment of this condition.
4. Describe Reye syndrome and the strategies for preventing it.
5. Describe the physiological causes, signs, symptoms, and management of syncope.

6. Describe the basic physiological principles, signs, symptoms, and management of shock.
7. Identify the four stages of hypertension and state the predisposing factors or diseases that place an individual at risk for developing hypertension.
8. Identify medications that can adversely elevate blood pressure.
9. Identify the factors and conditions that can lead to hypotension.
10. Describe the management of hypertension and hypotension.
11. Identify the epidemiological factors and basic physiological principles associated with sudden death in the physically active population.
12. Identify the cardiac-related causes of sudden death in individuals younger than and older than 30 years.
13. List the risk factors for coronary artery disease and Marfan syndrome.
14. Identify the noncardiac conditions that can lead to sudden death.
15. Describe the two-tiered approach that is used during a preparticipation physical examination to identify risk factors associated with sudden death.
16. Explain the ethical, legal, and practical considerations that affect the medical decision-making process for determining the eligibility of competitive athletes who have cardiovascular abnormalities.

INTRODUCTION

Participation in physical activity and sport yields positive effects on an individual's physical, mental, and social health, but participation in any

physical activity has inherent risks of injury. Preparticipation physical examinations (PPEs) often can identify conditions that may predispose an individual to injury. Even with a PPE, however, certain conditions may go undetected. As such, the chance exists for severe—and even catastrophic—injury because of preexisting conditions. Blood disorders (e.g., anemia and hemophilia) and cardiovascular disorders (e.g., syncope, hypertension, and hypotension) are conditions that warrant medical attention. If left untreated, these conditions can have serious physical effects on the body and can become even more problematic if they occur during exercise.

This chapter will provide a basic overview of the anatomy of the cardiovascular system, with a focus on the heart. (Discussion on the bronchial trees and lungs is included with Chapter 26.) Next, the causes, identification, and management of various cardiovascular diseases and disorders in the physically active population will be discussed. Recommendations are provided to reduce the risk of these conditions by means of a thorough cardiovascular history as part of preparticipation screening. Finally, issues concerning counseling individuals at risk for sudden death are discussed.

ANATOMY OF THE CIRCULATORY SYSTEM

The circulatory system includes the cardiovascular system and the lymphatic system. The heart and lungs have an intimate relationship both physically and functionally. The heart is positioned obliquely to the left of the midline of the body and is divided into four chambers: the right and left atria superiorly, and the right and left ventricles inferiorly (**Fig. 24.1**). The heartbeat consists of a simultaneous contraction of the two atria, followed immediately by a simultaneous contraction of the two ventricles. The contraction phase is known as systole; the phase in which the chambers relax and fill with blood is known as diastole. Two pairs of valves within the heart ensure that blood flow is unidirectional. The atrioventricular valves seal off the atria during contraction of the ventricles to prevent the backflow of blood. The aortic and pulmonary semilunar valves prevent the flow of blood from the aorta and pulmonary

artery back into the ventricles.

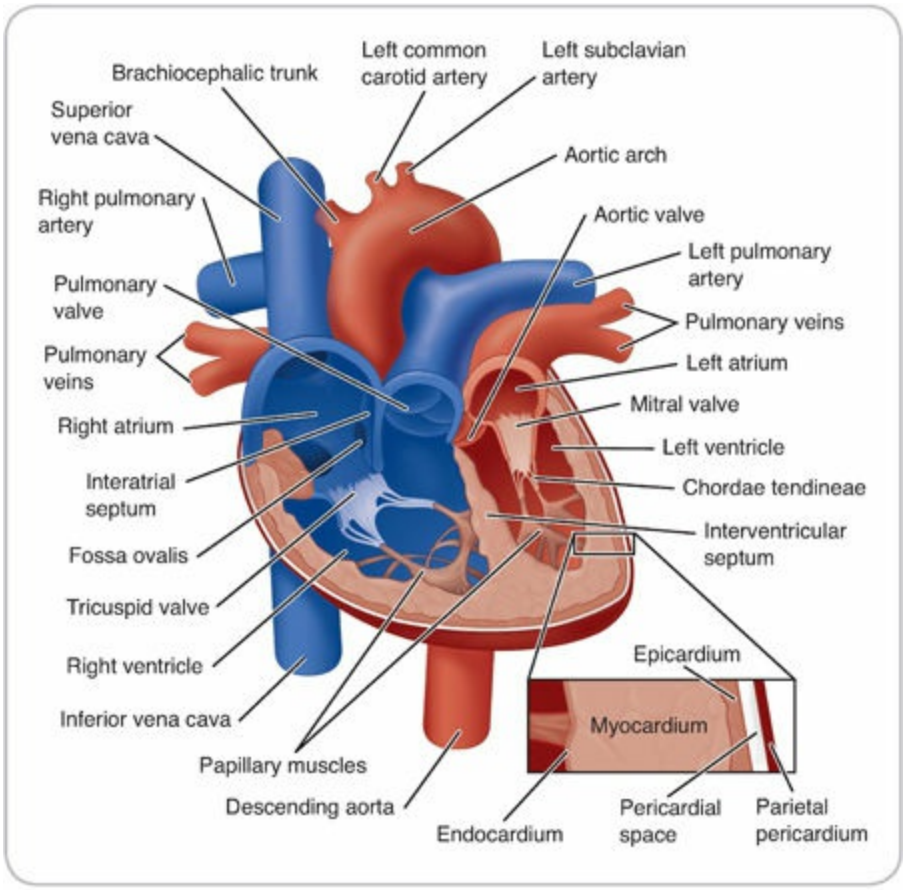


Figure 24.1. Heart.

The right side of the heart pumps blood to the lungs, where carbon dioxide and oxygen are exchanged. The left side of the heart receives the freshly oxygenated blood from the lungs and pumps it out to the systemic circulation. The vessels interconnecting the heart and lungs are known as the pulmonary circuit; the vessels that supply the body are known as the systemic circuit (Fig. 24.2). The systemic circulation is composed of numerous and different circuits in parallel, which allows wide variations in regional blood flow without changing the total systemic flow. The heart and blood vessels (e.g., arteries, arterioles, veins, venules, and capillaries) transport oxygen, nutrients, and hormones to cells; remove waste products and carbon dioxide from cells; defend the body against infections; and prevent blood loss through clotting.

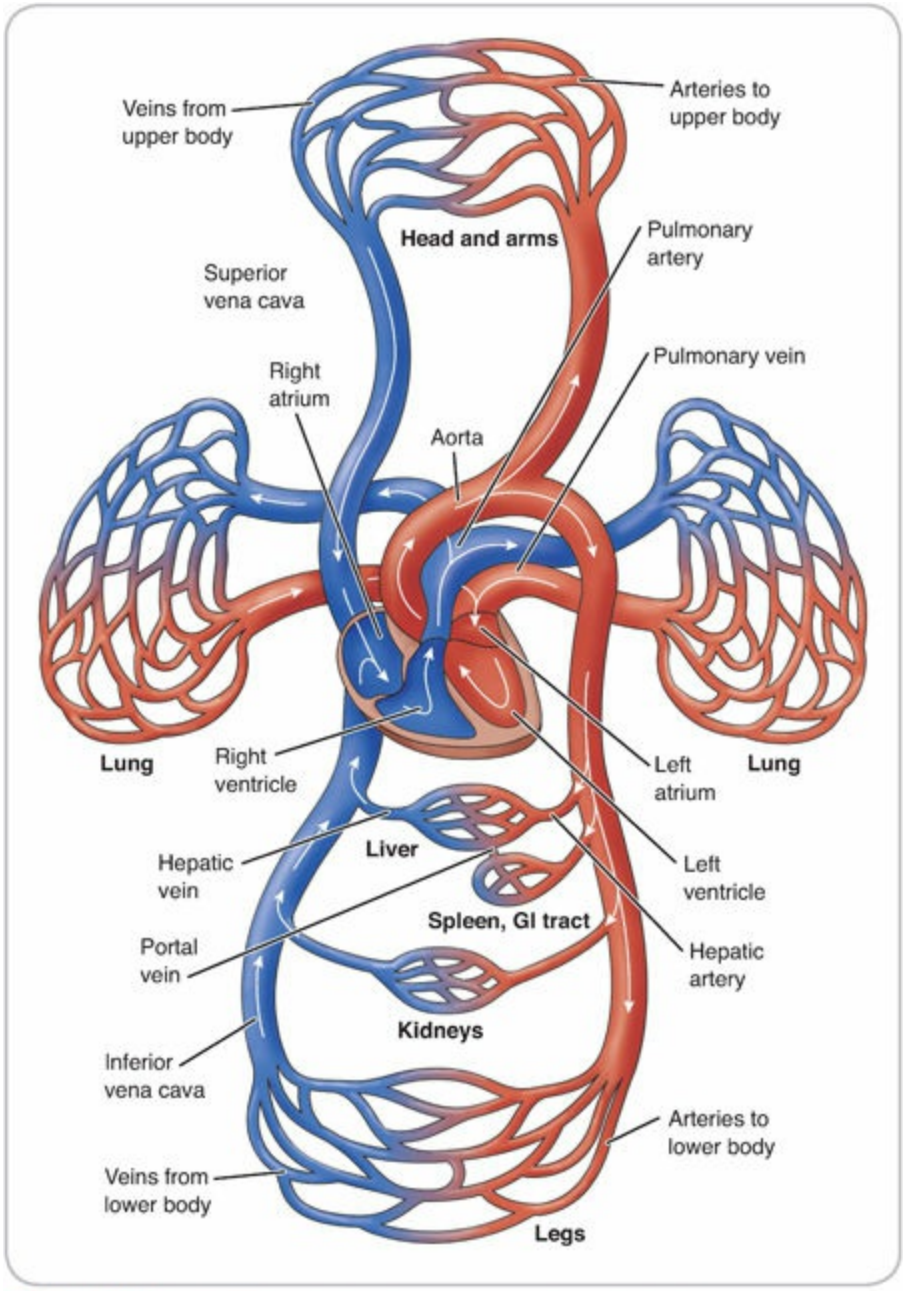


Figure 24.2. Pulmonary and systemic circulation in an adult.

Functioning as part of the immune system, the lymphatic system is composed of lymph capillaries (i.e., lacteals), nodes, vessels, and ducts (Fig. 24.3). This one-way system transports fluids, nutrients (e.g., fats and proteins), and tissue waste back into the bloodstream via connections with major veins, and it flows only toward the heart.

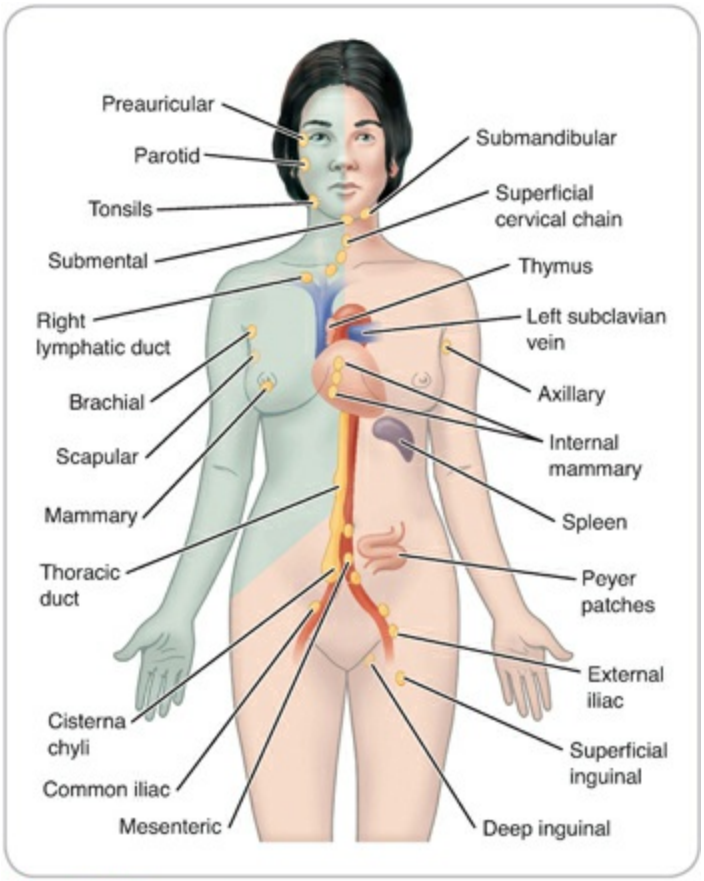


Figure 24.3. Lymphatic system with major lymph nodes. The *pale green area* denotes the body area drained by the right lymphatic duct; the rest of the body is drained by the thoracic duct.

BLOOD AND LYMPH DISORDERS

? A 30-year-old female recreational golfer is diagnosed with iron-deficiency anemia attributed to an inadequate diet. What implications will this have on her ability to continue participating in golf on a weekly basis, and what is the management for this condition?

Blood flow delivers nutrients to and delivers wastes from cells and is involved in gas exchange, absorbing nutrients, and forming urine. Lymphatic vessels return fluids that have leaked from the vascular system back to the blood, protect the body by removing foreign material from the lymph stream, and provide a site for immune surveillance. Blood and lymph disorders

presented in this chapter include anemia, hemophilia, Reye syndrome, and lymphangitis.

Anemia

Iron, which is present in all human cells, serves several functions, including carrying oxygen from the lungs to the tissues in the form of hemoglobin, facilitating oxygen use and storage in the muscles as myoglobin, serving as a transport medium for electrons within the cells in the form of cytochromes, and playing an integral part of enzyme reactions in various tissues. Insufficient amounts of iron can interfere with these vital functions and lead to serious illness or even death. A reduction in either the red blood cell (RBC) volume (i.e., hematocrit) or the hemoglobin concentration is called **anemia**. Although anemia has five separate classifications, each one is caused by impaired RBC formation, excessive loss, or destruction of RBCs.

The recommended dietary allowance (RDA) for iron in men is 8 mg per day and in women 18 mg per day except for pregnant women, in which case the RDA is 27 mg per day. The average diet contains 5 to 7 mg of iron per 1,000 kcal. Therefore, it is necessary to consume 3,000 kcal per day to meet the RDA. A major concern is that many physically active females consume less than 2,000 kcal per day. In addition, individuals consuming a modified vegetarian diet that is low in iron content and low in iron bioavailability may not be receiving sufficient iron. Good sources of iron include lean animal meat (e.g., liver, beef roasts, tenderloin, lamb, and chicken or turkey legs), tuna, oysters, shrimp, enriched raisin bran or corn flakes, bagels, bran muffins, dried apricots, baked potatoes with the skin, peas, kidney beans, chickpeas, tofu, and molasses.

Anemia reduces maximum aerobic capacity, decreases physical work capability at submaximal levels, increases lactic acidosis, increases fatigue, and decreases exercise time to exhaustion.[1] Although several predisposing factors can increase the risk of developing anemia (Box 24.1), physically active individuals tend to be more prone to specific anemic conditions.

BOX 24.1 Predisposing Factors for Development of Anemia

- Personal or family history of anemia, bleeding disorders, or chronic disease
- Intermittent jaundice early in life
- Excessive menstrual flow; increased duration, frequency, or volume
- Chronic blood loss through gastrointestinal bleeding
- Certain drugs and toxins (e.g., chronic use of aspirin or nonsteroidal anti-inflammatory drugs)
- Childbirth
- Disadvantaged socioeconomic background
- Poor diet or dietary restriction (e.g., vegetarian diet, weight loss diets, or fad diets)
- Cancer
- Volunteer blood donor
- Diminished hepatic, renal, or thyroid function

Adapted from Harris SS. Helping active women avoid anemia. *Phys Sportsmed*. 1995;23(5):35–48; with permission.

Stages of Anemia

Iron status is assessed through several laboratory tests. Because each test assesses a different aspect of iron metabolism, the results of one test may not always agree with those of other tests. The hemoglobin concentration and hematocrit commonly are tested, because they measure the amount of functional iron in the body. The concentration of hemoglobin in circulating RBCs is the more direct and sensitive measure. The hematocrit indicates the proportion of whole blood that is occupied by the RBCs, and it decreases only after the hemoglobin concentration falls. Hematological tests based on the characteristics of RBCs (i.e., hemoglobin, hematocrit, and total iron-binding capacity) are more popular and less expensive than biochemical tests. The

biochemical tests (e.g., free erythrocyte protoporphyrin concentration, serum ferritin concentration, and transferrin saturation), however, detect earlier changes in iron status.

Iron deficiency develops gradually, progressing through several stages before anemia is evident. These stages include the following:

- **Stage I.** Iron depletion is characterized by less than 12 mg per mL of an iron–protein complex known as ferritin, which is an indicator of reduced iron stores in the bone marrow. Other components of iron status—hemoglobin, hematocrit, free erythrocyte protoporphyrin, serum iron, iron-binding capacity, and transferrin saturation—remain normal.
- **Stage II.** Iron-deficiency erythropoiesis follows several months of iron depletion and is characterized by decreased levels of circulating iron, but hemoglobin and hematocrit remain normal.
- **Stage III.** Iron-deficiency anemia follows several weeks of iron-deficient erythropoiesis. Hemoglobin production diminishes, and the individual develops clinically recognized iron-deficiency anemia, which is referred to as frank anemia.

Iron-Deficiency Anemia

Etiology

Iron deficiency is the most common nutritional deficiency worldwide. Among men 18 years and older and postmenopausal women in the United States, however, iron-deficiency anemia is uncommon. During infancy and childhood, inadequate diet may be an underlying factor. In adults, blood loss through heavy menstrual bleeding or through slow, chronic bleeding associated with gastrointestinal ulcers, intestinal polyps, hemorrhoids, or cancer (e.g., uterine, colon) can lead to the condition. Other conditions that may increase the risk of anemia include alcoholism, use of certain medications (e.g., chemotherapy drugs, HIV medications, and seizure medications), autoimmune diseases, and insecticide exposure. The condition also is seen in endurance athletes and individuals who maintain a low percentage of body fat.

Signs and Symptoms

Early symptoms include exercise fatigue, tachycardia, blood mixed with feces, pallor, and epithelial abnormalities, such as a sore tongue. Symptoms that can develop at a later time include cardiac murmurs, congestive heart failure, loss of hair, and pearly sclera. Additional signs and symptoms of iron-deficiency anemia include the following:

- Muscle burning
- Nausea
- Shortness of breath
- Appetite for substances that have little or no nutritional value (e.g., starch, ice, or clay)
- Palpitations
- Spoon-shaped nails (koilonychia)
- Dry scaling and fissures of the lips (angular cheilosis)
- Inflammation of the tongue (glossitis)

Management

Treatment includes dietary iron supplementation (e.g., ferrous sulfate or ferrous gluconate) and ascorbic acid (i.e., vitamin C) to enhance iron absorption. Because caffeine hampers iron absorption, avoid colas, coffee, tea, chocolates, and other caffeine products. If active bleeding is attributed to polyps, ulcers, malignancies, or hemorrhoids, surgery may be necessary.

Exercise-Induced Hemolytic Anemia

Etiology

Exercise-induced hemolytic anemia, sometimes referred to as **runner's anemia**, occurs during exercise when RBCs are destroyed and hemoglobin is liberated into the medium in which the cells are suspended. This process of intravascular hemolysis can occur in both high- and low-impact sports and

physical activities. In high-impact activities, such as running, it is posited that the trauma of repetitive, hard foot strikes destroys the RBCs. This condition, which sometimes is referred to as **foot strike hemolysis**, is more commonly observed in marathoners and middle-age distance runners, particularly those who are overweight, run on hard surfaces, wear poorly cushioned shoes, and run with a stomping gait. Intravascular hemolysis, however, also has been reported in competitive swimmers and rowers. As such, damage to the cells could be attributed to other possibilities, including muscle contraction, acidosis, or increased body temperature.

Management

Runner's anemia rarely is severe enough to cause appreciable loss of iron. In general, the condition may be of more concern to highly competitive, world-class athletes, for whom a fractional physiological difference can result in a competitive disadvantage. Prevention and treatment focuses on encouraging runners to be lean, to run on soft surfaces, to avoid running with a stomping gait, and to wear well-cushioned shoes and insoles.

Sickle Cell Anemia

Etiology

The sickle cell gene is common in people whose origin is areas where malaria is widespread, such as in Africa, the Mediterranean, the Middle East, India, the Caribbean, and South and Central America, hence the required screening of all newborns in the United States.[2] **Sickle cell anemia** is more common in African Americans and results from abnormalities in hemoglobin structure producing a characteristic sickle- or crescent-shaped RBC that is fragile and unable to transport oxygen. The condition is attributed to inheriting an autosomal recessive gene or to possessing two sickle genes as opposed to having the sickle cell trait, in which only one sickle gene is inherited. Because of their rigidity and irregular shape, sickle cells clump together and block small blood vessels, leading to vascular occlusions, or infarcts, in the central nervous system and organs, such as the heart, lungs, kidneys, and spleen (**Fig.**

24.4). Although an individual with sickle cell trait may be asymptomatic for one's entire life, exercising excessively in high heat or humidity or at high altitude may lead to dehydration, increased body temperature, hypoxia, and acidosis, which are predisposing factors for increased protein concentration in the circulating blood cells. This high concentration of protein increases blood viscosity and impairs blood flow, which can lead to a stroke, congestive heart failure, acute renal failure, pulmonary embolism, or sudden death.

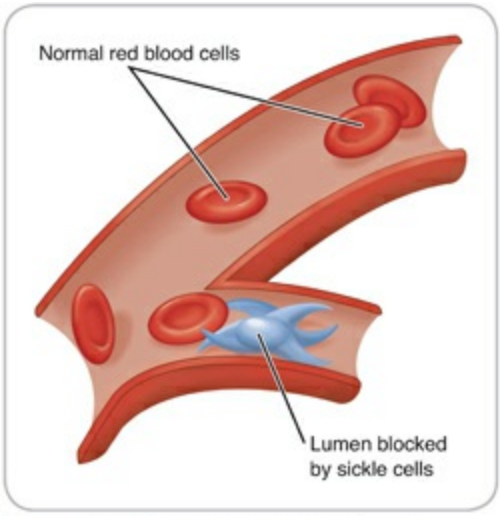


Figure 24.4. The sickle cell. Sickle cells are abnormal RBCs that are fragile and unable to transport oxygen. Because of their rigidity and irregular shape, they often clump together and block small blood vessels, leading to vascular occlusions in the vital organs.

Signs and Symptoms

Individuals with sickle cell anemia may be asymptomatic. When manifested, signs and symptoms may include recurrent bouts of swollen, painful, and inflamed hands and feet, irregular heartbeat, severe fatigue, headache, pallor, muscle cramping, and severe pain because of oxygen deprivation. The sclera of the eye also may appear to be jaundiced. Confusion may exist in differentiating between sickling and heat cramping. Heat cramping is often preceded by muscle twinges; sickling has none. People with heat cramps hobble to a stop, whereas sickling individuals collapse to the ground with weak muscles. Those with heat cramps yell in pain and contracted, rock hard muscles are noticeable, whereas sickling individuals lie still, do not yell in

pain, and the muscles look normal.[2]

Management

Currently, no treatment is known to reverse the condition. Because dehydration can complicate the condition, individuals should hydrate maximally before, during, and after exercise or physical exertion. Liquids with caffeine should be avoided because of their diuretic effect. Individuals should build up slowly during their physical training, allowing for longer periods of rest and recovery between repetitions. Because ambient heat stress, dehydration, asthma, illness, and altitude predispose an athlete with sickle cell trait to an onset of crisis due to physical exertion, efforts should be made to emphasize hydration, control asthma, prohibit exertion if the athlete is ill, adjust the work/rest cycles during environmental heat stress, and provide supplemental oxygen for competitions in individuals new to altitude.[3]

Hemophilia

Etiology

Hemophilia is a bleeding disorder characterized by a deficiency of selected proteins in the body's blood-clotting system. Three categories of blood proteins play a role in blood clotting:

1. Procoagulant proteins help to form clots.
2. Anticoagulant proteins prevent the formation of clots.
3. Fibrinolytic proteins help to dissolve clots that have formed.

The clotting process involves blood particles, called platelets, and procoagulant plasma proteins, called clotting factors. The process begins with the platelet phase, during which the platelets stick to a blood vessel at the site of an injury. An intricate cascade of enzyme reactions occurs, producing a web-like protein network that encircles the platelets and holds them in place. This is followed by the coagulation phase in which clot formation begins. In this cascade, each clotting factor is transformed from an inactive to an active form.

Hemophilia occurs in three types, which are differentiated by the deficient clotting factor. Hemophilias A, B, and C are inherited diseases. Because of the pattern of inheritance, hemophilias A and B occur almost always in males; hemophilia C can occur in both males and females. All three types, however, can cause prolonged bleeding. Complications may occur from the disease or from treatment of the disease (Box 24.2).[4]

BOX 24.2 Complications of Hemophilia

- **Deep internal bleeding.** Hemophilia may cause deep muscle bleeding. Swelling of a limb may press on nerves and lead to numbness or pain, which may result in reluctance to use the limb.
- **Damage to joints.** Internal bleeding may put pressure on and damage joints. Pain may be severe, resulting in reluctance to use a limb or joint. If bleeding occurs frequently and goes untreated, the irritation to a joint may lead to destruction of that joint or to the development of arthritis.
- **Infection.** People with hemophilia are more likely to receive blood transfusions and, as such, are at a greater risk of receiving contaminated blood products. Although risk of infection through blood products has decreased substantially since the introduction of genetically engineered clotting products, called recombinant factors, that are free of infection, it is still possible for people who rely on blood products to contract other diseases, such as infection with the HIV and hepatitis.
- **Adverse reaction to clotting factor treatment.** Some people with hemophilia develop proteins in their blood that inactivate the clotting factors used to treat bleeding.

Signs and Symptoms

Signs and symptoms may include large or deep bruises, joint pain and swelling caused by internal bleeding, intramuscular bleeding, blood in the urine or stool, and prolonged bleeding from cuts or injuries, surgical procedures, or tooth extraction. During infancy, hemophilia appears to be asymptomatic

because of limited mobility. As the child begins to fall and bump into things, superficial bruises occur. Soft-tissue bleeding becomes more frequent as the child becomes more active. In most cases, the bumps and bruises do not require medical treatment. Signs and symptoms that would indicate an emergency situation include the following:

- Suspected bleeding into the head, neck, or digestive tract
- Sudden pain, swelling, and warmth of extremity muscles and large joints, such as the knees, elbows, hips, and shoulders
- Persistent bleeding from an injury, especially if the individual has a severe form of hemophilia

Management

Treatment of hemophilia varies with its severity. Referral to a physician is necessary to ensure proper medication, and if the bleeding is serious enough, potential repeated infusions of blood plasma may be necessary.

Reye Syndrome

Etiology

Reye syndrome is a rare but serious, acute disease that almost exclusively affects individuals between 2 and 16 years of age.[5] The condition tends to occur in previously healthy children and almost always follows an upper respiratory viral infection. In particular, the condition is associated with type B influenza and varicella (chickenpox), but it may develop after a common cold. The precise cause of the condition is unknown, but using aspirin to treat the viral illness or infection may trigger the condition in children by disrupting the body's urea cycle. The result is an accumulation of ammonia and acidity in the blood, while the level of sugar drops (hypoglycemia). At the same time, the liver may swell and develop fat deposits. Severe brain edema may lead to critically high intracranial pressure or convulsions and, eventually, to a coma and brain death.

Signs and Symptoms

Reye syndrome progresses quickly and can result in permanent liver damage, irreversible neurological damage, coma, and even death. The signs and symptoms typically begin approximately 1 week after a viral infection (e.g., influenza, chickenpox, or a cold). Following appearance of recovery from an illness, a child may become much more seriously ill. The condition typically progresses through five stages[5]:

- **Stage I.** Lethargy, vomiting, and hepatic dysfunction, followed by a few days of recovery
- **Stage II.** Hyperventilation, delirium, and hyperactive reflexes
- **Stage III.** Coma and rigidity of organ cortices
- **Stage IV.** Deepening coma, large and fixed pupils, and loss of cerebral functions
- **Stage V.** Seizures, loss of deep tendon reflexes, flaccidity, and respiratory arrest

Management

Suspected cases of Reye syndrome should be immediately referred to a physician for advanced care. Children often require hospitalization and intensive treatment to restore blood sugar levels, control cerebral edema, and correct acid-base imbalances.

Lymphangitis

Etiology

Lymph nodes or glands filter the lymph fluid. **Lymphangitis** is an inflammation of the lymphatic channels that occurs as a result of infection at a site distal to the channel. Pathogenic organisms invade the lymphatic vessels, either directly, through an abrasion, or wound, or as a complication of an infection, and then spread along these channels toward regional lymph nodes.

Signs and Symptoms

Once the organisms enter the channels, local inflammation and subsequent infection ensue, manifesting as red streaks that are visible along the course of the vessels. The inflammation or infection then extends proximally, toward regional lymph nodes. Individuals often complain of headache, loss of appetite, fever, chills, malaise, and muscle aches. The condition can progress rapidly, particularly when caused by group A streptococci, to bacteremia and disseminated infection and sepsis.

Management

Immediate referral to a physician is required, and hospitalization usually is necessary. If possible, the affected area should be elevated and immobilized to reduce swelling, pain, and spread of infection. Hot, moist compresses may help to reduce inflammation and pain. Prescribed antimicrobial and antibiotic agents should be administered, and analgesics or anti-inflammatory medications can be used to reduce pain, inflammation, and swelling. An abscess may require surgical drainage.

Anemia can reduce maximum aerobic capacity, decrease physical work capability at submaximal levels, increase lactic acidosis, increase fatigue, and decrease exercise time to exhaustion. Because the golfer's anemia was attributed to her eating habits, management of the condition should include a diet rich in iron content as well as dietary iron supplementation and ascorbic acid to enhance iron absorption.

SYNCOPE

Following 60 minutes of an intense, running workout on a treadmill, a healthy, 25-year-old man steps off the treadmill, suddenly collapses, and loses consciousness. What might explain this sudden unconsciousness, and how should this condition be managed?

Syncope is a sudden, transient loss of consciousness (T-LOC), described as "fainting," that often occurs in healthy individuals and is not associated with seizures, coma, shock, or other states of altered consciousness. **Presyncope** is a sense of impending LOC, light-headedness, or weakness. It appears more frequently than syncope and provides a better source of injury/illness history, because the patient usually has better recollection of the event.[6] Questions concerning syncope should be part of the medical history during the PPE. If a history of syncope is noted, the individual should be referred to a physician for further evaluation. Syncope and presyncope have three primary classifications (Box 24.3).[6]

BOX 24.3 Classification of Syncope

Reflex (Neurally Mediated) Syncope

Vasovagal

- Mediated by emotional distress: fear, pain, instrumentation, blood phobia
- Mediated by orthostatic stress

Situational

- Cough, sneeze
- Gastrointestinal stimulation (swallow, defecation, visceral pain)
- Micturition (post-micturition)
- Postexercise
- Postprandial
- Others (e.g., laugh, brass instrument playing, weight lifting)

Carotid sinus syncope

Atypical forms (without apparent triggers and/or atypical presentation)

Syncope Due to Orthostatic Hypotension

Primary autonomic failure

- Pure autonomic failure, multiple system atrophy, Parkinson disease with autonomic failure, Lewy body dementia

Secondary autonomic failure

- Diabetes, amyloidosis, uremia, spinal cord injuries

Drug-induced orthostatic hypotension:

- Alcohol, vasodilators, diuretics, phenothiazines, antidepressants

Volume depletion

- Hemorrhage, diarrhea, vomiting, etc.

Cardiac Syncope (Cardiovascular)

Arrhythmia as primary cause

Bradycardia

- Sinus node dysfunction (including bradycardia/tachycardia syndrome)
- Atrioventricular conduction system disease
- Implanted device malfunction

Tachycardia

- Supraventricular
- Ventricular (idiopathic, secondary to structural heart disease or to channelopathies)

Drug-induced bradycardia and tachyarrhythmia

Structural disease

Cardiac

- Cardiac valvular disease, acute myocardial infarction/ischemia, hypertrophic cardiomyopathy, cardiac masses (atrial myxoma, tumors, etc.), pericardial disease/tamponade, congenital anomalies of coronary arteries, prosthetic valves dysfunction

Others

- Pulmonary embolus, acute aortic dissection, pulmonary hypertension

Of the various conditions that can lead to neurally mediated reflex faints, the best known is the common or vasovagal faint. Other neurally mediated reflex faints include carotid sinus syndrome and situational faints such as those triggered by blood draws, emotional upset, pain, micturition, defecation, coughing, and swallowing.[7] The common orthostatic (postural) hypotensive faints are associated with movement from lying or sitting to a standing position. Cardiac arrhythmias may cause faints if the heart rate is excessively rapid or too slow, such as during the onset of a paroxysmal supraventricular tachycardia (SVT). Structural cardiopulmonary diseases are relatively infrequent causes of faints. A common cause in this category is fainting due to an acute myocardial infarction or ischemic event. Although quite rare, cerebrovascular disease may lead to fainting during a vertebrobasilar transient ischemic attack (TIA).[7]

Etiology

Decreased blood flow to the brain can occur because (1) the heart fails to pump the blood, (2) the blood vessels don't have enough tone to maintain blood pressure to deliver the blood to the brain, (3) there is not enough blood or fluid within the blood vessels, or (4) a combination of these reasons. The end result is a sudden drop in blood pressure that reduces blood circulation to the brain and leads to LOC. This condition is more common in children and young adults, although it can occur at any age.

See **Causes of Syncope and Near Syncope**, available on the companion Web site at thePoint.

Signs and Symptoms

Typical neurally mediated syncope occurs while standing and often is

preceded by prodromal symptoms, including restlessness, pallor, weakness, sighing, yawning, diaphoresis, and nausea. These symptoms may be followed by light-headedness, blurred vision, collapse, and LOC. Occasionally, mild clonic seizures occur, but unless other signs indicate, a detailed seizure evaluation is not indicated. Other signs and symptoms may include dizziness, profuse sweating, paresthesia of the hands or feet, and unilateral or bilateral chest pain. Some forms of syncope suggest a serious disorder, including those occurring with exercise, those associated with palpitations or irregularities of the heart, and those associated with a family history of recurrent syncope or sudden death.

Management

Neurally mediated syncope usually responds well to avoidance of stimuli that trigger the event. If syncope does occur, initial management includes assessment of the patient's vital signs. The patient should be placed in a safe, lying-down position. Recovery usually occurs within minutes. If the patient does not regain consciousness within a few minutes or demonstrates signs of breathing or cardiac impairment, the emergency action plan (EAP), including summoning emergency medical services (EMS), should be activated.

A thorough medical evaluation should be conducted in the following cases:

- Syncope recurs.
- Syncope occurs with exercise.
- Syncope is associated with palpitations or irregularities of the heart.
- Family history of syncope

In cases of severe syncope, β-blockers, cardiac pacing, and antidepressants may be recommended; however, the success rate is poor.

The exerciser may have experienced syncope as a result of dehydration or heat illness. The patient's vital signs should be measured and monitored. If he does not regain consciousness within a few minutes, the EAP, including summoning EMS, should be activated.

SHOCK

? Following a slide into home plate that resulted in a collision with the catcher, a baseball player sustains a compound fracture to the tibia. The athletic trainer activated the EAP, including summoning EMS. The athletic trainer also immobilized the leg. What possible complications might occur as a result of this traumatic injury? What other treatment should the athletic trainer provide while waiting for EMS to arrive?

Shock occurs with injuries involving severe pain, bleeding, the spinal cord, fractures, or intra-abdominal or intrathoracic regions, but shock also may occur, to some degree, with minor injuries. The severity of shock depends on one's age, physical condition, pain tolerance, fatigue, dehydration, presence of any disease, extreme cold or heat exposure, or handling of an injured area. Types of shock include anaphylactic, cardiogenic, hypovolemic, metabolic, neurogenic, respiratory, psychogenic, and septic (Box 24.4).

BOX 24.4 Types and Causes of Shock

- **Hypovolemic.** From excessive blood or fluid loss leading to inadequate circulation and oxygen supply to all body organs. Possible causes include hemorrhage, dehydration, multiple trauma, and severe burns.
- **Respiratory.** From insufficient oxygen in the blood as a result of inadequate breathing. Possible causes are spinal injury to the respiratory nerves, airway obstruction, or chest trauma, such as from a pneumothorax, hemothorax, or punctured lung.
- **Neurogenic.** Occurs when peripheral blood vessels dilate and an insufficient blood volume cannot supply oxygen to the vital organs. This may occur in a spinal or head injury when nerves that control the vascular system are impaired, thereby altering the integrity of blood vessels.

- **Psychogenic.** Refers to a temporary dilation of blood vessels, resulting in the draining of blood from the head with pooling of blood in the abdomen. A common example is an individual fainting from the sight of blood.
- **Cardiogenic.** Occurs when the heart muscle is no longer able to sustain enough pressure to pump blood through the system. Possible causes are injury to the heart or previous heart attack.
- **Metabolic.** Results from a severe loss of body fluids because of an untreated illness that alters the biochemical equilibrium. Possible causes are insulin shock, diabetic coma, vomiting, or diarrhea.
- **Septic.** Derives from severe, usually bacterial, infection whereby toxins attack the walls of small blood vessels, causing them to dilate, thereby decreasing blood pressure.
- **Anaphylactic.** Refers to a severe allergic reaction of the body to a foreign protein that is ingested, inhaled, or injected (e.g., foods, drugs, or insect stings).

Etiology

Shock occurs if the heart is unable to exert adequate pressure to circulate enough oxygenated blood to the vital organs. This condition may result from a damaged heart that fails to pump properly, low blood volume from blood loss or dehydration, or dilation of blood vessels that leads to blood pooling in larger vessels away from vital areas (Fig. 24.5). The result is a lack of oxygen and nutrition at the cellular level. The heart pumps faster, but because of reduced volume, the pulse rate is weakened and the blood pressure drops (i.e., **hypotension**). A rapid, weak pulse is the most prominent sign of shock. As an individual's condition deteriorates, breathing becomes rapid and shallow, and sweating is profuse. Vital body fluids pass through weakened capillaries, thereby causing further circulatory distress. If not corrected, circulatory collapse can lead to unconsciousness and even death.

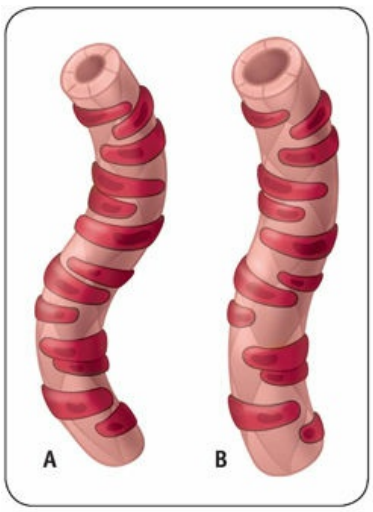


Figure 24.5. Shock. The diameter of an arteriole blood vessel is controlled by circular layers of smooth muscles that either constrict or relax to regulate peripheral blood flow **(A)**. Blood vessels dilate during shock. This action increases the size of the vascular bed and decreases the resistance to blood flow, resulting in blood pooling in larger vessels, depriving the brain and vital organs of needed oxygen. As a result, heart rate increases, giving the characteristic rapid, weak pulse that often is the first sign of shock **(B)**.

Signs and Symptoms

Signs and symptoms of shock can develop over time (Box 24.5). Initially, the individual may have a feeling of uneasiness or restlessness, increased respirations, and increased weakened heart rate. The skin turns pale and clammy, which usually is accompanied by profuse sweating. The lips, nail beds, and membranes of the mouth appear to be cyanotic. Thirst, weakness, nausea, and vomiting may then become apparent. During later stages, a rapid, weak pulse and labored, weakened respirations may lead to decreased blood pressure and possible unconsciousness.

BOX 24.5 Signs and Symptoms of Shock

- Restlessness, anxiety, fear, or disorientation
- Nausea and/or vomiting
- Cold, clammy, moist skin
- Shallow, irregular breathing; breathing also may be labored, rapid, or gasping
- Profuse sweating
- Extreme thirst
- Dizziness
- Eyes are dull, sunken, and with the pupils dilated

- Rapid and weak pulse
- Skin that is chalklike but that later may appear to be cyanotic

Management

The EAP, including summoning EMS, should be activated. While waiting for EMS to arrive, the clinician should maintain the airway, control any bleeding, splint any fractures, and maintain normal body temperature. If a head or neck injury is not suspected, the feet and legs should be elevated 8 to 12 in. Vital signs should be taken and recorded every 5 minutes. **Application Strategy 24.1** summarizes the immediate care of shock.

APPLICATION STRATEGY 24.1

Management of Shock

- Activate EAP, including summoning EMS. Secure and maintain an open airway. Control any major bleeding. Monitor vital signs.
- If a head or neck injury or a leg fracture is not suspected, elevate the feet and legs 8–12 in. With breathing difficulties, the patient might be more comfortable with the head and shoulders raised in a semireclining position. If a head injury is suspected, elevate the head and shoulders to reduce pressure on the brain. The feet also may be slightly elevated. In a suspected neck injury, keep the patient lying flat.
- A patient who vomits or is unconscious should be placed on one's side to avoid blocking the airway with any fluids. This allows the fluids to drain from the mouth.
- Splint any fractures. This reduces shock by slowing bleeding and helps to ease pain. If the patient has a leg fracture, keep the leg level while splinting the fracture. Raise the leg only after it has been properly immobilized.
- Maintain normal body temperature. This action may require removing any wet clothing, if possible, and covering the patient with a blanket. Keep the patient quiet and still. Avoid rough or excessive handling of

the patient.

- Do not give the patient anything by mouth.
- Monitor vital signs every 2–5 minutes until EMS arrives.

The compound fracture sustained by the baseball player can lead to shock. Shock is considered to be a medical emergency. As such, the EAP, including summoning EMS, should be activated. While waiting for EMS to arrive, the clinician should maintain the airway, maintain normal body temperature, and position the athlete lying down with the leg raised after—and only if—it has been properly immobilized. The management of shock is explained in Application Strategy 24.1.

BLOOD PRESSURE DISORDERS

A senior athlete reports feeling dizzy when getting up from a lying position and when suddenly changing body positions while playing tennis. What might this person be experiencing, and what is the management for this condition?

Blood pressure is the force per unit area exerted on the walls of an artery, generally considered to be the aorta. It is the result of two factors—namely, cardiac output and total peripheral resistance. Cardiac output is determined by heart rate, myocardial contractility (i.e., force of contraction), blood volume, and venous return. Peripheral resistance is determined by arteriolar constriction. As one of the most important vital signs, blood pressure reflects the effectiveness of the circulatory system.

Although blood pressure varies among individuals, normal blood pressure is considered to be 120 mm Hg systolic blood pressure and 80 mm Hg diastolic blood pressure. **Systolic blood pressure** (SBP) is measured when the left ventricle contracts and expels blood into the aorta. **Diastolic blood pressure** (DBP) is the residual pressure in the aorta between heartbeats. It is

not unusual for a relatively physically fit person to have a blood pressure of 90/70 mm Hg. Blood pressure may be affected by gender, weight, race, lifestyle, and diet, and it can vary throughout the day, depending on the time of day and the individual's fitness level. Blood pressure is measured in the brachial artery with a sphygmomanometer and stethoscope (refer to **Application Strategy 2.1**).

Any change in either cardiac output or peripheral resistance results in an increase or decrease in blood pressure. **Hypertension** is defined as a sustained, elevated blood pressure greater than 140 mm Hg SBP or greater than 90 mm Hg DBP. A fall of 20 mm Hg or more from a person's normal baseline SBP characterizes hypotension.

Hypertension

Hypertension affects approximately 50 million individuals in the United States and approximately 1 billion individuals worldwide.[8] The higher the blood pressure, the greater the chance of myocardial infarction, heart failure, stroke, and kidney disease. For individuals 40 to 70 years of age, each increment of 20 mm Hg in SBP or of 10 mm Hg in DBP doubles the risk of cardiovascular disease across the entire blood pressure range of 115/75 to 185/115 mm Hg.[8,9]

Several factors increase the risk of developing hypertension (**Table 24.1**). Onset generally is between the ages of 20 and 50 years, with the frequency being greatest in African Americans. Hypertension can be caused by a variety of substances, including the following:

- Certain prescribed medications
- Oral contraceptives
- Anabolic steroids
- Amphetamines
- Chronic alcohol use
- Nasal decongestants containing sympathomimetic amines
- Some nonsteroidal anti-inflammatory drugs

- Sleep apnea
- Chronic kidney disease
- Renovascular disease
- Coarctation of the aorta
- Thyroid or parathyroid disease

TABLE 24.1 Risk Factors for Developing Hypertension

RISK FACTOR	REASON
Age	Arteries lose their elasticity, and blood pressure increases with age. High incidence of hypertension after age 60 years
Diabetes	High incidence in those with diabetes is attributed to insulin resistance promoted by abdominal obesity.
Heredity	Higher incidence with a family history of hypertension and heart disease in women (<65 years) and men (<55 years)
High blood	High blood lipids contribute to atherosclerosis and hypertension. A high-fat diet also contributes to lipids hypertension.
Obesity	Excess body fat, especially abdominal fat, is closely associated with hypertension.
Race	Prevalence differs among racial and ethnic groups, but incidence in African Americans is among the highest in the world.
Sex	Higher incidence in men and postmenopausal women
Smoking	Smoking increases the workload of the heart and, as such, increases blood pressure.

Hypertension is classified into prehypertension, stage 1 hypertension, and stage 2 hypertension (Table 24.2). This categorization applies to adults 18 years of age and older who are not taking antihypertensive medication and are not acutely ill. When SBP and DBP fall into different categories, the higher category is used to classify the individual's blood pressure status. For more detailed standards in children and adolescents, refer to Chapter 2.

TABLE 24.2 Classification of Blood Pressure in Adults

CLASSIFICATION	SYSTOLIC (mm Hg)[a]		DIASTOLIC (mm Hg)[a]	LIFESTYLE MODIFICATION
Normal	<120	and	<80	Encourage
Prehypertension	120–139	or	80–89	Yes
Stage 1 hypertension	140–159	or	90–99	Yes
Stage 2 hypertension	≥160	or	≥100	Yes

[a]Treatment determined by highest category.

From James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311(5):507–520. http://jama.jamanetwork.com/article.aspx?articleid=1791497. Accessed July 1, 2015.

Categories of Hypertension

Classifications of hypertension are based on the mean of two or more properly measured, seated blood pressure readings on each of two or more occasions.

In contrast to past standards, new standards classify a designated prehypertension category to identify patients who are at increased risk for progression to hypertension. Individuals in the 130/80 to 139/89 mm Hg range are at twice the risk of developing hypertension compared with those who have lower values.[8]

Hypertension is divided into two categories. Primary, or essential, hypertension is a chronic, progressive disorder with no identifiable cause that often attacks the heart, brain, kidneys, and eyes and that is associated with increased morbidity and mortality. The condition can be treated successfully with medication, diet modification, and exercise. Secondary hypertension has an identified cause, which often is associated with chronic renal disease, renovascular disease, **coarctation**, and other conditions. If the cause is identified, secondary hypertension can be controlled.

Clearance for Sport and Physical Activity Participation

An individual who has mild or moderate hypertension should not participate in competitive sports and physical activity until cleared by a physician. These individuals often are given physician clearance to participate in activity if their blood pressure is well controlled and no target organ damage or heart disease is present. These individuals should have their blood pressure rechecked by the athletic trainer on a weekly basis and by the physician every 2 to 4 months. For individuals with stage 2 hypertension, physical activity is restricted, especially in sports or activities with a high static component (e.g., wrestling, gymnastics, weight lifting, rock climbing, and rowing) until the hypertension is well controlled.

Management of Hypertension

Treatment of hypertension focuses on reducing SBP and DBP as well as on preventing long-term complications. Target blood pressure readings of less than 140/90 mm Hg are associated with a decrease in cardiovascular complications. The target for a patient with hypertension along with diabetes or renal disease is less than 130/80 mm Hg.[8] Nonpharmaceutical treatment includes lifestyle modifications and aerobic exercise. Pharmaceutical

treatment may involve antihypertensive medications.

Lifestyle Modifications

Because hypertension has a significant impact on risk for cardiovascular disease, individuals with hypertension should be encouraged to establish lifestyle modifications in collaboration with an exercise program. These steps may include measures such as the following[8,10]:

- Lose weight if overweight or obese (body mass index 18.5 to 24.9 kg per m^2).
- Engage in regular aerobic physical activity, such as brisk walking.
- Limit alcohol intake. For most males, alcohol intake should be no more than two drinks per day (<1 oz per day of ethanol; e.g., 24 oz of beer, 10 oz of wine, or 3 oz of 80-proof whiskey); females and lighter weight males should not have more than one drink per day.
- Avoid consuming sugar-containing beverages and energy-dense snacks.
- Reduce sodium intake to less than 2,300 mg per day.
- Consume a diet rich in fruits, vegetables, and low-fat dairy products, with a reduced content of saturated and total fats.
- Maintain adequate dietary potassium, calcium, and magnesium intake.
- Do not smoke cigarettes.

Diet modifications should include a diet low in saturated fats, with total dietary fat not exceeding 30% of total caloric intake, and increased potassium, calcium, and magnesium intake. In severe cases of hypertension, more significant sodium and alcohol restrictions, weight loss, and reduction of other cardiovascular risk factors must be incorporated into the program.

Exercise Program

Aerobic exercise has a positive effect on the blood pressure of individuals with hypertension by lowering resting SBP an average of 4 to 9 mm Hg and

DBP an average of 6 mm Hg. Specific mechanisms for this response are still unknown, but significant contributing factors may include the following[8]:

- Reduced activity of the sympathetic nervous system, which decreases peripheral resistance and blood flow
- Altered renal function, which facilitates the elimination of sodium by the kidneys, subsequently reducing fluid volume and blood pressure
- Decreased body fat
- Decreased smoking and alcohol consumption that often accompany exercise conditioning
- Increased relaxation during exercise

According to the U.S. Department of Health and Human Services, adults who wish to attain substantial health benefits should perform aerobic exercise a minimum of 150 minutes (2 hours and 30 minutes) per week at a moderate intensity, such as brisk walking, or 75 minutes (1 hour and 15 minutes) a week of vigorous-intensity aerobic physical activity, or an equivalent combination of moderate- and vigorous-intensity aerobic activity. Aerobic activity should be performed in episodes of at least 10 minutes, and preferably, be spread throughout the week. Children and adolescents should do 60 minutes (1 hour) or more of physical activity daily including primarily moderate- or vigorous-intensity at least 3 days a week. The sessions should include primarily moderate or vigorous aerobic physical activity, muscle strengthening, and bone strengthening.[11] Individuals with hypertension should avoid isometric exercises and heavy resistance training.

Pharmaceutical Medications

When lifestyle modifications, diet, and physical activity fail to control hypertension, diuretics and antihypertensive agents often are prescribed. Most patients require two or more antihypertensive medications to achieve a desirable blood pressure. Thiazide-type diuretics, which are used in the initial therapy for most patients, enhance the antihypertensive efficacy of multidrug

regimens, can be useful in achieving blood pressure control, and are more affordable than other antihypertensive agents. Diuretics lower blood pressure by increasing fluid loss; however, some diuretics can lead to a potassium deficiency. As such, individuals should be aware of the signs of potassium imbalance, including weakness (particularly of the legs), unexplained numbness or tingling sensations, cramps, irregular heartbeats, and excessive thirst and urination.[8]

Hypotension

When arterial blood pressure is lower than normal, inadequate blood is circulated to the heart, brain, and other vital organs. The result often is a collapse of bodily functions. Hypotension, or low blood pressure, is defined as a decrease of 20 mm Hg or more in an individual's normal SBP. It can be caused by a variety of factors, including shock, acute hemorrhage, dehydration, orthostatic hypotension, **postural hypotension**, or overtreatment of hypertension (Box 24.6).

BOX 24.6 Nonneurogenic Causes of Hypotension

- Shock as a response to stress or trauma
- Heat (e.g., hot environments, hot showers and baths, or fever)
- Hemorrhage
- Drug toxicity (e.g., alcohol, anesthesia, diuretics, analgesics, or vasodilators)
- Diabetes mellitus
- Overtreatment of hypertension (e.g., diuretics, antihypertensives, or vasodilators)
- Allergic drug reaction
- Dehydration
- Orthostatic or postural hypotension
- Low-salt diets

- Straining on heavy lifting, urination, or defecation
- Diarrhea
- Vasovagal syncope (fainting)

Orthostatic hypotension is not unusual. Estimated to occur in approximately half of all elderly people, it can lead to fainting and falls. In the absence of symptoms, however, treatment is not required. Symptoms that indicate a medical referral include dimming or loss of vision, light-headedness, dizziness, excessive perspiration, diminished hearing, pallor, nausea, and weakness. It should be noted that certain medications (e.g., vasodilators and antidepressants) may cause orthostatic hypotension. This complication can be addressed by having a physician change the medication or dosage.

Physically active people do not usually need to be concerned about hypotension. In general, the lower the blood pressure, the better—as long as the person feels well. Older individuals, however, should always check with their physician before performing heavy resistance exercises. Nonpharmaceutical steps that can be taken to reduce the effects of orthostatic hypotension include the following:

- Avoid prolonged standing, vigorous exercise, alcohol, hot environments, hot showers or baths.
- Execute slow, careful changes in position, especially when arising in the morning.
- Eat multiple, small meals.
- Schedule physical activities in the afternoon.
- Increase salt and fluid intake.

The senior athlete most likely has experienced orthostatic hypotension. Management for this condition can include avoiding prolonged standing, vigorous exercise, alcohol, and hot environments; slowly and carefully changing body positions; eating multiple, small meals;

scheduling physical activities in the afternoon; and increasing salt and fluid intake.

SUDDEN CARDIAC DEATH

? A Little League Baseball pitcher receives a line drive to his chest. He immediately collapses. What condition should be suspected, and how should this condition be managed?

Sudden cardiac death (SCD) has been termed the "silent killer." It is defined as an unexpected death resulting from sudden cardiac arrest within 6 hours of an otherwise normal, healthy clinical state.[12]

Epidemiology

The prevalence of SCD during physical activity is rare, yet it is the leading cause of death in young athletes. It is estimated that sudden death in athletes occurs approximately once every 3 days in the United States.[13–15] Males have an increased death rate from SCD than that of females, with the highest rates being seen in basketball and football players.[16] Explanation for the low occurrence in female athletes is inconclusive; however, some researchers postulate that the decreased incidence can be attributed to the following[17,18]:

- Fewer females participate in sports.
- Fewer females participate in highly intense sports that require full-body protective equipment (e.g., football or ice hockey).
- Gender differences exist regarding cardiac adaptation to training demands.
- Females have smaller hearts.

SCD often is precipitated by physical activity and may be caused by an array of cardiovascular conditions. The age of the individual appears to dictate the underlying physiological pathology for the occurrence of SCD. The most

common causes of SCD in athletes younger than 35 years of age in the United States are hypertrophic cardiomyopathy, commotio cordis, and anomalous coronary arteries.[16,18] Noncardiac causes, a small percentage of sudden death in athletes, may be due to death from heat stroke, asthma, cerebral artery rupture, and exertional rhabdomyolysis secondary to sickle cell trait.[16,18] Atherosclerotic coronary artery disease, such as myocardial ischemia and myocardial infarction, is the leading cause for individuals older than 35 years.

Cardiac Causes of Sudden Cardiac Death

Cardiac anomalies are the most direct cause of SCD in individuals younger than 35 years, with hypertrophic cardiomyopathy being the most common. Other reported cardiac-related causes include cardiac mass, mitral valve prolapse, myocarditis, acquired valvular heart disease, coronary artery anomalies, ruptured aortic aneurysm (often from intrinsic aortic weakness with Marfan syndrome), Wolff-Parkinson-White syndrome, and arrhythmogenic right ventricular dysplasia. Other rare cardiac conditions that can contribute to sudden death involve abnormalities of the cardiac conduction system (i.e., long QT syndrome), which can result in lethal cardiac rhythm problems.

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM), characterized by an abnormal thickening of the left ventricle wall, is the most common inherited cardiac disorder, occurring in 1:500 of the general population.[19] The condition typically goes undetected during routine physical examination. By definition, HCM is a hypertrophied, nondilated left ventricle in the absence of another cardiac or systemic disease capable of producing the degree of hypertrophy that is present. A normal left ventricle is approximately 1 cm thick. In HCM, the wall thickness typically ranges from 2 to 4 cm—and can be greater than 15 cm.[20] This abnormal thickness can lead to electrical problems and abnormal rhythms, including ventricular fibrillation and lethal rhythm.

Physical examinations should include a thorough cardiac history and cardiac examination (see “Cardiovascular Preparticipation Screening”

section). Unfortunately, HCM often goes undetected during routine physical examinations. HCM should be suspected in a young athlete who presents with exertional dyspnea, chest pain, unexplained syncope, or prior recognition of a heart murmur. A family history of HCM, SCD, heart disease in a close relative younger than 50 years of age, or unexplained syncope should also raise red flags during the assessment of the patient. During the physical examination, the most important finding is a systolic murmur increased/elicited by provocative maneuvers such as Valsalva, standing, or exertion.[19] As a result, periods of arrhythmia or blood flow obstruction may produce syncope during physical exertion. Any individual exhibiting these signs or symptoms should be seen immediately by a physician or a cardiologist.

Mitral Valve Prolapse

Mitral valve prolapse (MVP) is not a frequent cause of sudden death but can affect a small portion of the population, spanning all ages.[21] MVP is a condition in which redundant tissue is found on one or both leaflets of the mitral valve. During a ventricular contraction, a portion of the redundant tissue on the mitral valve pushes back beyond the normal limit and, as a result, produces an abnormal sound. This sound is followed by a systolic murmur as blood is regurgitated back through the mitral valve into the left atrium. Because of the characteristic sound, this condition often is referred to as a "click-murmur syndrome." Individuals with MVP usually experience some degree of chest pain, dyspnea, palpitations, and fatigue with exertion.[21] Athletes with MVP can engage in all competitive sports unless any of the following features is present, in which case the individual should participate in only low-intensity competitive sports[22]:

- Prior syncope judged to be arrhythmogenic in origin
- Sustained or repetitive and nonsustained SVT or frequent and/or complex ventricular tachyarrhythmias on ambulatory monitoring
- Severe mitral regurgitation
- Left ventricular (LV) systolic dysfunction

- Prior embolic event
- Family history of MVP-related sudden death

Myocarditis

Myocarditis is an inflammatory condition of the muscular walls of the heart that can result from a bacterial or viral infection. The condition is characterized by the infiltration of inflammatory cells into the myocardium, leading to an abnormally enlarged left ventricle. SCD occurs when the inflammatory changes in the myocardium lead to degeneration or death of adjacent muscle cells, resulting in electrical instability and life-threatening arrhythmias. Although some individuals may be asymptomatic, others exhibit symptoms commonly associated with viral infections, including fever, body aches, fatigue, cough, or vomiting; this similarity often impedes establishing the diagnosis. Exercise intolerance, shortness of breath, and more serious cardiac symptoms, including **palpitations** or syncope, may occur without warning.

Any sport participant with probable or definitive evidence of myocarditis should be withdrawn from all competitive sports and undergo a thorough medical examination, which may lead to 6 months of total rest following the onset of clinical manifestations. Return to training and competition may occur after the following[22]:

- LV function, wall motion, and cardiac dimensions return to normal.
- Clinically relevant arrhythmias are absent on ambulatory monitoring and graded exercise testing.
- Serum markers of inflammation and heart failure have normalized.
- The 12-lead electrocardiogram (ECG) has normalized.

Acquired Valvular Heart Disease

Acquired valvular heart disease stems from a defect or insufficiency in a heart valve that can lead to improper blood flow through the heart. The condition is manifested as either **valvular stenosis**, which is a narrowing of the orifice

around the cardiac valves, or **regurgitation**, which involves a backward flow of blood. The condition is named according to the affected valve (e.g., mitral valve, aortic valve, and tricuspid valve). If more than one valve is affected, the condition is called **multivalvular disease**, which occurs in the context of rheumatic heart disease. The characteristic murmurs associated with this condition are detected during a physical examination.

Individuals with mild-to-moderate mitral and tricuspid valve stenosis or regurgitation typically can participate in sports and physical activity. Each case, however, must be evaluated on an individual basis.

Initially, experts posited that aortic stenosis was a common cause of sudden death; however, new research has not supported their theory. Aortic stenosis, like other valve conditions, often can be detected early through physical examination. Individuals with mild aortic stenosis may participate in physical activity and competitive sports. Individuals with mild or moderate asymptomatic aortic stenosis and a history of **supraventricular tachycardia** (i.e., rapid heartbeats proximal to the ventricles in the atrium or atrioventricular node) or ventricular arrhythmias at rest should participate only in low-intensity physical activity and sports. Individuals with severe aortic stenosis or aortic regurgitation and significant dilation of the proximal ascending aorta associated with Marfan syndrome should not engage in any competitive sport.[22]

Coronary Artery Disease

The most common cause of sudden death in individuals older than 35 years is coronary artery disease (CAD), also referred to as **atherosclerosis**. An excessive buildup of cholesterol within the coronary arteries narrows the diameter of the arteries and impedes the flow of blood, which in turn reduces the amount of oxygen supplied to the heart. Subsequent to the diminished oxygen, **angina** is a common symptom. If excessive cholesterol buildup blocks a coronary artery, the person is at risk for a **myocardial infarction**. If the blockage is in a major coronary artery, death often occurs.

Unlike young individuals, who rarely know if they have a cardiac anomaly that may put them at risk for sudden death, physically active individuals older

than 35 years usually have experienced prodromal cardiovascular symptoms or have a known medical history of CAD. Therefore, individuals older than 35 years can choose to ignore the symptoms and continue participating at the same level, placing them at risk for sudden death; change their lifestyle according to CAD recommendations and continue to participate in limited activity; or stop physical activity.

The American College of Sports Medicine (ACSM) has developed a list of risk factors for CAD and recommends an exercise ECG before beginning a moderate or vigorous exercise program for anyone who has had a history of angina, palpitations, syncope, or dyspnea during physical activity (**Table 24.3**).[23] For individuals with CAD, regular and recreational physical activity and moderate-intensity exercise training are recommended.[22]

TABLE 24.3 American College of Sports Medicine Coronary Artery Disease Risk Factors

POSITIVE RISK FACTORS	DEFINING CRITERIA
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 first-degree female relative (i.e., sister or daughter)
Cigarette smoking	Current cigarette smoker or someone who quit within the previous 6 months
Hypertension	Systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg confirmed by measurements on at least two separate occasions, or using antihypertensive medication
Hypercholesterolemia	Total serum cholesterol >200 mg/dL (5.2 mmol/L) or high-density lipoprotein cholesterol <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 >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
Obesity	Body mass index ≥30 kg/m^2, or waist girth >100 cm
Sedentary lifestyle	Persons making up the least active 25% of the population, as defined by the combination of a sedentary job involving sitting for a large part of the day and no regular exercise or active recreational pursuits
NEGATIVE RISK FACTOR	**DEFINING CRITERIA**
High serum high-density	>60 mg/dL (1.6 mmol/L) lipoprotein cholesterol

From American College of Sports Medicine. *ACSM's Guidelines for Exercise Testing and Prescription.* 7th ed. Baltimore, MD: Lippincott Williams & Wilkins; 2010; with permission.

Marfan Syndrome

Marfan syndrome does not necessarily lead to SCD. When SCD does occur, it usually is caused by the condition's hallmark characteristic—namely, a weakened aorta. Marfan syndrome is a genetic disorder of the connective tissue that can affect the skeleton, lungs, eyes, heart, and blood vessels. A single mutant gene is linked to the condition. Although usually inherited, 25% of all cases have no family history.[24]

Individuals with Marfan syndrome are tall in stature, with overly long extremities; the arm span exceeds the person's height. Joints usually are hypermobile, and the person may have a pigeon (sunken) chest, stretch marks, scoliosis, and increased incidence of hernias. A positive thumb test and wrist test are classic signs of Marfan syndrome. The thumb test involves adduction of the thumb across the palm of the hand and flexion of the fingers around the thumb; the test is positive if the thumb extends past the fifth finger. The wrist test involves the person encircling a wrist with the thumb and fifth finger of the opposite hand; the test is positive if the thumb and fifth finger overlap. In addition to orthopedic anomalies, it is not unusual to find an excessively high palate, eye defects (i.e., myopia or nearsightedness), MVP, and defects in the connective tissue layers of the aorta. As a result of the defect in the aorta tissues, death from Marfan syndrome usually is associated with an aortic dissection or rupture.

Screening for Marfan syndrome includes a musculoskeletal and eye examination as well as an echocardiogram to determine abnormalities of the aorta. Marfan syndrome has no cure, but with careful medical management, most people can live a normal life. An individual with Marfan syndrome should avoid contact activities because of the risk of injury to the eyes and aorta. Individuals can participate in low and moderate static/low dynamic competitive sports, such as brisk walking, leisure bicycling, golf, slow jogging, and slow-paced tennis if they do not have aortic root dilation, moderate-to-severe mitral regurgitation, or a family history of dissection causing sudden death in a Marfan relative.[22]

See **Screening for Marfan Syndrome**, available on the companion Web site at thePoint.

Rare Cardiac Conditions

Other rare cardiac conditions, such as a long QT syndrome and right ventricular dysplasia, may contribute to SCD. Both conditions produce serious arrhythmias. Long QT syndrome, which usually affects children and young adults, is a hereditary disorder of the heart's electrical system. The individual

is susceptible to arrhythmia, which produces inefficient contraction of the heart. The result is the reduction of normal blood volume in the body and the brain. Inability of the heart to regain its normal rhythm can lead to ventricular fibrillation, which is a fatal arrhythmia.

Right ventricular dysplasia, a disorder of the right ventricle, is characterized by the formation of adipose or fibrous tissue extending from the epicardium to the endocardium. This abnormal growth of tissue increases the risk of ventricular fibrillation. Any unstable ventricular heart rhythm, such as ventricular tachycardia or ventricular fibrillation, can lead to death. In fact, most cardiac conditions that produce SCD are the result of an abnormal ventricular rhythm.[21,22]

Wolff-Parkinson-White syndrome is an abnormality of cardiac rhythm that manifests as an SVT. The condition is associated with an accessory electrical pathway in the heart proximal to the ventricles that can spontaneously produce episodes of rapid twitching of the atrium muscle fibers within a range of 200 to 300 heartbeats per minute. The condition often is seen in asymptomatic, healthy individuals during electrocardiographic examination. Although rarely associated with sudden death, the condition may complicate other heart conditions, such as myocarditis or ischemic heart disease.

Congenital coronary artery anomalies are another cause of sudden death in young athletes. The most common anomaly is an abnormal origin of the left coronary artery. The anomaly necessitates an acute takeoff angle, forcing the artery to pass between the aorta and the pulmonary artery, both of which can decrease blood flow to the heart. Other anomalies may involve an abnormal origin of the right coronary artery, presence of a right coronary artery without a left coronary artery, and abnormal coronary artery spasm. Congenital coronary artery anomalies may not be detected during exercise because symptoms, such as syncope, near syncope, and chest pain, are intermittent and unpredictable.

Noncardiac Causes of Sudden Death

SCD usually is directly related to cardiac conditions. Noncardiac conditions, however, also can lead to SCD; these include commotio cordis, substance

abuse, head injuries, heat illness, exertional hyperthermia, exercise-induced anaphylaxis, exertional rhabdomyolysis, and sickle cell trait. Often, noncardiac conditions are more easily identifiable through physical examination and field evaluation. As such, if a condition can be identified, it usually can be treated.

Commotio Cordis

Cardiac arrest from a low-impact, blunt blow to the chest in the absence of a structural cardiovascular disease is rare in sports. According to the U.S. Commotio Cordis Registry, since 1995, 188 athletes have died from blunt force injury to the heart (commotio cordis). The mean age was 14.7 years, and 96% were males.[25] The leading mechanism of injury was from a projectile striking the chest, including baseballs, softballs, hockey pucks, and lacrosse balls. Other less frequent projectiles were a soccer ball and a cricket ball.[26] Also, many deaths occur around the home or on the playground in informal activities related or unrelated to recreational sports (often involving close relatives) in which the chest impact is delivered in an innocent fashion (e.g., light blows during playful "shadow boxing").[22] Postmortem analyses of individuals dying from commotio cordis often reveal no structural damage to the heart or overlying protective structures (e.g., sternum or ribs), but soft-tissue contusions of the left chest wall are commonly visualized.[27] This suggests that sudden death in these cases resulted from blunt force–induced conduction abnormalities.

Upon impact, collapse may be instantaneous or preceded by brief periods of consciousness and physical activity. Resuscitation efforts were previously rarely successful; however, resuscitation has been proven to be more successful (35%).[28,29] Because commotio cordis usually is a fatal event, focus has been aimed at prevention. Possible prevention strategies include the use of protective padded equipment that covers the area of the chest over the heart, switching to age-appropriate safety balls or pucks made of softer materials, and encouraging all coaches and officials to become trained in cardiopulmonary resuscitation (CPR), automated external defibrillator (AED) use, and first aid.

Substance Abuse

Many nutritional supplements and drugs are marketed to improve exercise duration or physical strength, to shorten recovery time from exertion, to reduce fat, or to enhance athletic performance in other ways. Certain types of drug abuse can lead to cardiac changes that predispose an individual to SCD. Amphetamines are central nervous system stimulants that increase heart rate, respiration rate, and blood pressure. Cocaine, which is an anesthetic, constricts coronary arteries and has been known to lead to myocardial infarction in those with and in those without CAD. In addition, myocarditis has been found in autopsy reports of cocaine-related SCD cases. It must be noted that a massive dose of cocaine is not required to produce cardiac or respiratory consequences. Several reported cases of SCD have involved anabolic steroid users, although a direct relationship between steroid use and sudden death has not yet been established.

Produced in the kidneys, **erythropoietin** is a hormone that stimulates bone marrow to increase production of RBCs. Erythropoietin (EPO/EPOGEN) became synthetically available in the late 1980s when its use as an ergogenic aid for endurance athletes was first documented. The use of EPO is equal to blood doping and, like blood doping, can increase blood volume and viscosity of the blood, leading to decreased circulation, thrombosis, and myocardial infarction. These adverse cardiovascular effects have led to SCD; EPO may be one of the most deadly ergogenic aids available.[30]

Head Injuries

Catastrophic brain injuries that can lead to SCD include skull fracture, epidural hematoma, acute and subacute subdural hematoma, second-impact syndrome, or various other brain hemorrhage conditions. Individuals should be observed closely after sustaining any type of head injury, regardless of the level of consciousness. Signs of rapidly increasing intracranial pressure include a dilated or irregular pupil or pupils, reduced pulse, nausea or vomiting, dyspnea, **photophobia**, mood swings, muscle weakness, and decreased level of consciousness. **Decortication**, a condition that presents as extension of the legs with flexion of the elbows, wrists, and fingers, or

decerebration, a condition that presents as extension of all four extremities, often is present in severe injuries (see Chapter 7). An individual who is unable to remember events leading up to (i.e., **retrograde amnesia**) or following the injury (i.e., **anterograde amnesia**) should not be permitted to continue activity and should be referred immediately to a physician. Additional information concerning head injuries can be found in Chapter 20.

Heat Illness

Heat illness is one of the most preventable causes of SCD, yet individuals die from exertional hyperthermia each year. In football, heat stroke is second only to head injuries as the most frequent cause of death. The condition also is seen in distance runners and wrestlers who are dehydrated through weight loss. Heat stroke almost always is preceded by prolonged, strenuous physical exercise in individuals who are poorly acclimatized or in situations during which evaporation of sweat is inhibited.

The individual often complains of a feeling of "burning up." Deep breaths, irritability, hysterical behavior, and an unsteady gait may be present. As the condition deteriorates, the skin is hot and dry and appears to be red or flushed, and the pulse becomes rapid and strong (as high as 150 to 170 heartbeats per minute). Brain tissue damage by excessive body heat leads to vasomotor collapse, shallow breathing, decreased blood pressure, and a rapid and weak pulse. Muscle twitching or seizures may occur just before the individual lapses into a coma. This condition is a medical emergency and, as such, warrants activation of the EAP, including summoning EMS. Further information concerning heat illness is presented in Chapter 29.

Sickle Cell Trait

Sickle cell trait is an inherited disorder in which RBCs tend to form into a sickle-shaped structure (**Fig. 24.4**). This sickling formation of RBCs prevents efficient transportation of oxygen to the tissues and can lead to vascular occlusion, coagulation, and even death. Eight percent of all African Americans have sickle cell trait, and individuals in the 23- to 30-year-old age range have a 1.3 in 1,000 chance of dying from sudden death during exertional activities,

particularly during extreme conditions of heat, humidity, and increased altitude.[31] Unexpected death is almost always associated with severe exertional **rhabdomyolysis**, a fatal disease stemming from renal failure caused when cellular contents of damaged skeletal muscle (myoglobin) enter the circulation. Sickle cell anemia is easily screened through laboratory testing. As such, recommendations can be made to ensure that individuals with sickle cell anemia can safely participate in sport and physical activity.

Following the collapse of the Little League Baseball, an immediate assessment of airway, breathing, and circulation (ABCs) is essential. If the athlete is not breathing or does not have a pulse, the EAP, including summoning EMS, should be activated and CPR administered. The player may have experienced cardiac arrest from the blow to the chest. The condition, known as commotio cordis, results in conduction abnormalities. Because it usually is fatal, focus has been on prevention rather than treatment.

CARDIOVASCULAR PREPARTICIPATION SCREENING

In preparation for PPEs of high school and college athletes, several areas must be addressed, including obtaining a cardiovascular history. What questions should be asked as part of the medical history to assist in screening for those at risk of sudden death?

Identification of the risks associated with SCD requires a two-tiered approach. First, a standard screening examination should be done 6 to 8 weeks before engaging in physical activity or sport and be performed by a health care provider with the medical skills, requisite training, and clinical experience to obtain a detailed cardiovascular history, to perform an extensive physical examination, and to recognize potential heart disease or cardiac anomalies. In an attempt to improve the screening and identification of individuals at

potential risk of SCD, the American Heart Association (AHA) recommends 12 items (8 for personal and family history and 4 for physical examination) be investigated (Box 24.7). At the discretion of the primary care physician, a positive response or finding in any 1 or more of the 12 items may be judged sufficient cause to refer the individual for cardiovascular evaluation. Parental verification is recommended for high school and middle school age children.

BOX 24.7 The 12-Element American Heart Association Recommendations for Preparticipation Cardiovascular Screening of Competitive Athletes

Medical History[a]

Personal history

1. Exertional chest pain/discomfort
2. Unexplained syncope/near syncope[b]
3. Excessive exertional and unexplained dyspnea/fatigue associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure

Family history

6. Premature death (sudden and unexpected, or otherwise) before age 50 years due to heart disease in at least one relative
7. Disability from heart disease in a close relative younger than 50 years of age
8. Specific knowledge of certain cardiac conditions in family members: hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome, or clinically important

arrhythmias

Physical examination

9. Heart murmur[c]
10. Femoral pulses to exclude aortic coarctation
11. Physical stigmata of Marfan syndrome
12. Brachial artery blood pressure (sitting position)[d]

[a]Parental verification is recommended for high school and middle school athletes.

[b]Judged not to be neurocardiogenic (vasovagal); of particular concern when related to exertion.

[c]Auscultation should be performed in both supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction.

[d]Preferably taken in both arms.

From American Heart Association. Preparticipation cardiovascular screening of young competitive athletes: policy guidance (June 2012). https://www.heart.org/idc/groups/ahaecc-public/@wcm/@adv/documents/downloadable/ucm_443945.pdf.

Although these guidelines present a somewhat standard approach for reducing the incidence of SCD, no universal standard for screening high school athletes exists. Most preparticipation standards are set by state legislation, state athletic associations, or individual school districts, and most often consist of a medical history review and basic physical examination.[32] Identification of a possible cardiovascular abnormality during the cardiac history or standard physical examination is the first tier of recognition. Many of these conditions were listed in **Box 2.3**. The second tier involves referral to a cardiologist for more extensive screening, including an echocardiogram.

See **Diagnostic Testing for Various Causes of Sudden Cardiac Death**, available on the companion Web site at thePoint.

The decision regarding whether to permit an individual to participate after identifying a cardiovascular abnormality must be resolved on an individual

basis under the Americans with Disabilities Act of 1990, the Rehabilitation Act of 1973, and similar state statutes prohibiting unjustified discrimination against the physically impaired. These laws permit an individual with the physical capabilities and skills to participate in a sport despite the fact that a cardiovascular abnormality is present. Exclusion from participation must be based on reasonable medical judgments, given the state of scientific research on the specific condition. These laws require careful balancing of the individual's right to participate, the physician's evaluation of the medical risks of participation, and an organization's interest in conducting a safe athletic program.

Many health care professionals, including members of the National Athletic Trainers' Association, believe that emergency preparedness and management of sudden cardiac arrest are a priority for every sport venue, including high school and college athletic programs.[33] As part of a well-defined EAP, every facility should establish catastrophic incident guidelines that address the immediate action plan, chain of command responsibilities, standards for documentation of the event, and long-term support for individuals who may be affected by SCD. This action plan should include CPR and AED training for all targeted first responders, and it recommends that access to early defibrillation is essential within 3 to 5 minutes from the time of the collapse to the first shock. The review of equipment readiness and the EAP by on-site personnel for each athletic event is desirable, as is the annual review of the EAP.[33] It is critical to have an EAP in place to help any facility or sport program work through an emotional and highly visible situation.

The medical history for cardiovascular conditions should include questions regarding previous chest pain during physical exertion; exercise-induced syncope or near syncope; excessive, unexplained shortness of breath, fatigue, and dizziness with exercise; history of heart murmur; history of hypertension; family history of death from cardiovascular disease in a relative younger than 50 years; and family history of HCM, dilated cardiomyopathy, long QT syndrome, or Marfan syndrome.

SUMMARY

1. Anemia is a reduction in either the RBC volume (hematocrit) or hemoglobin concentration. Anemia reduces maximum aerobic capacity, decreases physical work capability at submaximal levels, increases lactic acidosis, increases fatigue, and decreases exercise time to exhaustion.
2. Hemophilia occurs in three types—namely, A, B, and C. These types are distinguished by the clotting factor that is deficient. All three types can cause prolonged bleeding.
3. Reye syndrome is a rare but serious, acute illness that disrupts the body's urea cycle, resulting in the accumulation of ammonia in the blood, hypoglycemia, severe brain edema, and critically high intracranial pressure. The disease almost exclusively affects individuals from 2 to 16 years of age. It is linked to the use of aspirin to treat a viral infection.
4. Syncope is a transient LOC, described as "fainting," that often occurs in healthy individuals.
5. Shock occurs when the heart is unable to exert adequate pressure to circulate enough oxygenated blood to vital organs.
6. A rapid and weak pulse is the most prominent sign of shock, but other signs include rapid and shallow breathing, profuse sweating, and cold, clammy skin.
7. Reflex (neurally mediated) syncope is the most common type of syncope that produces fainting and results from a sudden drop in blood pressure, which reduces blood circulation to the brain, leading to an LOC.
8. Blood pressure is the force per unit area exerted on the walls of an artery, generally considered to be the aorta. It is the result of two factors—namely, cardiac output and total peripheral resistance.
9. Blood pressure varies among individuals, but normal is considered to be 120 mm Hg SBP over 80 mm Hg DBP. Hypertension is defined as a sustained, elevated blood pressure of greater than 140 mm Hg SBP or

greater than 90 mm Hg DBP. Hypotension is characterized by a fall of 20 mm Hg or more from a person's normal baseline SBP.

10. An individual in stage 1 or 2 hypertension should not participate in sport and physical activity until cleared by a physician. Individuals with stage 1 hypertension usually can participate if the blood pressure is well controlled and no target organ damage or heart disease is present. The blood pressure should be rechecked every week by a qualified specialist (e.g., athletic trainer) and every 2 to 4 months by the physician.
11. For individuals in stage 2 hypertension, restrictions are placed on physical activity, especially in sports or activities with a high static component.
12. Diet modifications for hypertension include limiting sodium and saturated fats, with the total dietary fat not to exceed 30% of the total caloric intake, and increasing potassium, calcium, and magnesium intake.
13. Aerobic exercise has been shown to reduce blood pressure among individuals in the prehypertension stage and in stage 1 hypertension.
14. Orthostatic hypotension is not unusual; however, a physician should be consulted if dizziness or light-headedness becomes more frequent with a sudden change in body position.
15. Physically active people do not usually need to be concerned with hypotension. In general, a lower blood pressure is desirable as long as the person feels well.
16. SCD is rare. When it does occur, however, the on-site personnel should immediately activate the EAP and begin CPR.
17. HCM is the most common cardiac-related cause of sudden death.
18. Other cardiac-related causes of SCD include MVP, myocarditis, acquired valvular heart disease, CAD, Marfan syndrome, long QT syndrome, Wolff-Parkinson-White syndrome, and arrhythmogenic right ventricular dysplasia.
19. Noncardiac causes of SCD include commotio cordis, heat illness, head

injuries, substance abuse, exertional hyperthermia, exercise-induced anaphylaxis, exertional rhabdomyolysis, and sickle cell anemia.

20. The risk of SCD in physically active individuals can be reduced by using a two-tiered system. First, a PPE should include an extensive cardiac medical history and physical examination. If a cardiac abnormality is identified, the individual should then be referred to a specialist for further evaluation.
21. Determination of continued participation in sports should rest with the individual and the team physician after consultation with expert cardiologists, family, and team officials.

APPLICATION QUESTIONS

1. You are employed as an athletic trainer at a high school. During a preseason meeting with athletes and their parents, one of the parents expresses concern about recent news stories reporting an increased incidence of SCD in young athletes. The parent asks you to describe the screening process for SCD as it pertains to the student athletes at your school and discuss the effectiveness of the screening process. How would you respond to the parent's questions?
2. You are providing athletic training services for a high school lacrosse tournament. A 17-year-old goalie takes a line drive shot to his chest. He immediately collapses. What condition might be suspected? What is the immediate management for this potential condition?
3. A senior athlete reports feeling dizzy when getting up from a lying-down position and when suddenly changing body positions while playing tennis. What open-ended questions might you use to gather a thorough medical history? What might this person be experiencing? How would you manage this situation?
4. A collegiate wrestler has been diagnosed with sickle cell anemia. What

implications will this have on his sport performance? What can be done to lessen the impact of this disease on the individual?

5. You are the athletic trainer for a National Collegiate Athletic Association (NCAA) Division I football program. During the preparticipation examination, one of the players was found to have a blood pressure of 150/90 mm Hg. Should you be concerned with this measurement? Why or why not? What implications might this blood pressure have on athletic participation? What is your immediate course of action?

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